VASCULAR DISEASES OF THE EXTREMITIES

ARTERIAL DISORDERS

PERIPHERAL ARTERIAL DISEASE

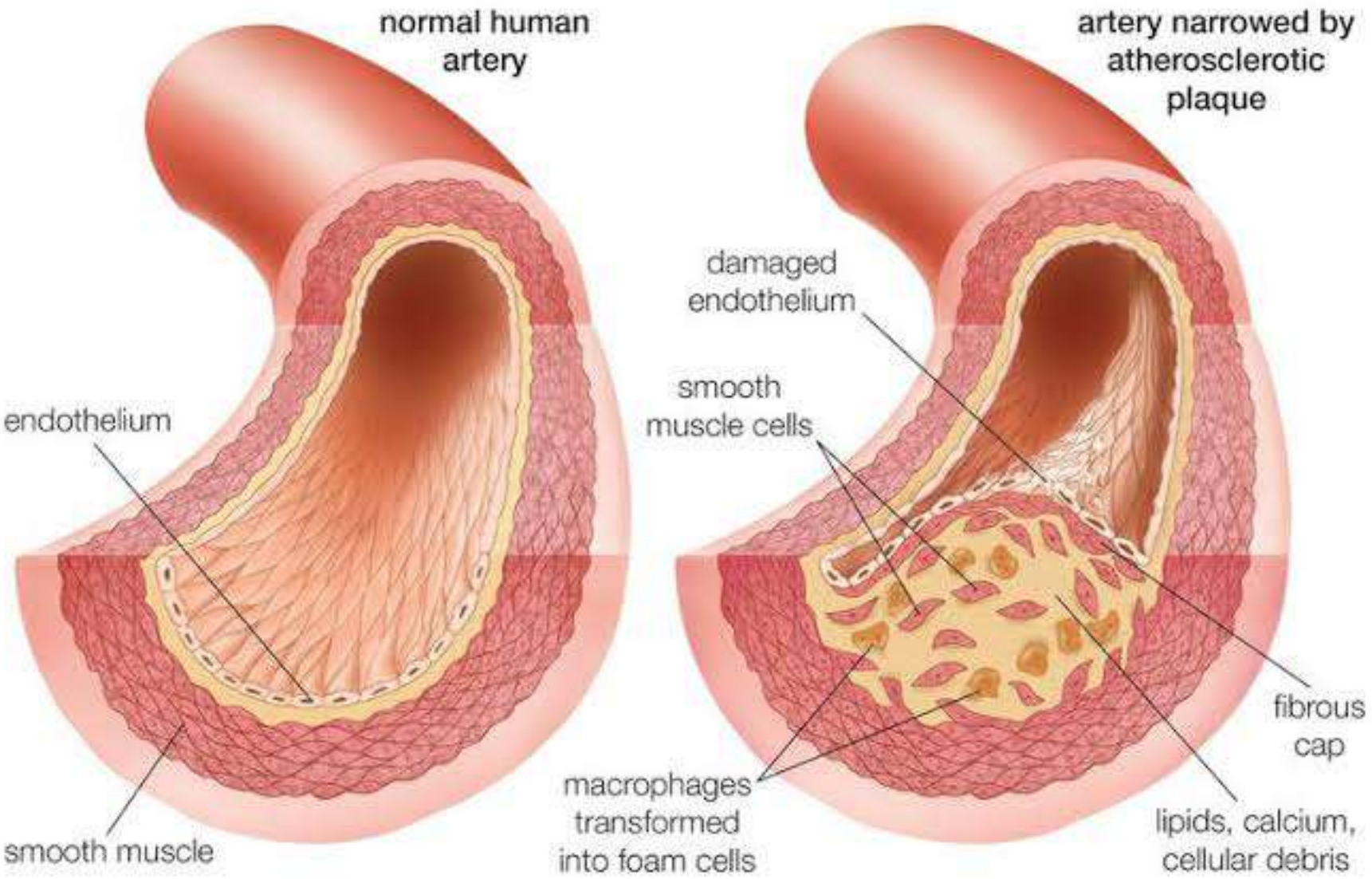
- Atherosclerosis (arteriosclerosis obliterans) is the leading cause of occlusive arterial disease
- occurs in the sixth and seventh decades of life
- increased prevalence of peripheral atherosclerotic disease

    - diabetes mellitus
    - hypercholesterolemia
    - hypertension
    - hyperhomocysteinemia
    - cigarette smokers

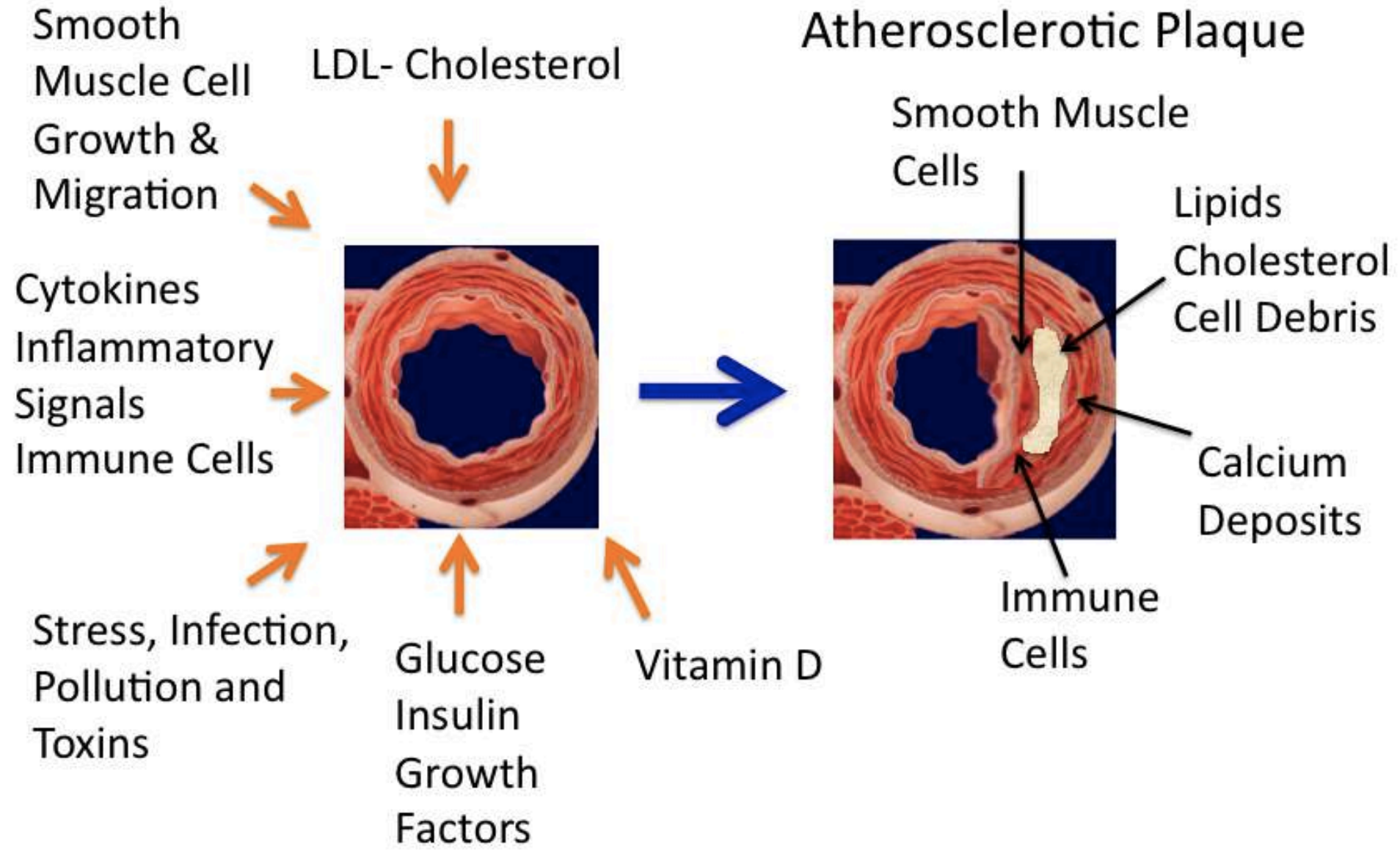
Pathology

- are usually localized in large and medium-sized vessels
- pathology - atherosclerotic plaques with calcium deposition, thinning of the media, patchy destruction of muscle and elastic fibers, fragmentation of the internal elastic lamina, and thrombi composed of platelets and fibrin
- primary sites of involvement are :
    - abdominal aorta and iliac arteries (30% of patients)
    - the femoral and popliteal arteries (80 to 90% of patients)
    - tibial and peroneal arteries (40 to 50% of patients)
- lesions occur preferentially at arterial branch points, sites of increased turbulence, altered shear stress, and intimal injury
- Involvement of the distal vasculature is most common in elderly individuals and patients with diabetes mellitus.

Atherosclerosis



Biology of Atheroma Development



Clinical Manifestation

- intermittent claudication – most common symptom and is defined as a pain, ache, cramp, numbness, or a sense of fatigue in the muscles; it occurs during exercise and is relieved by rest
- site of claudication is distal to the location of the occlusive lesion
    - aortoiliac disease (Leriche syndrome) - buttock, hip, and thigh discomfort occurs
    - femoral-popliteal disease - calf claudication
- severe arterial occlusive disease
    - rest pain or a feeling of cold or numbness in the foot and toes
    - occur at night when the legs are horizontal and improve when the legs are in a dependent position

Physical Findings

- decreased or absent pulses distal to the obstruction
- presence of bruits over the narrowed artery
- muscle atrophy, hair loss, thickened nails, smooth and shiny skin, reduced skin temperature
- pallor or cyanosis
- ulcers or gangrene
- elevation of the legs and repeated flexing of the calf muscles produce pallor of the soles of the feet
- rubor, secondary to reactive hyperemia, may develop when the legs are dependent
- peripheral edema because they keep their legs in a dependent position much of the time
- Ischemic neuritis - result in numbness and hyporeflexia.

Noninvasive Testing

- history and physical examination are usually sufficient to establish the diagnosis
- objective assessment of the severity of disease
  - digital pulse volume recordings
  - Doppler flow velocity waveform analysis
  - duplex ultrasonography (which combines B-mode imaging and pulse-wave Doppler examination)
  - segmental pressure measurements
  - transcutaneous oximetry
  - stress testing (usually using a treadmill)

Ankle-Brachial Index

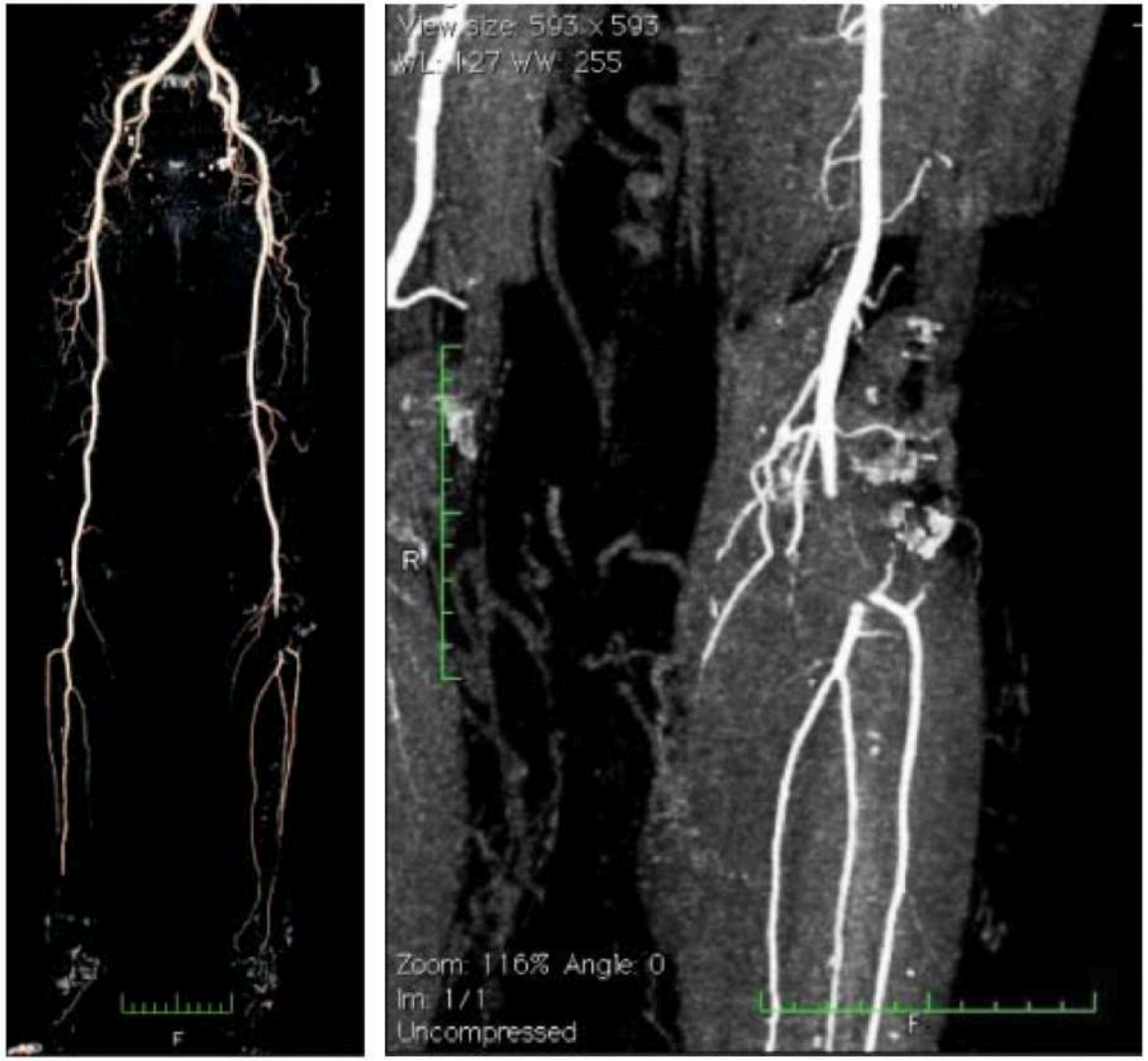
- ≥1.0 in normal individuals
- <1.0 in patients with peripheral arterial disease
- <0.5 is consistent with severe ischemia.

Treadmill testing

- assess functional limitations objectively
- allows simultaneous evaluation for the presence of coronary artery disease.

Contrast angiography

- should not be used for routine diagnostic testing but is performed prior to potential revascularization
- useful in defining the anatomy to assist operative planning
- is also indicated if nonsurgical interventions are being considered, such as percutaneous transluminal angioplasty (PTA) or thrombolysis
- Recent studies have suggested that magnetic resonance angiography has diagnostic accuracy comparable to that of contrast angiography.



(a) (b)

Figure 6.2 Computed tomography angiogram (CTA) of a patient with a popliteal artery embolus. (a) The entire arterial tree of both lower limbs looks normal, apart from an abrupt cut-off (filling defect) in the left below-the-knee popliteal artery, which is in keeping with the diagnosis of embolism. (b) The embolus lodged at the point where the artery bifurcates into the anterior tibial artery and the tibioperoneal trunk.

Prognosis

- influenced primarily by the extent of coexisting coronary artery and cerebral vascular disease
- approximately one-half of patients with symptomatic peripheral arterial disease also have significant coronary artery disease
- patients with claudication have a 70% 5-year and a 50% 10-year survival rate
- deaths are either sudden or secondary to myocardial infarction
- approximately 75% of nondiabetic patients who present with mild to moderate claudication remain symptomatically stable or improve
- approximately 5% of the group ultimately undergoing amputation
- prognosis is worse in patients who continue to smoke cigarettes or who have diabetes mellitus.

TREATMENT

Supportive measures:

- meticulous care of the feet, which should be kept clean and protected against excessive drying with moisturizing creams
- well-fitting and protective shoes are advised to reduce trauma
- sandals and shoes made of synthetic materials that do not "breathe" should be avoided
- elastic support hose should be avoided, as they reduce blood flow to the skin
- patients with ischemia at rest, shock blocks under the head of the bed together with a canopy over the feet may improve perfusion pressure and ameliorate some of the rest pain

- life-style modification – discontinue smoking
- control blood pressure in hypertensive patients but to avoid hypotensive levels
- treatment of hypercholesterolemia - it has been shown to prevent or to slow progression of the disease and to improve survival in patients with coronary atherosclerosis
- Supervised exercise training programs may improve muscle efficiency and prolong walking distance
- Patients also should be advised to walk for 30 to 45 min daily, stopping at the onset of claudication and resting until the symptoms resolve before resuming ambulation.

Pharmacologic Management

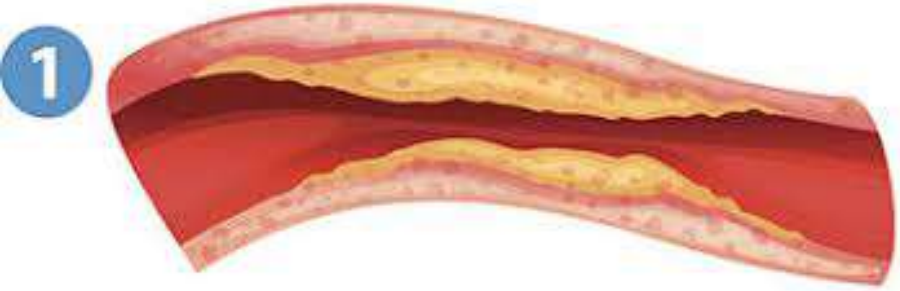
- has not been as successful as the medical treatment of coronary artery disease
- vasodilators as a class have not proved to be beneficial
- Drugs such as α-adrenergic blocking agents, calcium channel antagonists, papaverine, and other vasodilators have not been shown to be effective in patients with peripheral arterial disease
- Pentoxifylline - a substituted xanthine derivative
    - decrease blood viscosity and to increase red cell flexibility, thereby increasing blood flow to the microcirculation and enhancing tissue oxygenation
    - increased the duration of exercise in patients with claudication

- - Cilostazol - a phosphodiesterase inhibitor with vasodilator and antiplatelet properties
  - has been reported to increase claudication distance
- - Other drugs are being studied that potentially may improve claudication, such as L-arginine, which is the precursor of the endothelium-dependent vasodilator, nitric oxide, and vasodilator prostaglandins
- - Clinical trials with angiogenic growth factors such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF) are proceeding. A preliminary report suggested that intramuscular gene transfer of DNA encoding VEGF may promote collateral blood vessel growth in patients with critical limb ischemia.

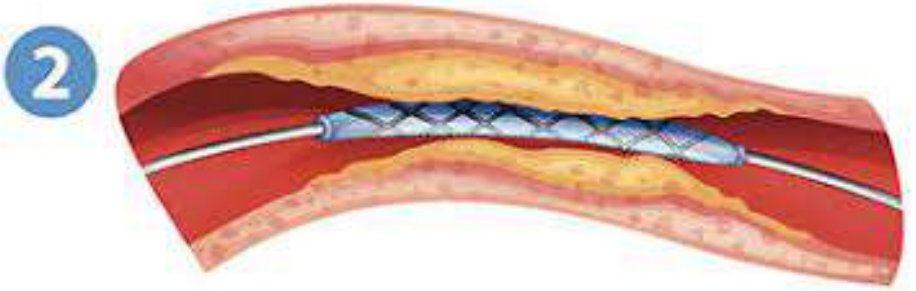
- - Aspirin and Clopidogrel - reduce the risk of adverse cardiovascular events in patients with peripheral arterial disease
- - heparin and warfarin - have not been shown to be effective in patients with chronic peripheral arterial disease but may be useful in acute arterial obstruction secondary to thrombosis or systemic embolism
- - thrombolytic intervention using drugs such as streptokinase, urokinase, or recombinant tissue plasminogen activator (tPA) may have a role in the treatment of acute thrombotic arterial occlusion but is not effective in patients with chronic arterial occlusion secondary to atherosclerosis.

- Revascularization
  - - reserved for patients with progressive, severe, or disabling symptoms and ischemia at rest, as well as for individuals who must be symptom-free because of their occupation
  - - Nonoperative interventions - include Percutaneous Transluminal Angioplasty , stent placement, and atherectomy
    - o Approximately 90 to 95% of iliac PTAs are initially successful, and the 3-year patency rate is in excess of 75%. Patency rates may be higher if a stent is placed in the iliac artery
    - o success rate for femoral-popliteal PTA - approximately 80%, with a 60% 3-year patency rate
    - o Patency rates are influenced by the severity of pretreatment stenoses; the prognosis of total occlusive lesions is worse than that of nonocclusive stenotic lesions.

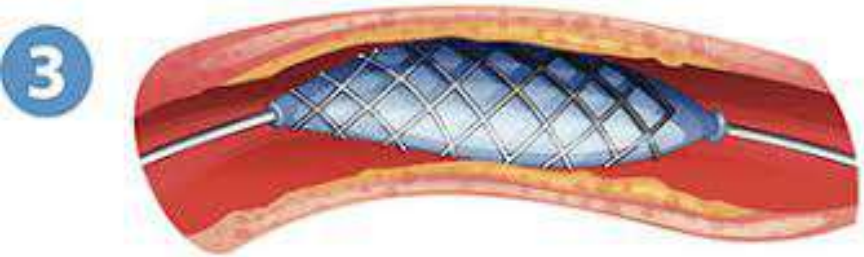
Stent with Balloon Angioplasty



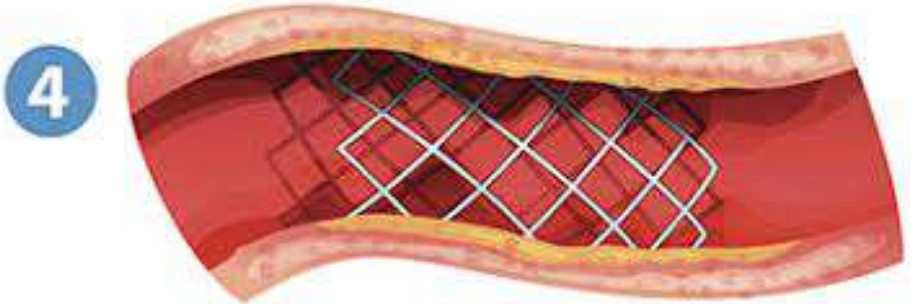
Build up of cholesterol partially blocking blood flow through the artery.



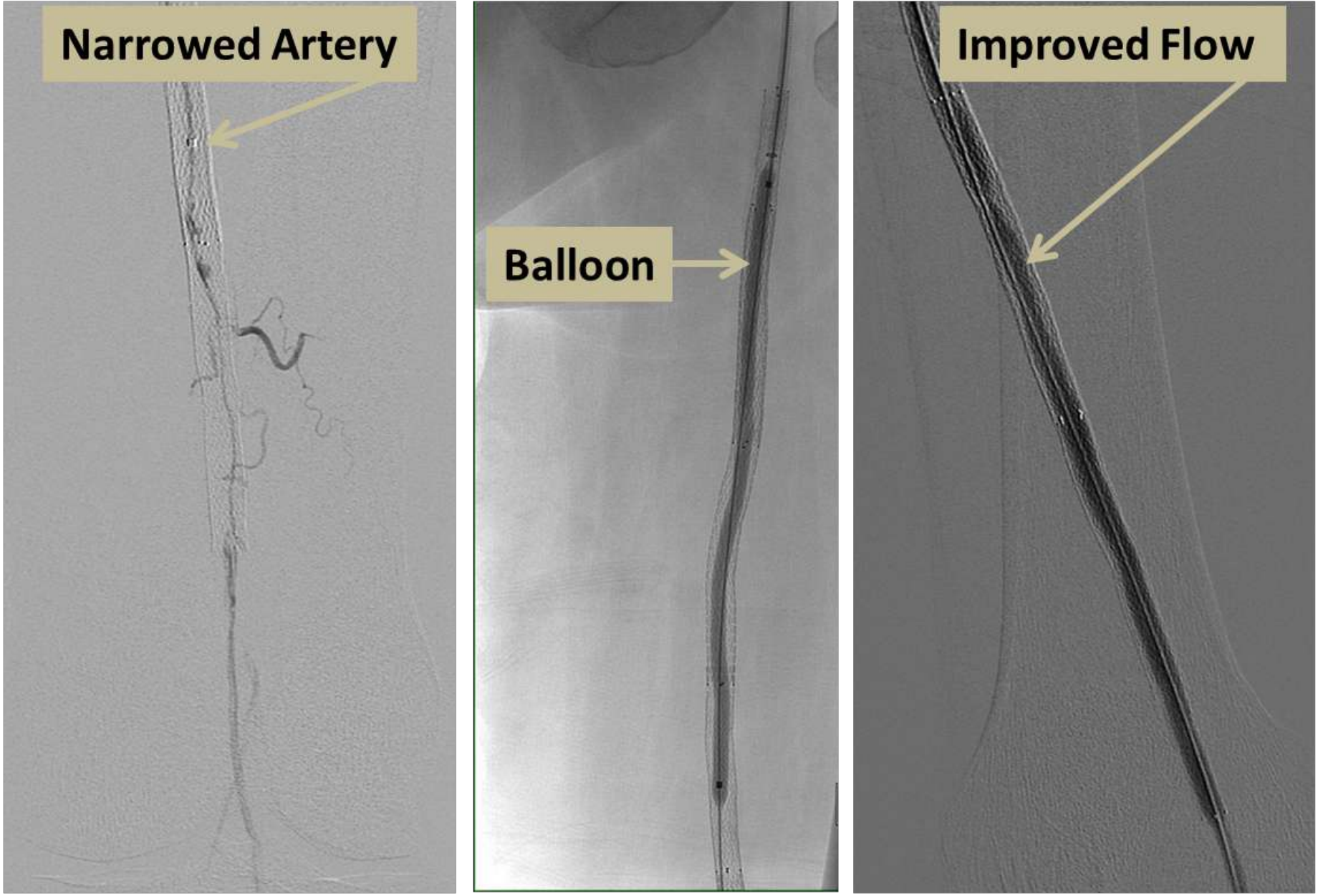
Stent with balloon inserted into partially blocked artery.



Balloon inflated to expand stent.



Balloon removed from expanded stent.



Narrowed artery (note the stent in place from prior procedure). Vessel is "opened" with balloon angioplasty. Flow in the artery is improved.

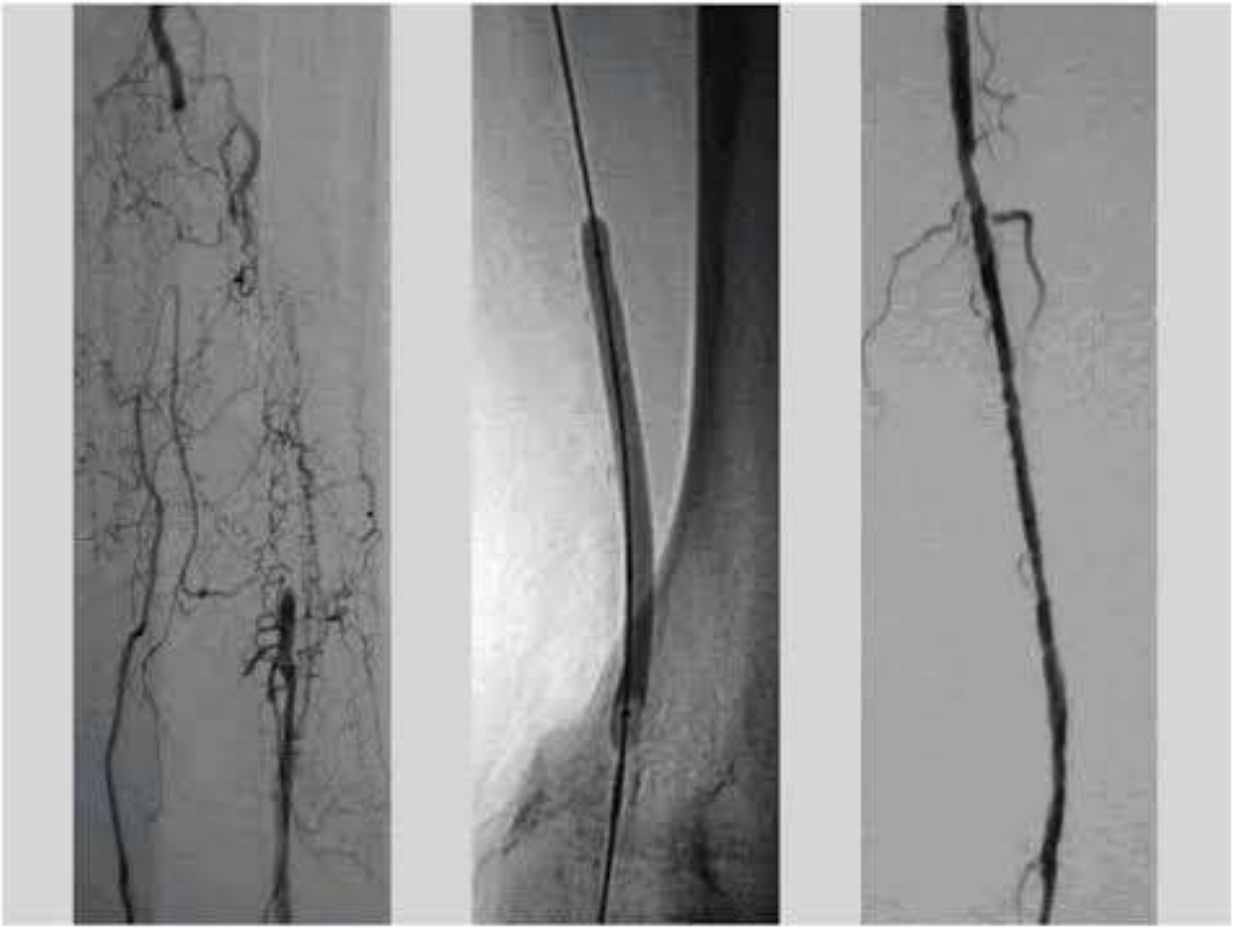
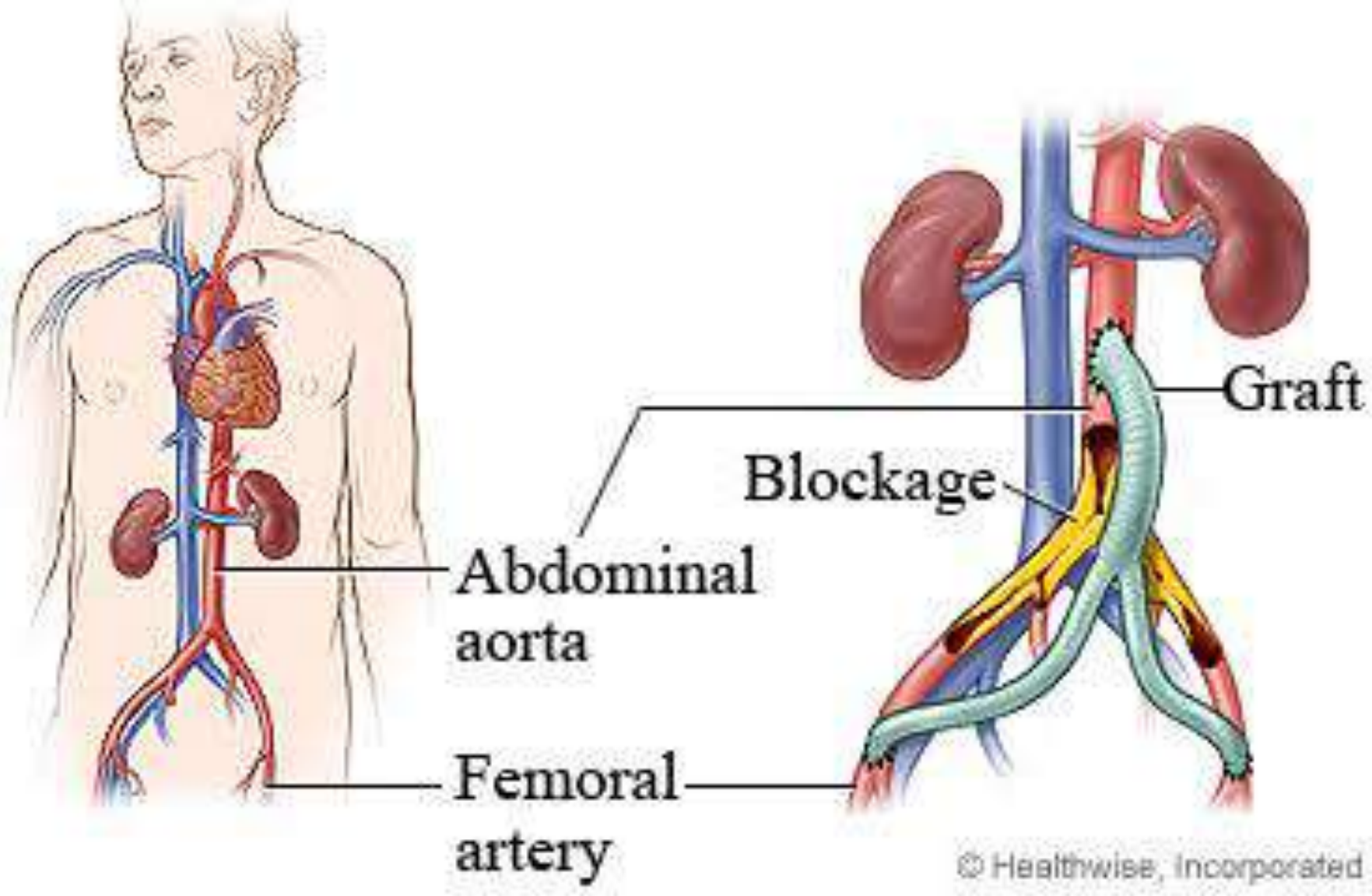


Figure 3: (A): Angiography demonstrated diffuse atherosclerotic disease with a long segment superficial femoral artery occlusion. (B): Angioplasty was performed using a 6 mm non-compliant angioplasty balloon. (C): Subsequent angiography shows resolution of the occlusion.

- Operative procedures for aortoiliac disease include aortobifemoral bypass, axillofemoral bypass, femoral-femoral bypass, and aortoiliac endarterectomy
- aortobifemoral bypass - most frequently used procedure using knitted Dacron grafts
    - Immediate graft patency approaches 99%
    - 5- and 10-year graft patency in survivors is in excess of 90 and 80%, respectively
    - Operative complications include myocardial infarction and stroke, infection of the graft, peripheral embolization, and sexual dysfunction from interruption of autonomic nerves in the pelvis. Operative mortality ranges from 1 to 3%, mostly due to ischemic heart disease.


Graft
Blockage
Abdominal aorta
Femoral artery
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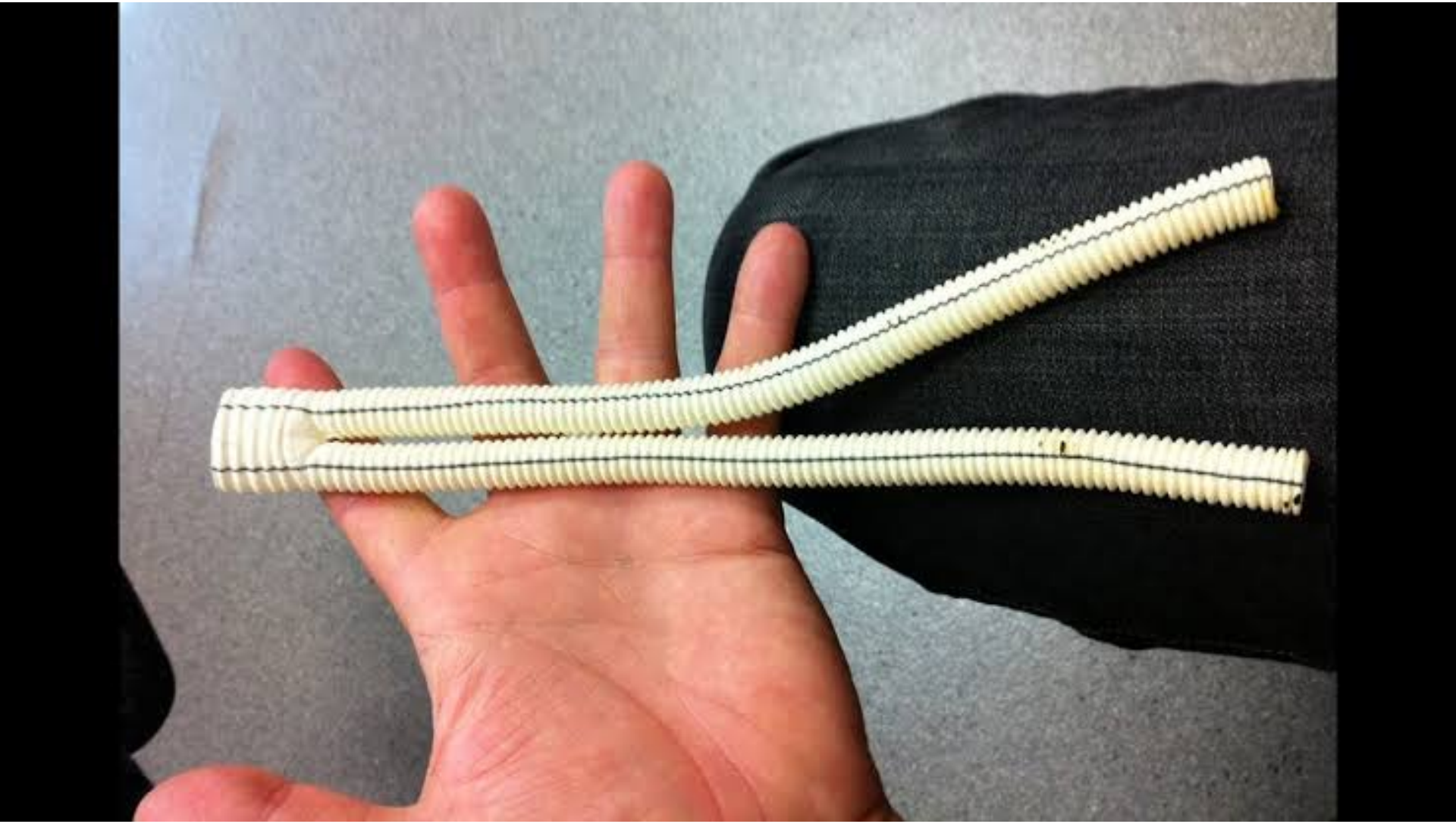

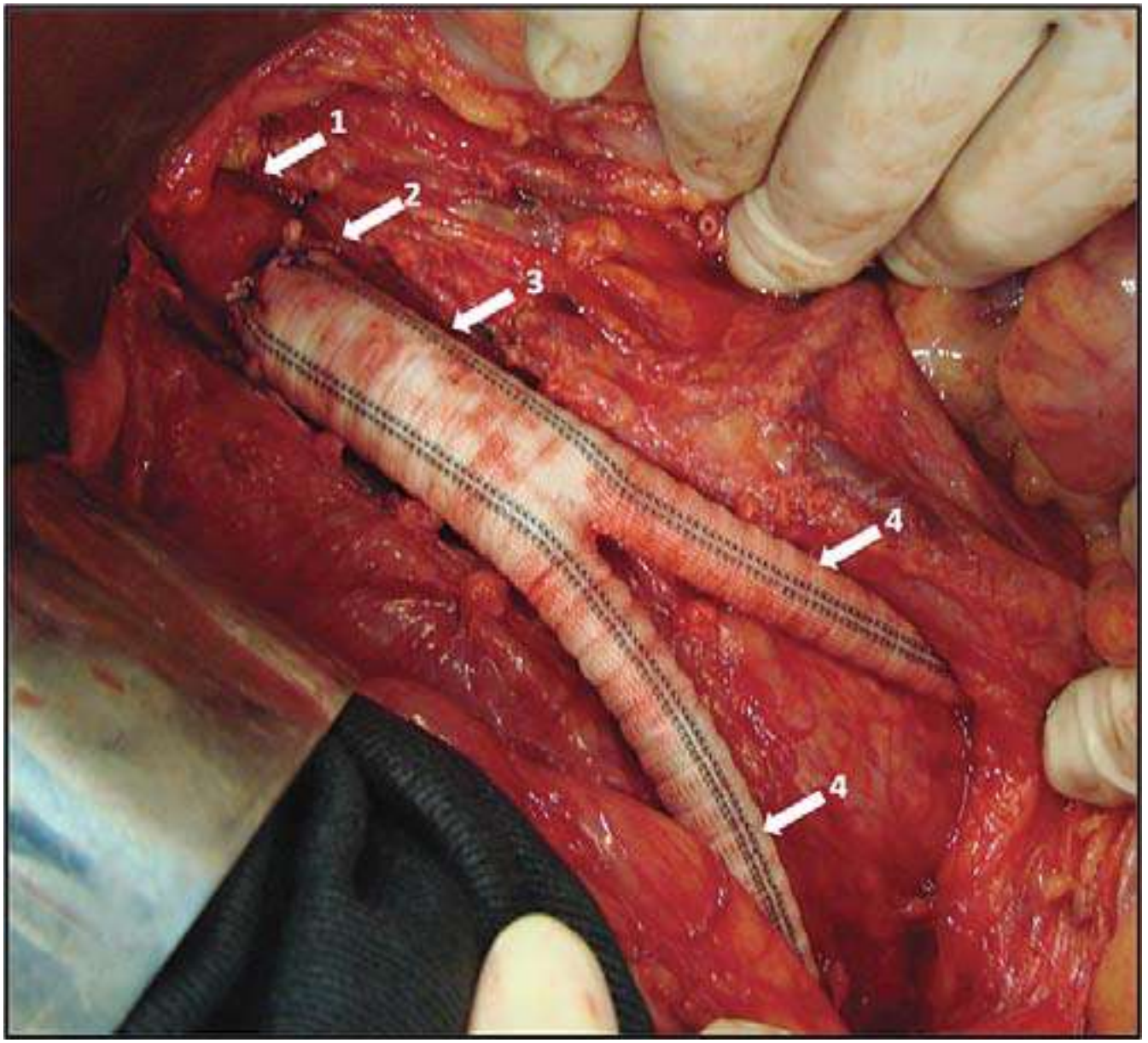
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- Operative therapy for femoral-popliteal artery disease includes in situ and reverse autogenous saphenous vein bypass grafts, placement of polytetrafluoroethylene (PTFE) or other synthetic grafts, and thromboendarterectomy
    - Operative mortality ranges from 1 to 3%
    - long-term patency rate depends on the type of graft used, the location of the distal anastomosis, and the patency of runoff vessels beyond the anastomosis.
    - Patency rates of femoral-popliteal saphenous vein bypass grafts at 1 year approach 90% and at 5 years, 70 to 80%
    - 5-year patency rates of infrapopliteal saphenous vein bypass grafts are 60 to 70%
    - 5-year patency rates of infrapopliteal PTFE grafts are less than 30%

- increased risk for cardiovascular complications - with angina, prior myocardial infarction, ventricular ectopy, heart failure, or diabetes
- Noninvasive tests, such as treadmill testing (if feasible), dipyridamole thallium or sestamibi scintigraphy, dobutamine echocardiography, and ambulatory ischemia monitoring permit further stratification of patient risk
- cardiac catheterization should be considered in patients suspected of having left main or three-vessel coronary artery disease.

THROMBOANGIITIS OBLITERANS (Buerger's disease)

- is an inflammatory occlusive vascular disorder involving small and medium-sized arteries and veins in the distal upper and lower extremities
- Cerebral, visceral, and coronary vessels may also be affected
- most frequently in men under age 40
- prevalence is higher in Asians and individuals of eastern European descent
- cause is not known
- definite relationship to cigarette smoking in patients with this disorder

- Initial stages of thromboangiitis obliterans - polymorphs infiltrate the walls of the small and medium-sized arteries and veins. The internal elastic lamina is preserved, and thrombus may develop in the vascular lumen
- As the disease progresses - mononuclear cells, fibroblasts, and giant cells replace the neutrophils
- Late stages - are characterized by perivascular fibrosis and recanalization

- Clinical features of thromboangiitis obliterans
    - claudication of the affected extremity - this disorder primarily affects distal vessels, usually confined to the calves and feet or the forearms and hands
    - Raynaud's phenomenon
    - migratory superficial vein thrombophlebitis

- Physical examination - shows normal brachial and popliteal pulses but reduced or absent radial, ulnar, and/or tibial pulses

- Arteriography - smooth, tapering segmental lesions in the distal vessels are characteristic, as are collateral vessels at sites of vascular occlusion. Proximal atherosclerotic disease is usually absent

- The diagnosis can be confirmed by excisional biopsy and pathologic examination of an involved vessel.

- no specific treatment except abstention from tobacco
- prognosis is worse in individuals who continue to smoke
- Arterial bypass of the larger vessels may be used in selected instances, as well as local debridement, depending on the symptoms and severity of ischemia
- Antibiotics may be useful
- anticoagulants and glucocorticoids are not helpful
- If these measures fail, amputation may be required

FIBROMUSCULAR DYSPLASIA

- is a hyperplastic disorder affecting medium-sized and small arteries
- occurs predominantly in females
- usually involves renal and carotid arteries but can affect extremity vessels such as the iliac and subclavian arteries
- histologic classification includes intimal, medial, and periadventitial dysplasia
- Medial dysplasia - is the most common type and is characterized by hyperplasia of the media with or without fibrosis of the elastic membrane
- identified angiographically by a "string of beads" appearance caused by thickened fibromuscular ridges contiguous with thin, less involved portions of the arterial wall
- PTA and surgical reconstruction have been beneficial in patients with debilitating symptoms or threatened limbs.

ACUTE ARTERIAL OCCLUSION

- results in the sudden cessation of blood flow to an extremity
- severity of ischemia and the viability of the extremity depend
    - the location and extent of the occlusion
    - presence and subsequent development of collateral blood vessels

- two principal causes of acute arterial occlusion:
    - embolism
    - thrombus in situ

- most common sources of arterial emboli are the heart, aorta, and large arteries
- Arterial emboli tend to lodge at vessel bifurcations
- emboli lodge most frequently in the femoral artery, followed by the iliac artery, aorta, and popliteal and tibioperoneal arteries
- arterial thrombosis in situ occurs most frequently in atherosclerotic vessels at the site of a stenosis or aneurysm and in arterial bypass grafts
- Trauma - also result in the formation of an acute arterial thrombus
- complicate arterial punctures and placement of catheters
- Less frequent causes - include the thoracic outlet compression syndrome, which causes subclavian artery occlusion, and entrapment of the popliteal artery by abnormal placement of the medial head of the gastrocnemius muscle
- Polycythemia and hypercoagulable disorders are also associated with acute arterial thrombosis.

Clinical Features

- depend on the location, duration, and severity of the obstruction
- severe pain, paresthesia, numbness, and coldness develop in the involved extremity within 1 h
- Paralysis may occur with severe and persistent ischemia
- PE - loss of pulses distal to the occlusion, cyanosis or pallor, mottling, decreased skin temperature, muscle stiffening, loss of sensation, weakness, and/or absent deep tendon reflexes
- in the presence of an adequate collateral circulation, the symptoms and findings may be less impressive
- Arteriography - is useful for confirming the diagnosis and demonstrating the location and extent of occlusion.

TREATMENT

- IV heparin - to prevent propagation of the clot
- In cases of severe ischemia of recent onset - immediate intervention to ensure reperfusion is indicated
    - Surgical thromboembolectomy or arterial bypass procedures
- Intraarterial thrombolytic therapy
    - effective when acute arterial occlusion is caused by a thrombus in an atherosclerotic vessel or arterial bypass graft
    - may also be indicated when the patient's overall condition contraindicates surgical intervention or when smaller distal vessels are occluded
    - Intraarterial urokinase
        - » 240,000 IU/h for 4 h, followed by 120,000 IU/h for a maximum of 48 h.
    - Intraarterial recombinant tPA
        - » Infusion at 1 mg/h or 0.05 mg/kg per hour

- If the limb is not in jeopardy, a more conservative approach that includes observation and administration of anticoagulants may be taken
    - Anticoagulation - prevents recurrent embolism and reduces the likelihood of thrombus propagation
    - Intravenous heparin and followed by oral warfarin

- Emboli resulting from infectious endocarditis, the presence of prosthetic heart valves, or atrial myxoma often require surgical intervention to remove the cause

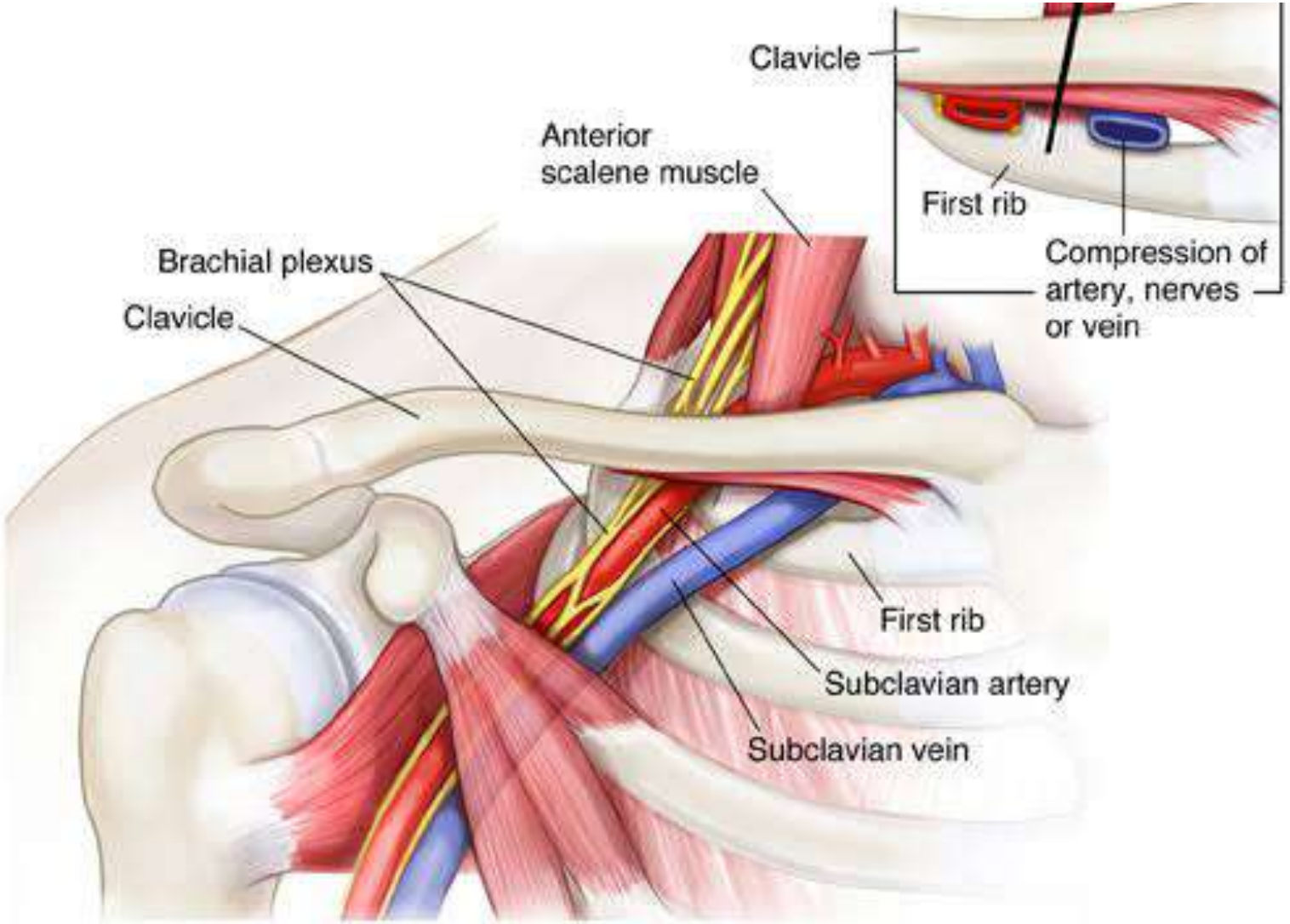
ATHEROEMBOLISM

- small deposits of fibrin, platelet, and cholesterol debris embolize from proximal atherosclerotic lesions or aneurysmal sites
- emboli tend to lodge in the small vessels of the muscle and skin and may not occlude the large vessels, distal pulses usually remain palpable
- result in ischemia and the "blue toe" syndrome
- digital necrosis and gangrene may develop
- localized areas of tenderness, pallor, and livedo reticularis occur at sites of emboli

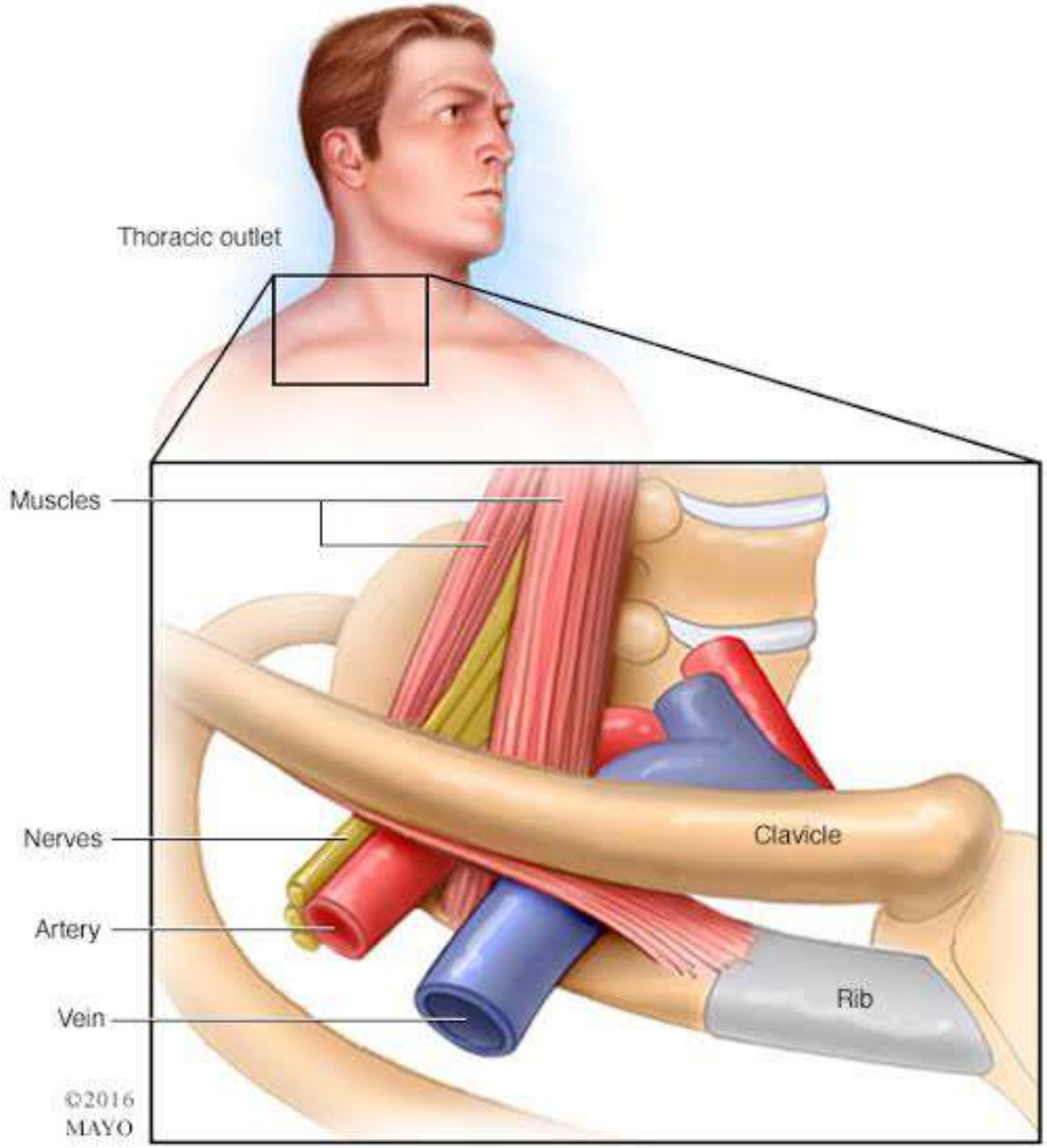
- Skin or muscle biopsy may demonstrate cholesterol crystals
- is notoriously difficult to treat
- Neither surgical revascularization procedures nor thrombolytic therapy is helpful because of the multiplicity, composition, and distal location of the emboli
- some evidence suggests that platelet inhibitors prevent atheroembolism
- Surgical intervention to remove or bypass the atherosclerotic vessel or aneurysm that causes the recurrent atheroemboli may be necessary.

THORACIC OUTLET COMPRESSION SYNDROME

- a symptom complex resulting from compression of the neurovascular bundle (artery, vein, or nerves) at the thoracic outlet
- shoulder and arm pain, weakness, paresthesia, claudication, Raynaud's phenomenon, and even ischemic tissue loss and gangrene
- PE is often normal unless provocative maneuvers are performed
- Abducting the affected arm by 90° and externally rotating the shoulder may precipitate symptoms
- Scalene maneuver (extension of the neck and rotation of the head to the side of the symptoms)



Thoracic Outlet Syndrome

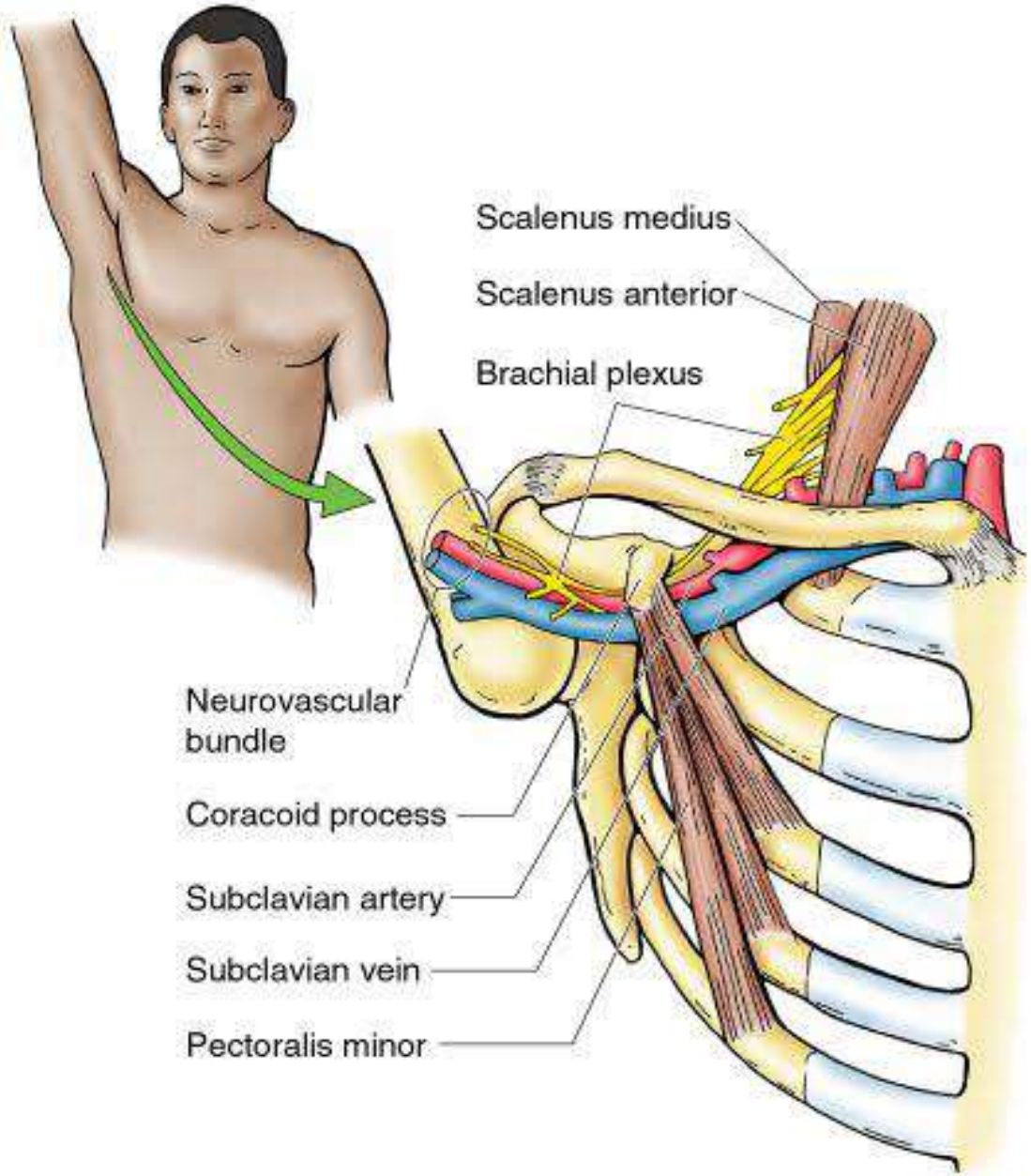


- Costoclavicular maneuver (posterior rotation of shoulders)
- Hyperabduction maneuver (raising the arm 180°)
- Chest x-ray will indicate the presence of cervical ribs
- Electromyogram - abnormal if the brachial plexus is involved

TREATMENT

- conservatively
- advised to avoid the positions that cause symptoms
- patients benefit from shoulder girdle exercises
- Surgical procedures - removal of the first rib or resection of the scalenus anticus muscle are necessary occasionally for relief of symptoms or treatment of ischemia.

6.86. Compression of the neurovascular bundle in the axilla.



ARTERIOVENOUS FISTULA

- abnormal communications between an artery and a vein, bypassing the capillary bed, may be congenital or acquired
- clinical features depend on the location and size of the fistula
- a pulsatile mass is palpable, and a thrill and bruit lasting throughout systole and diastole are present over the fistula
- clinical manifestations of chronic venous insufficiency, including peripheral edema, large, tortuous varicose veins, and stasis pigmentation become apparent because of the high venous pressure
- evidence of ischemia may occur in the distal portion of the extremity
- skin temperature is higher over the arteriovenous fistula
- large arteriovenous fistulas may result in an increased cardiac output with consequent cardiomegaly and high-output heart failure

Diagnosis

- is often evident from the physical examination
- Nicoladoni-Branham sign - compression of a large arteriovenous fistula may cause reflex slowing of the heart rate
- Arteriography can confirm the diagnosis and is useful in demonstrating the site and size of the arteriovenous fistula.

TREATMENT

- surgery, radiotherapy, or embolization
- many of these lesions are best treated conservatively using elastic support hose to reduce the consequences of venous hypertension
- embolization with autologous material, such as fat or muscle, or with hemostatic agents, such as gelatin sponges or silicon spheres, is used to obliterate the fistula
- Acquired arteriovenous fistulas are usually amenable to surgical treatment that involves division or excision of the fistula

RAYNAUD'S PHENOMENON

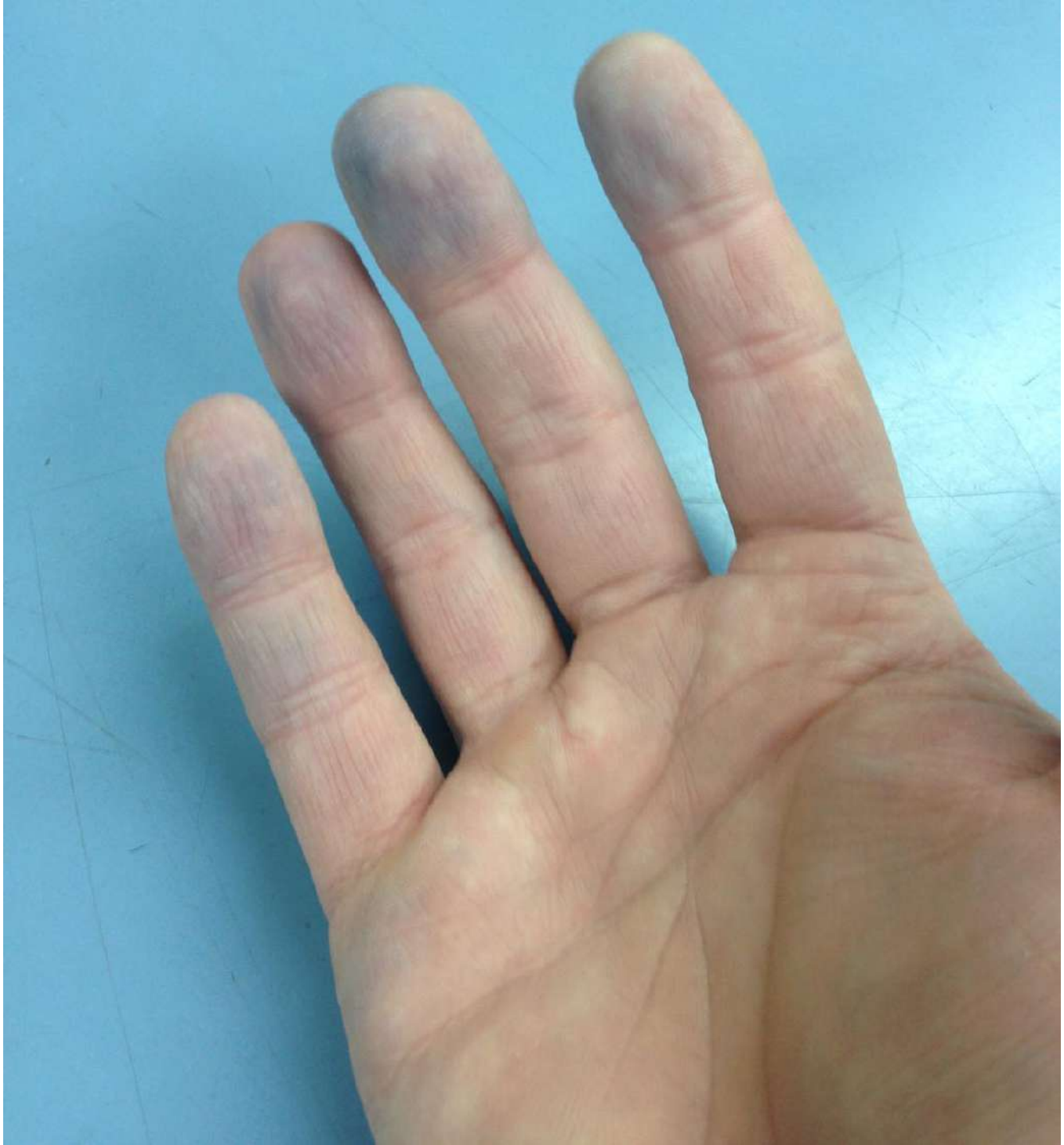
- is characterized by episodic digital ischemia, manifested clinically by the sequential development of digital blanching, cyanosis, and rubor of the fingers or toes following cold exposure and subsequent rewarming
- emotional stress may also precipitate Raynaud's phenomenon
- During the ischemic phase - blanching, or pallor, and results from vasospasm of digital arteries, capillaries and venules dilate, and cyanosis results from the deoxygenated blood that is present in these vessels
- sensation of cold or numbness or paresthesia of the digits often accompanies the phases of pallor and cyanosis.
- "reactive hyperemia" - with rewarming, the digital vasospasm resolves, and blood flow into the dilated arterioles and capillaries increases dramatically
- imparts a bright red color to the digits

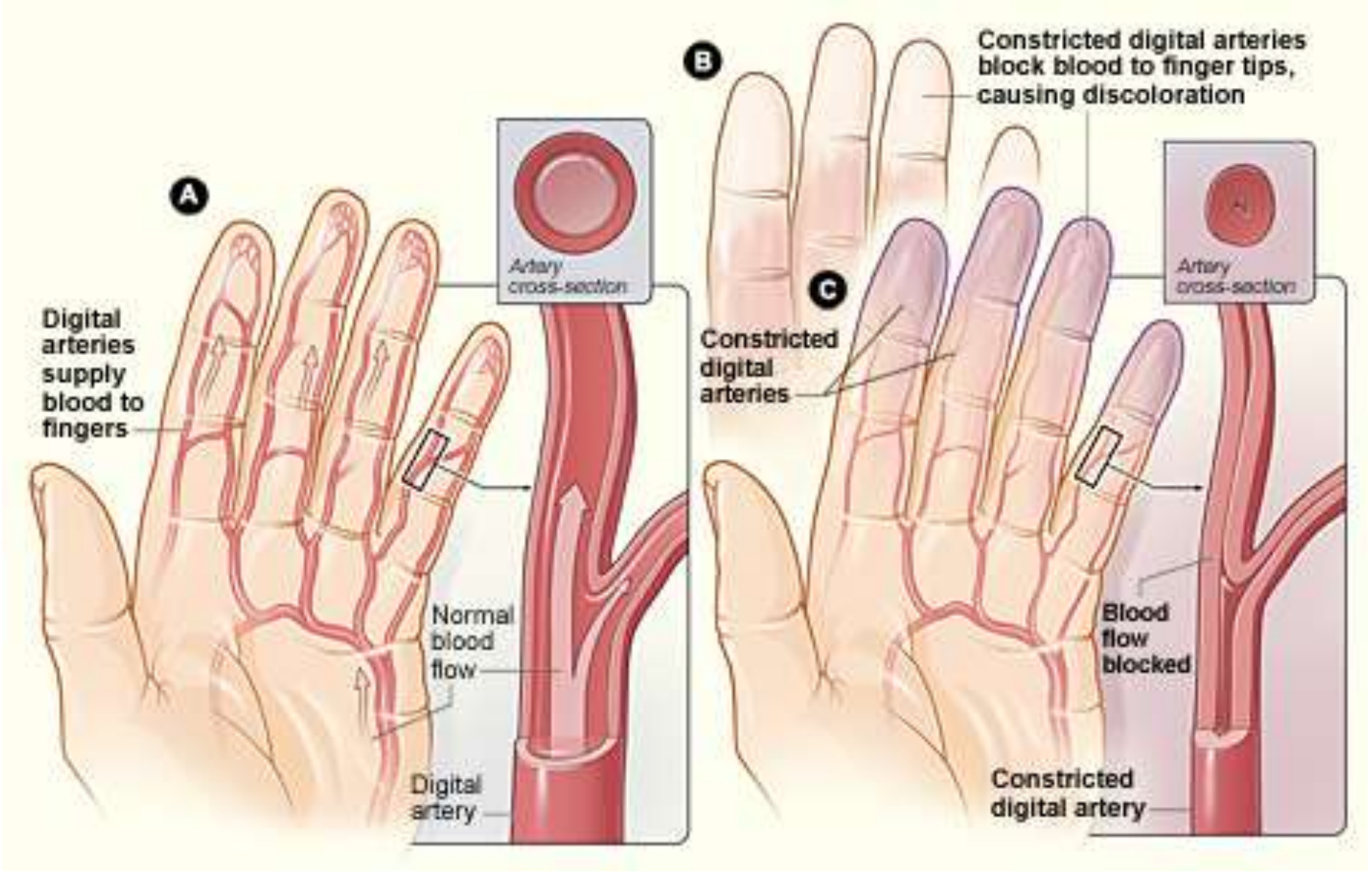
Raynaud's Phenomenon

White due to lack of blood flow

Blue due to lack of oxygen

Red when blood flow returns




A
Digital arteries supply blood to fingers
Artery cross-section
Normal blood flow
Digital artery
B
Constricted digital arteries block blood to finger tips, causing discoloration
C
Constricted digital arteries
Artery cross-section
Blood flow blocked
Constricted digital artery

Pathophysiology

- secondary to exaggerated reflex sympathetic vasoconstriction
- supported by the fact that α-adrenergic blocking drugs as well as sympathectomy decrease the frequency and severity of Raynaud's phenomenon

Raynaud's phenomenon is broadly separated into two categories:

- the idiopathic variety, termed *Raynaud's disease*
- secondary variety, which is associated with other disease states or known causes of vasospasm

Raynaud's Disease

- secondary causes of Raynaud's phenomenon have been excluded
- Over 50% of patients with Raynaud's phenomenon have Raynaud's disease
- Women are affected about five times more often than men
- age of presentation is usually between 20 and 40 years
- fingers are involved more frequently than the toes
- toes are affected in 40% of patients
- rarely, the earlobes and the tip of the nose are involved
- occurs frequently in patients who also have migraine headaches or variant angina

- physical examination often are entirely normal
- fingers and toes may be cool between attacks and may perspire excessively
- thickening and tightening of the digital subcutaneous tissue (*sclerodactyly*) develop in 10% of patients
- In general, patients with Raynaud's disease appear to have the milder forms of Raynaud's phenomenon
- Fewer than 1% of these patients lose a part of a digit
- the disease improves spontaneously in approximately 15% of patients and progresses in about 30%.

Secondary Causes of Raynaud's Phenomenon

- occurs in 80 to 90% of patients with systemic sclerosis (scleroderma)
- About 20% of patients with systemic lupus erythematosus
- about 30% of patients with dermatomyositis or polymyositis
- frequently develops in patients with rheumatoid arthritis
- Atherosclerosis of the extremities is a frequent cause
- occurs in patients with primary pulmonary hypertension
- variety of blood dyscrasias - cold agglutinins, cryoglobulinemia, or cryofibrinogenemia

- Hyperviscosity syndromes that accompany myeloproliferative disorders and Waldenstrom's macroglobulinemia
- occurs often in patients whose vocations require the use of vibrating hand tools, such as chain saws or jackhammers
- increased in pianists and typists
- electric shock injury to the hands or frostbite may lead to the later development of Raynaud's phenomenon.
- Drugs - ergot preparations, methysergide, β-adrenergic receptor antagonists, and the chemotherapeutic agents bleomycin, vinblastine, and cisplatin

TREATMENT

- most patients experience only mild and infrequent episodes and need reassurance and should be instructed to dress warmly and avoid unnecessary cold exposure
- Tobacco use is contraindicated.
- Drug treatment - reserved for the severe cases
- Calcium channel antagonists, especially nifedipine and diltiazem - decrease the frequency and severity
- Adrenergic blocking agents, reserpine - have been shown to increase nutritional blood flow to the fingers
- postsynaptic α_1-adrenergic antagonist prazosin has been used with favorable responses. Doxazosin and terazosin may also be effective
- Other sympatholytic agents, such as methyldopa, guanethidine, and phenoxybenzamine, may be useful in some patients
- Surgical sympathectomy - helpful in some patients who are unresponsive to medical therapy, but benefit is often transient

ACROCYANOSIS

- there is arterial vasoconstriction and secondary dilation of the capillaries and venules with resulting persistent cyanosis of the hands and, less frequently, the feet
- Cyanosis may be intensified by exposure to a cold environment
- Women are affected much more frequently than men
- age of onset is usually less than 30 years
- Generally, patients are asymptomatic but seek medical attention because of the discoloration
- distinguished from Raynaud's phenomenon because it is persistent and not episodic, the discoloration extends proximally from the digits, and blanching does not occur
- should be reassured and advised to dress warmly and avoid cold exposure

LIVEDO RETICULARIS

- localized areas of the extremities develop a mottled or netlike appearance of reddish to blue discoloration
- may be more prominent following cold exposure
- Idiopathic form of this disorder
    - occurs equally in men and women
    - age of onset is in the third decade
    - are usually asymptomatic and seek attention for cosmetic reasons
- can also occur following atheroembolism

PERNIO (CHILBLAINS)

- is a vasculitic disorder associated with exposure to cold
- raised erythematous lesions develop on the lower part of the legs and feet in cold weather
- are associated with pruritus and a burning sensation, and they may blister and ulcerate.
- Pathologic examination - demonstrates angiitis characterized by intimal proliferation and perivascular infiltration of mononuclear and polymorphonuclear leukocytes. Giant cells may be present in the subcutaneous tissue
- avoid exposure to cold, and ulcers should be kept clean and protected with sterile dressings
- Sympatholytic drugs may be effective in some patients

ERYTHROMELALGIA (ERYTHERMALGIA)

- is characterized by burning pain and erythema of the extremities
- feet are involved more frequently than the hands
- males are affected more frequently than females
- may occur at any age but is most common in middle age
- may be primary or secondary to myeloproliferative disorders such as polycythemia vera and essential thrombocytosis, or it may occur as an adverse effect of drugs such as nifedipine or bromocriptine
- complain of burning in the extremities that is precipitated by exposure to a warm environment and aggravated by a dependent position

- symptoms are relieved by exposing the affected area to cool air or water or by elevation.
- distinguished from ischemia secondary to peripheral arterial disorders and peripheral neuropathy because the peripheral pulses are present and the neurologic examination is normal
- no specific treatment
- aspirin may produce relief in patients with erythromelalgia secondary to myeloproliferative disease
- Treatment of associated disorders in secondary erythromelalgia may be helpful

FROSTBITE

- tissue damage results from severe environmental cold exposure or from direct contact with a very cold object
- injury results from both freezing and vasoconstriction
- usually affects the distal aspects of the extremities or exposed parts of the face, such as the ears, nose, chin, and cheeks
- pain or paresthesia, and the skin appears white and waxy
- on rewarming, there is cyanosis and erythema, wheal-and-flare formation, edema, and superficial blisters
- Deep frostbite involves muscle, nerves, and deeper blood vessels. It may result in edema of the hand or foot, vesicles and bullae, tissue necrosis, and gangrene

- Initial treatment is rewarming, accomplished by immersion of the affected part in a water bath at temperatures of 40 to 44°C (104 to 111°F)
- Massage, application of ice water, and extreme heat are contraindicated
- area should be cleansed with soap or antiseptic and sterile dressings applied
- Analgesics are often required during rewarming. Antibiotics are used if there is evidence of infection. The efficacy of sympathetic blocking drugs is not established. Following recovery, the affected extremity may exhibit increased sensitivity to cold

VENOUS DISORDERS

VENOUS THROMBOSIS

- presence of thrombus within a superficial or deep vein and the accompanying inflammatory response in the vessel wall is termed *venous thrombosis* or *thrombophlebitis*
- Virchows Triad - factors that predispose to venous thrombosis
  - stasis
  - vascular damage
  - hypercoagulability

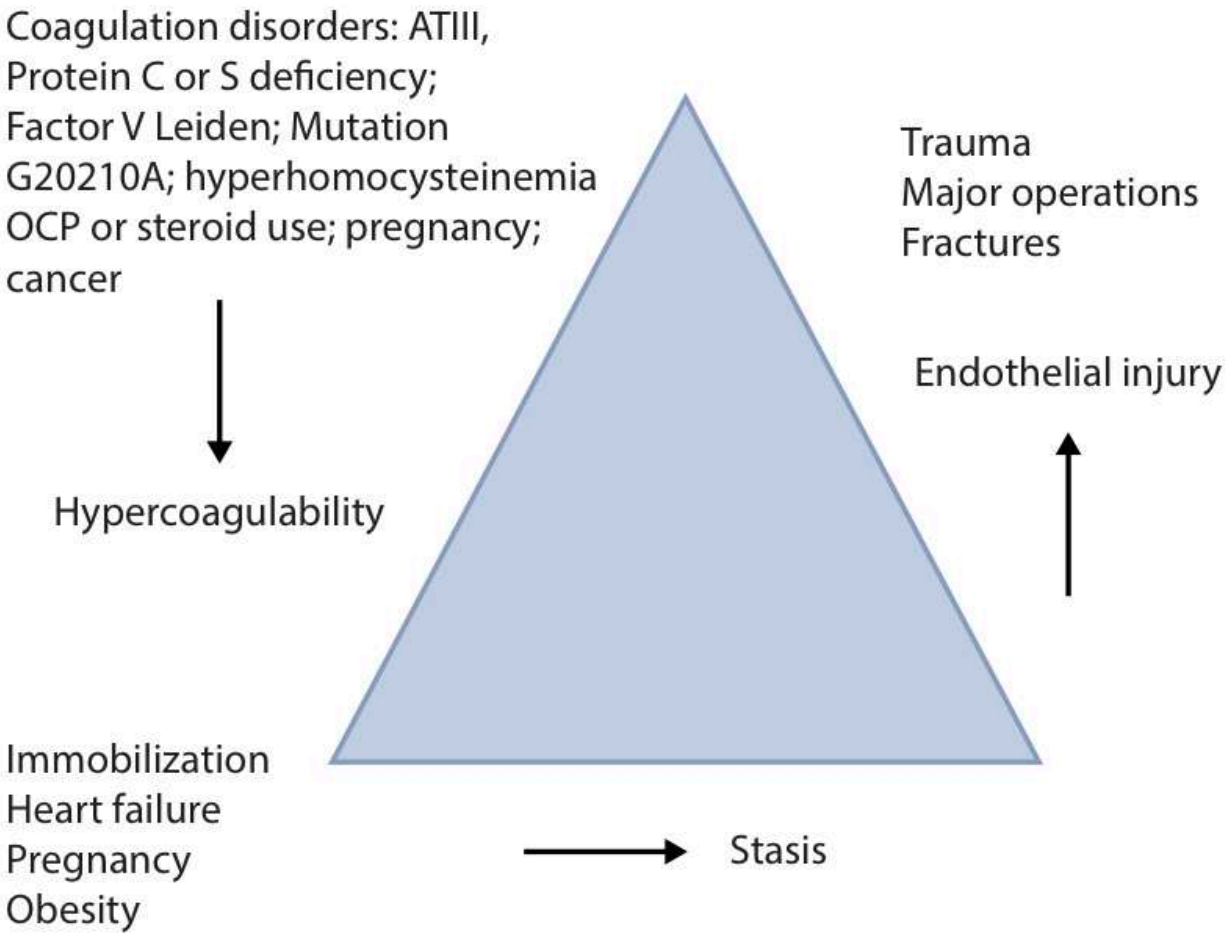


Figure 45.1 Coagulation disorders. (From Original artwork created by Meryl Simon, Division of Vascular Surgery, University of California, Davis. Sacramento, CA, 2015.)

Table 45.2 Risk factors of venous thromboembolism.

Strong risk factors (odds ratio >10)
Fracture (hip or leg)
Hip or knee replacement
Major general surgery
Major trauma
Spinal cord injury
Moderate risk factors (odds ratio 2–9)
Arthroscopic knee surgery
Central venous lines
Chemotherapy
Congestive heart or respiratory failure
Hormone replacement therapy
Malignancy
Oral contraceptive therapy
Paralytic stoke
Pregnancy/postpartum
Previous venous thromboembolism
Thrombophilia
Weak risk factors (odds ratio <2)
Bed rest >3 days
Immobility due to sitting (e.g. prolonged car or air travel)
Increasing age
Laparoscopic surgery (e.g. cholecystectomy)
Obesity
Pregnancy/antepartum
Varicose veins

Source: Anderson FA Jr and Spencer FA, *Circulation*, 107(23 Suppl. 1), 2003.

- Venous thrombosis may occur in more than 50% of patients having orthopedic surgical procedures, particularly those involving the hip or knee
- 10 to 40% of patients who undergo abdominal or thoracic operations
- prevalence of venous thrombosis is particularly high in patients with cancer of the pancreas, lungs, genitourinary tract, stomach, and breast
- Approximately 10 to 20% of patients with idiopathic deep vein thrombosis have or develop clinically overt cancer
- Immobilization - is a major predisposing cause of venous thrombosis

DEEP VENOUS THROMBOSIS

- the most important consequences of this disorder are:
  - pulmonary embolism
  - syndrome of chronic venous insufficiency

DVT of the iliac, femoral, or popliteal veins

- suggested by unilateral leg swelling, warmth, and erythema
- Tenderness may be present along the course of the involved veins, and a cord may be palpable
- There may be increased tissue turgor, distention of superficial veins, and the appearance of prominent venous collaterals
- deoxygenated hemoglobin in stagnant veins imparts a cyanotic hue to the limb, a condition called *phlegmasia cerulea dolens*
- In markedly edematous legs, the interstitial tissue pressure may exceed the capillary perfusion pressure, causing pallor, a condition designated *phlegmasia alba dolens*

DVT of the calf

- most common complaint is calf pain
- PE may reveal posterior calf tenderness, warmth, increased tissue turgor or modest swelling, and, rarely, a cord
- Homans' sign - increased resistance or pain during dorsiflexion of the foot is an unreliable diagnostic sign

DVT of the upper extremities

- incidence is increasing because of greater utilization of indwelling central venous catheters

Table 10-4. Important signs of thrombosis

Sign	Location of pain
Payr	Pain in sole both with tapping but also when bearing weight
Bisgaard	Tenderness in the retro-malleolar area
Homan	Calf pain with plantar flexion
Lowenberg	Calf pain with blood pressure cuff; difference between left and right
Meyer	Tender pressure points along the deep veins
Pratt	Pain with pressure in the popliteal space

Diagnosis

- Duplex venous ultrasonography (B-mode, i.e., two-dimensional, imaging, and pulse-wave Doppler)
  - thrombus can be detected either by direct visualisation or by inference when the vein does not collapse on compressive manoeuvres
  - measures the velocity of blood flow in veins
  - positive predictive value of duplex venous ultrasonography approaches 95% for proximal deep vein thrombosis
  - In the calf, because calf veins are more difficult to visualise than proximal veins, the sensitivity of this technique is only 50 to 75%, although its specificity is 95%

- Impedance plethysmography
  - measures changes in venous capacitance during physiologic maneuvers
  - predictive value of this test for detecting occlusive thrombi in proximal veins is approximately 90%
  - less sensitive for diagnosing deep venous thrombosis of the calves
- Magnetic resonance imaging (MRI)
  - accuracy for assessing proximal deep vein thrombosis is similar to that of duplex ultrasonography
  - useful in patients with suspected thrombosis of the superior and inferior venae cavae or pelvic veins.
- Venography
  - contrast medium is injected into a superficial vein of the foot and directed to the deep system by the application of tourniquets
  - presence of a filling defect or absence of filling of the deep veins is required to make the diagnosis

TREATMENT

Anticoagulants

- prevent thrombus propagation and allow the endogenous lytic system to operate
- Unfractionated heparin
    - initial bolus of 7500 to 10,000 IU, followed by a continuous infusion of 1000 to 1500 IU/h
    - partial thromboplastin time (aPTT) - approximately twice the control value
    - In fewer than 5% of patients, heparin therapy may cause thrombocytopenia. Infrequently, these patients develop arterial thrombosis and ischemia

- Low-molecular-weight (4000 to 6000 Da) heparins are reported to be as effective as or better than conventional, unfractionated heparin in preventing extension or recurrence of venous thrombosis
    - enoxaparin is 1 mg/kg subcutaneously bid
    - incidence of thrombocytopenia is less with low-molecular-weight heparin than with conventional preparations
- Hirudin - a direct thrombin inhibitor
    - for patients in whom heparin is contraindicated because of heparin-induced thrombocytopenia
- Warfarin - dose of warfarin should be adjusted to maintain the prothrombin time at an international normalized ratio (INR) of 2.0 to 3.0

- for patients with proximal deep vein thrombosis, anticoagulation is indicated since pulmonary embolism may occur in approximately 50% of untreated individuals
- for isolated deep vein thrombosis of the calf is controversial
- However, approximately 20 to 30% of calf thrombi propagate to the thigh, thereby increasing the risk of pulmonary embolism
- overall incidence of pulmonary embolism in patients presenting initially with deep calf vein thrombosis is 5 to 20%

- - Anticoagulant treatment should be continued for at least 3 to 6 months for patients with acute idiopathic deep vein thrombosis and for those with a temporary risk factor for venous thrombosis to decrease the chance of recurrence
- - is indefinite for patients with recurrent deep vein thrombosis and for those in whom associated causes, such as malignancy or hypercoagulability, have not been eliminated
- - IVC filter insertion - if treatment with anticoagulants is contraindicated because of a bleeding diathesis or risk of hemorrhage

- Thrombolytics
  - - there is no evidence that thrombolytic therapy is more effective than anticoagulants in preventing pulmonary embolism
  - - thrombolytic drugs may accelerate clot lysis, preserve venous valves, and decrease the potential for developing postphlebitic syndrome.

Prophylaxis

- in clinical situations where the risk of deep vein thrombosis is high
- Low-dose unfractionated heparin
  - 5000 units 2 h prior to surgery and then 5000 units every 8 to 12 h postoperatively
- warfarin, and external pneumatic compression are all useful
- Low-molecular-weight heparins - are said to be more effective than conventional heparin and to cause an equal or lower incidence of bleeding
- Danaparoid - a low-molecular-weight heparinoid, may be used for prophylaxis in patients undergoing hip surgery
- Warfarin - prothrombin time equivalent to an INR of 2.0 to 3.0 is effective in preventing deep vein thrombosis associated with bone fractures and orthopedic surgery
- External pneumatic compression devices – if anticoagulation might cause serious bleeding, as during neurosurgery or transurethral resection of the prostate

SUPERFICIAL VEIN THROMBOSIS

- thrombosis of the greater or lesser saphenous veins or their tributaries
- does not result in pulmonary embolism
- associated with intravenous catheters and infusions
- occurs in varicose veins
- may develop in association with deep vein thrombosis
- Migrating superficial vein thrombosis is often a marker for a carcinoma
- may also occur in patients with vasculitides, such as thromboangiitis obliterans

TREATMENT

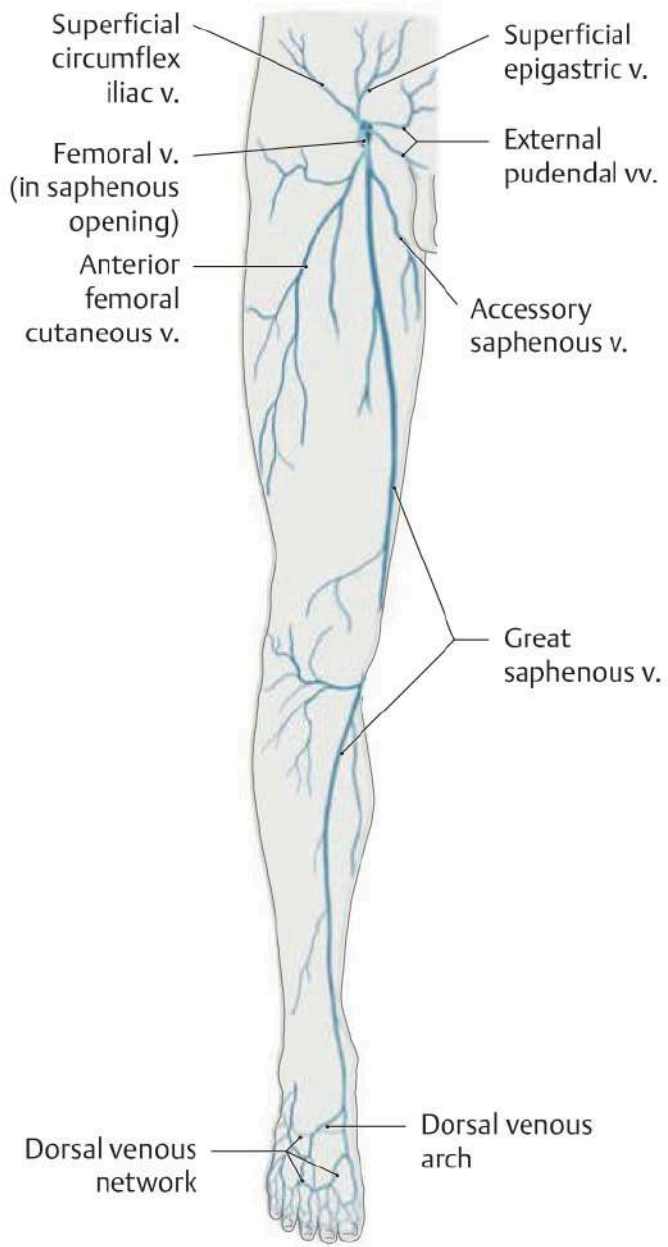
- is primarily supportive
- bed rest with leg elevation and application of warm compresses
- Nonsteroidal antiinflammatory drugs - may provide analgesia but may also obscure clinical evidence of thrombus propagation

Table 10-6. Differential diagnosis: thrombophlebitis versus phlebothrombosis

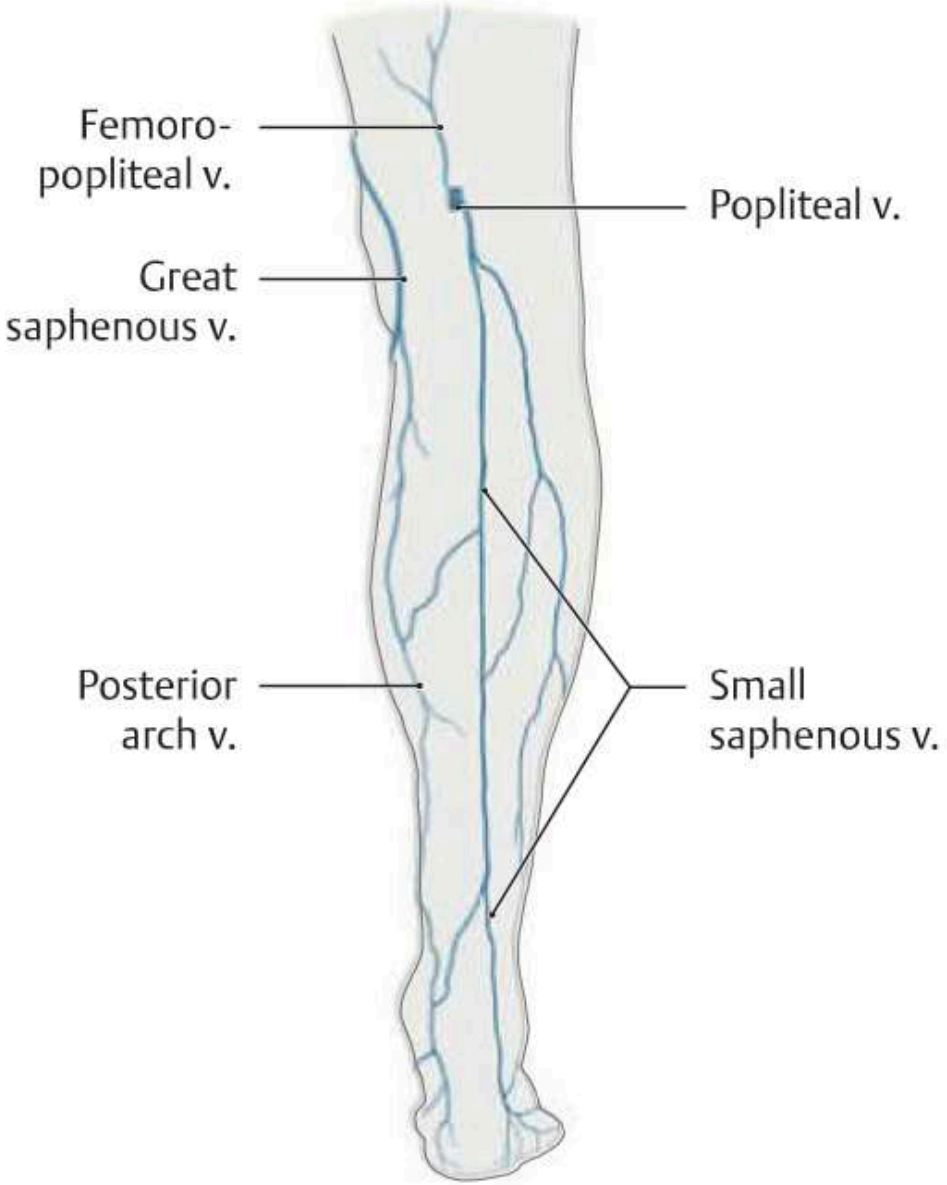
Thrombophlebitis	Phlebothrombosis
Signs of inflammation: erythema, warmth, pain, localization	Cyanosis of leg, especially when standing
Usually localized edema, although can be diffuse	Diffuse swelling of limb (difference in circumference compared to normal limb, firmer consistency)
Painful inflamed superficial venous cord	No visible or palpable thrombosis Dull pain in entire leg Filled "sentinel veins" on back of foot, over tibia Positive signs (see Table 10-4)

Surgical Anatomy of Lower Limb Veins

Fig. 34.5 Superficial (epifascial) veins of the lower limb



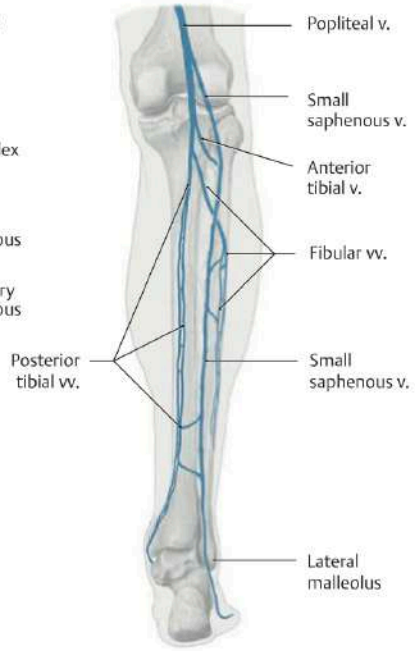
A Right limb, anterior view.



B Right limb, posterior view.

Fig. 34.6 Deep veins of the lower limb

A Right limb, anterior view.



B Right limb, posterior view.

Fig. 34.7 Veins of the sole of the foot
Right foot, plantar view.

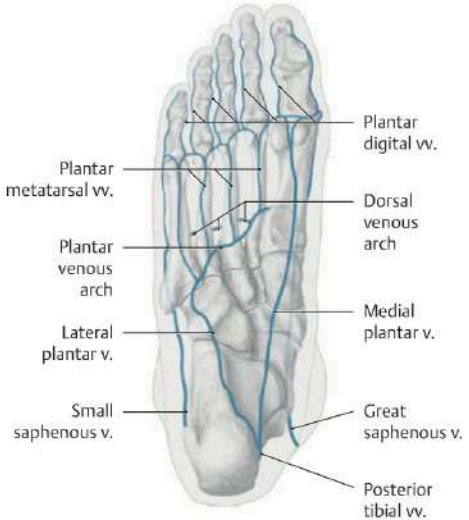
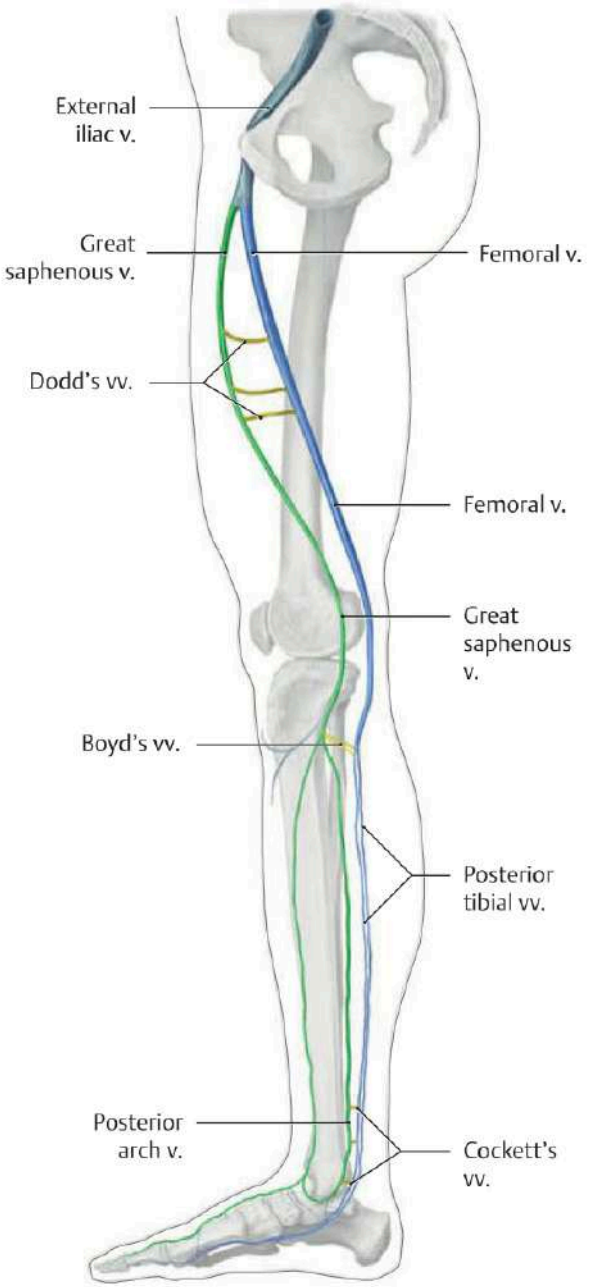
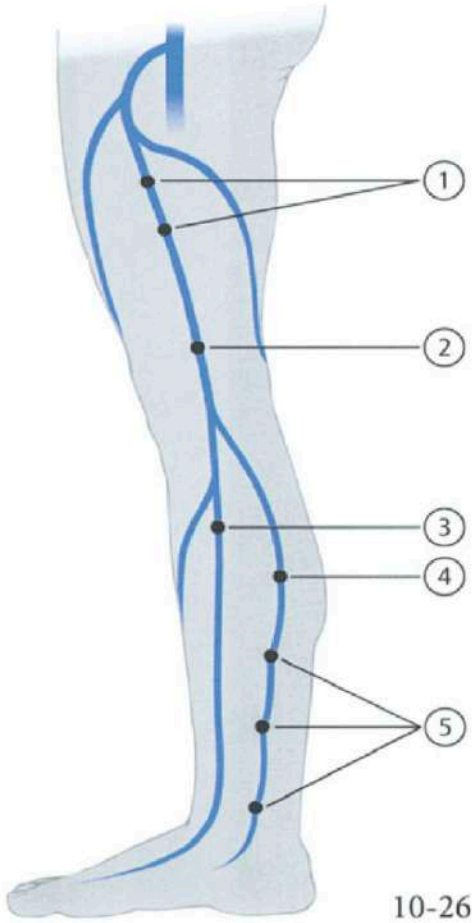


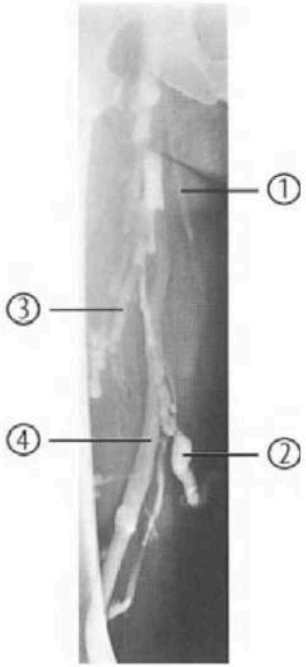
***Fig. 34.8* Clinically important perforating veins**
Right leg, medial view.





10-26

Fig. 10-26. The clinically important perforating veins on the medial aspect of the leg. The perforating veins are the veins that connect the deep and superficial veins, passing through the fascia. There are more than 100 perforating veins in each leg. In a healthy leg, the flow goes from the superficial to the deep venous system. (After HACH-WUNDERLE 1997)
① DODD veins, ② HUNTER vein, ③ BOYD veins, ④ SHERMAN vein, ⑤ COCKETT veins



10-27

Fig. 10-27. Phlebography showing widened perforating veins of the greater saphenous vein. In a complete varicosity, the saphenofemoral junction valves are incompetent. In an incomplete varicosity, the problem begins with the perforating veins and accessory veins. Here the perforator is in the middle of the thigh and is a DODD vein.
① Greater saphenous vein
② Dilated DODD perforating vein
③ Deep femoral vein
④ Superficial femoral vein

Figs. 10-28 – 10-30. Various degrees of severity of incomplete varicosity of the greater saphenous vein with DODD perforating veins

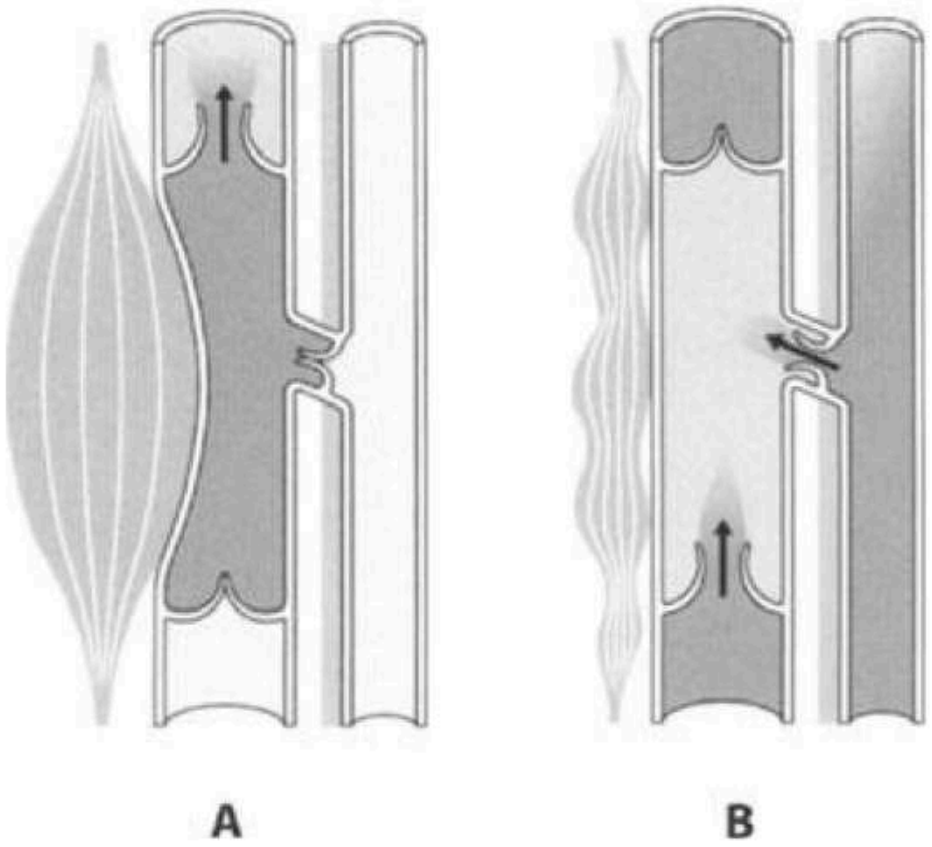
Table 10-1. Number of venous valves

Vein	Number of valves	
	Average	Range
Superficial femoral	4	1–9
Popliteal	2	1–5
Posterior tibial	10	7–20
Anterior tibial	10	9–12
Peroneal	10	6–12
Greater saphenous	10	7–20
Lesser saphenous	8	5–15

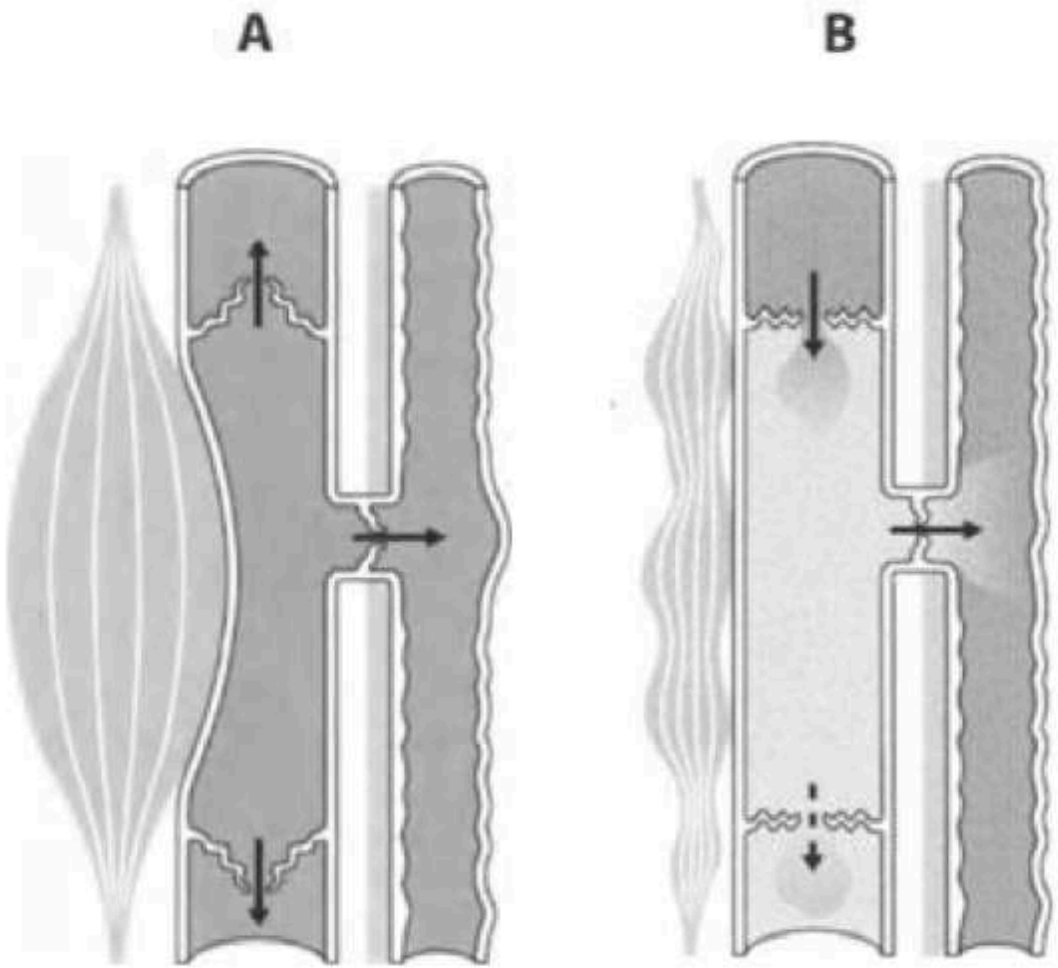
VARICOSE VEINS

- are dilated, tortuous and elongated superficial veins
- result from defective structure and function of the valves of the saphenous veins, from intrinsic weakness of the vein wall, from high intraluminal pressure, or, rarely, from arteriovenous fistulas
- Primary varicose veins
    - originate in the superficial system
    - two to three times as frequently in women as in men
    - half of patients have a family history of varicose veins
- Secondary varicose veins
    - result from deep venous insufficiency and incompetent perforating veins or

      from deep venous occlusion causing enlargement of superficial veins that are serving as collaterals



Normal vein



Varicose vein

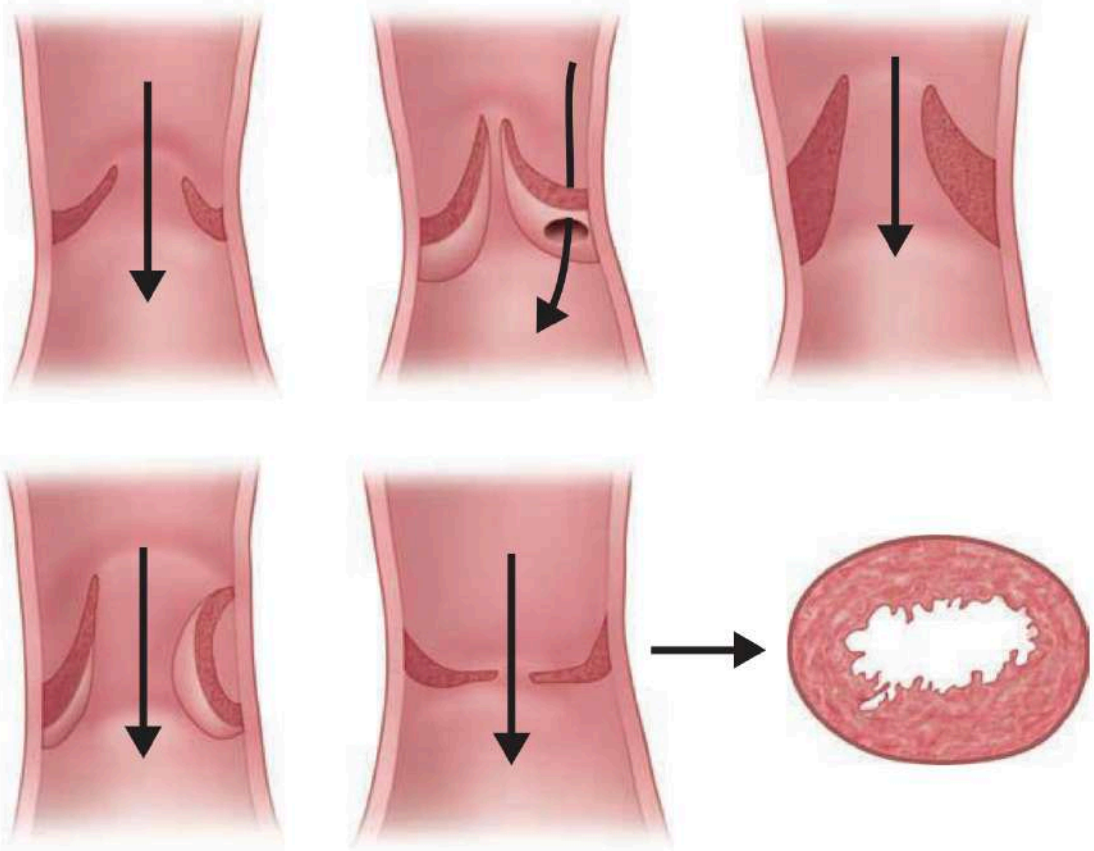


Figure 46.2 Mechanism of post-thrombotic reflux. Various degrees of valve cusp damage result in reflux. In some cases, valve cusps are relatively undamaged, but become redundant and refluxive due to valve station fibrosis and restriction.

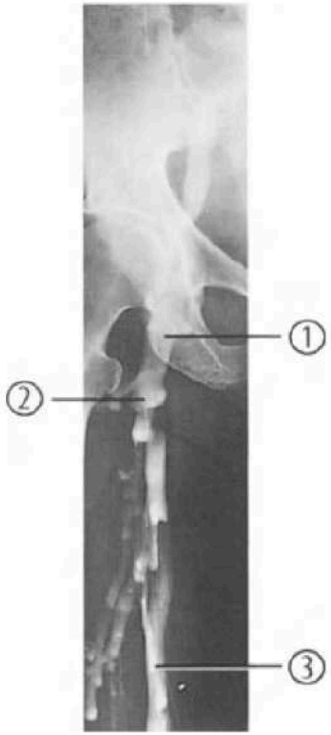
- dull ache or pressure sensation in the legs after prolonged standing
- it is relieved with leg elevation
- legs feel heavy, and mild ankle edema develops occasionally
- rarely, a varicosity ruptures and bleeds and results to skin ulcerations near the ankle
- treated with conservative measures
- symptoms often decrease when the legs are elevated periodically, when prolonged standing is avoided, and when elastic support hose are worn
- External compression stockings
- Sclerotherapy - small symptomatic varicose veins

Table 46.1 Clinical stage, etiology, anatomy and pathology classification: summary.

Clinical	Etiologic	Anatomic	Pathophysiologic
C0: no visible or palpable signs of venous disease	**Ec:** congenital	**As:** superficial veins	**Pr:** reflux
C1: telangiectasies or reticular veins	**Ep:** primary	**Ap:** perforator veins	**Po:** obstruction
C2: varicose veins	**Es:** secondary (post-thrombotic)	**Ad:** deep veins	**Pr,o:** reflux and obstruction
C3: edema	**En:** no venous cause identified	**An:** no venous location identified	**Pn:** no venous pathophysiology identifiable
C4a: pigmentation or eczema			
C4b: lipodermatosclerosis or atrophic blanche			
C5: healed venous ulcer			
C6: active venous ulcer			
S: symptomatic			
A: asymptomatic			

Table 46.2 Common clinical patterns in chronic venous insufficiency (CVI).

Clinical pattern	Symptoms and presentation
Varices	
Uncomplicated	Cosmetic or health concerns or both.
With local symptoms	Pain is confined to the varices and is not diffuse.
With local complications	Superficial thrombophlebitis, internal rupture with hematoma or external rupture through a 'pinpoint' ulcer.
Complex varicose disease	Diffuse limb pain, ankle swelling, skin changes or ulcer.
Venous hypertension syndrome	Severe orthostatic venous pain; patients are often young or middle-aged; other features of CVI are minimal or absent.
Venous leg swelling	Other features of CVI may be absent or variable; swelling may be bilateral. Iliac vein obstruction is common.
Complex multisystem venous disease	Clinical features of advanced CVI (i.e. pain, swelling, stasis dermatitis) or ulceration present in varying combinations. Multisystem involvement with combined obstruction reflux involvement.



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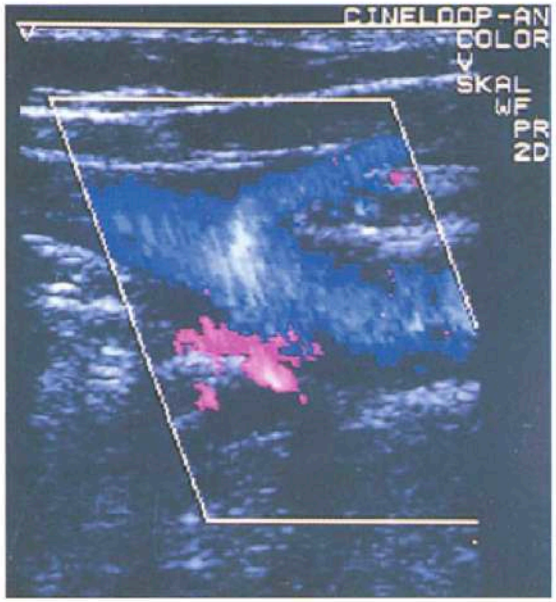
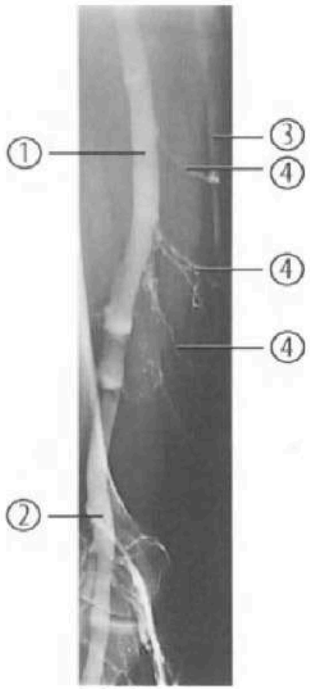


10-8

Fig. 10-7. Phlebography showing the right external iliac, common femoral, superficial femoral and deep femoral veins. The common femoral vein arises 5–10 cm below the inguinal ligament from the joining of the superficial and deep femoral veins.
① Common femoral vein
② Deep femoral vein
③ Superficial femoral vein

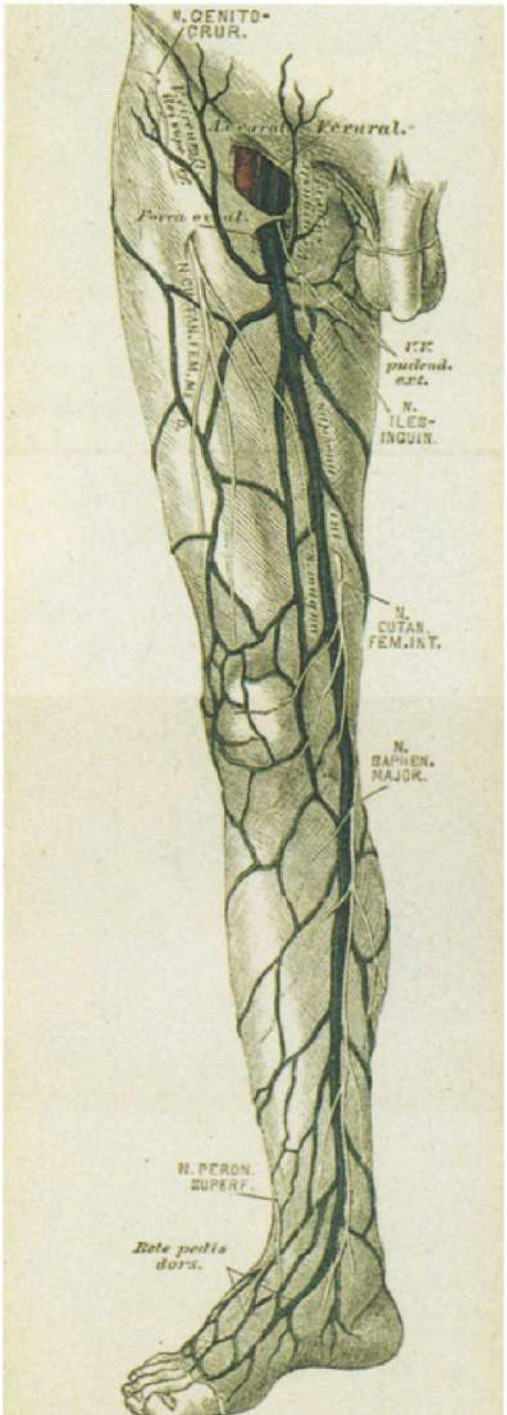
Fig. 10-8. Color duplex ultrasonography, showing the normal appearance of the sapheno-femoral junction during expiration. Blood is flowing into the junction via the greater saphenous vein and the epigastric veins from the abdominal wall



10-9

Fig. 10-9. Phlebography showing the superficial femoral, popliteal and greater saphenous veins, as well as the group of DODD perforating veins. The superficial femoral vein begins in the adductor canal as a continuation of the popliteal vein. The greater saphenous vein courses from the medial edge of the foot and along the medial aspect of the calf, draining numerous superficial veins. These accessory veins usually become visible when varicosities develop.

① Superficial femoral vein
② Popliteal vein
③ Greater saphenous vein
④ DODD perforating veins



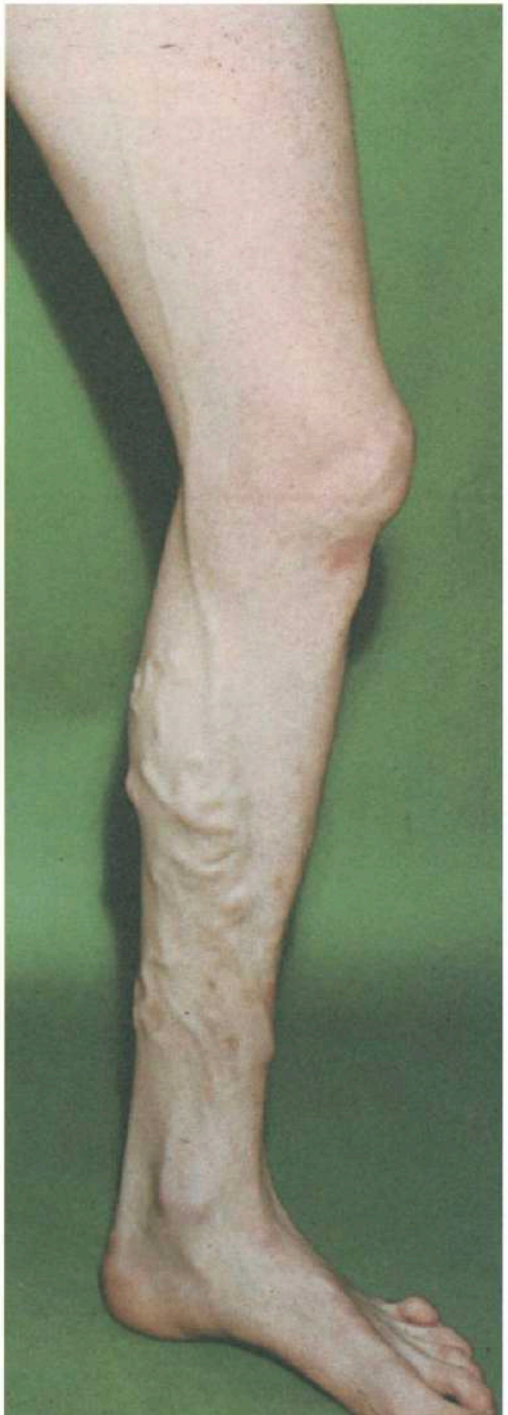
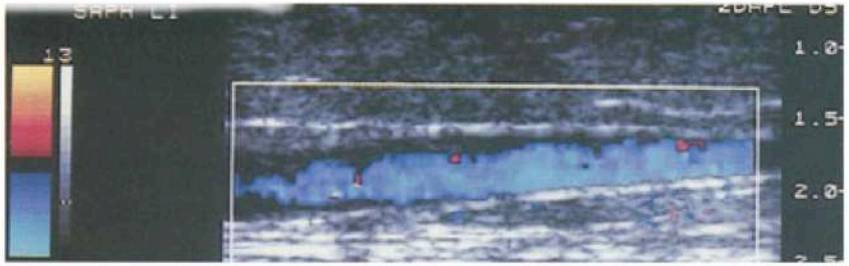


Fig. 10-16. Superficial veins of the leg. (From C. HEITZMANN, *Deskriptive und topographische Anatomie der Menschen*, 1893)

Fig. 10-17. Stage IV varicosity of the greater saphenous vein caused by an incompetent saphenofemoral junction. Varicosities of the larger superficial limb veins always develop proximally and extend distally (groin to foot). One distinguishes between primary varicosities caused by congenital vein valve or wall defects and secondary varicosities usually related to the post-thrombotic syndrome. In this picture, a cylindrical varicosity courses across the thigh. The calf shows marked varicosities with coiling; the defect extends to the level of the malleolus, making this stage IV

Fig. 10-19. Color duplex ultrasonography of the greater saphenous vein showing flow towards the heart in expiration

Fig. 10-20. Same section of the greater saphenous vein with Valsalva maneuver. The reversal of flow indicates incompetent venous valves



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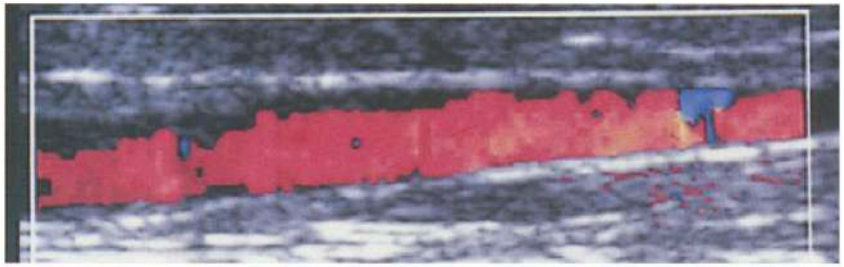

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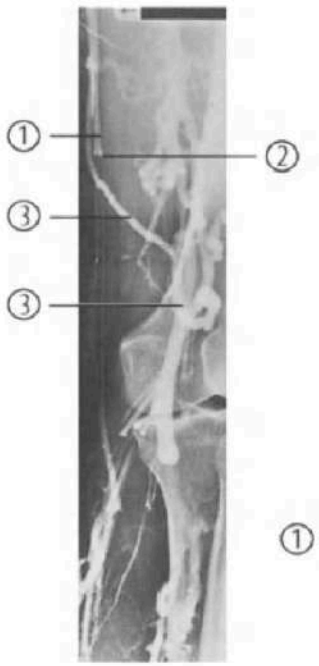
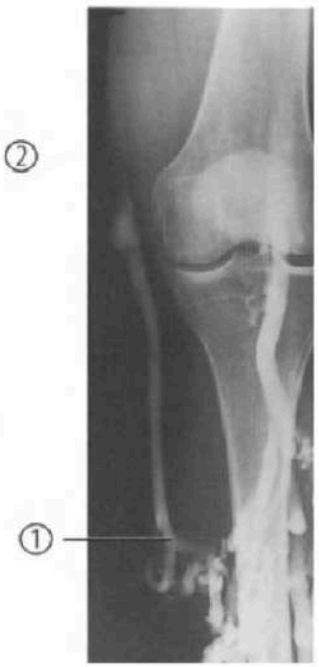


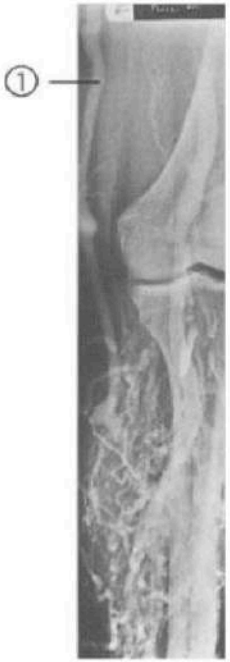
10-21

Fig. 10-21. Phlebography showing a varicosity of the greater saphenous vein reaching from the sapheno-femoral junction to the distal calf, thus a stage II lesion. In addition, the GIACOMINI vein (CARLO GIACOMINI, 1840–1898, Italian anatomist) is incompetent (stage II: incompetent valves from the inguinal area to one handbreadth above the knee).
① Greater saphenous vein
② Distal extent of incompetent valves
③ Giacomini anastomosis



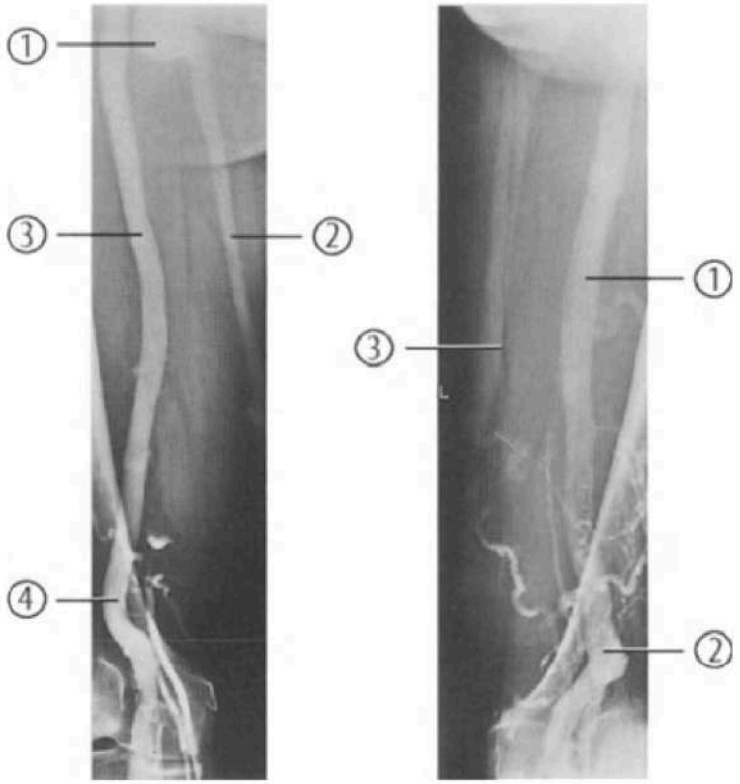
10-22

Fig. 10-22. Phlebography showing a stage III varicosity of the greater saphenous vein, extending to the calf and also involving some of the accessory veins (stage III: incompetent valves extending to one handbreadth below the knee).
① Greater saphenous vein



10-23

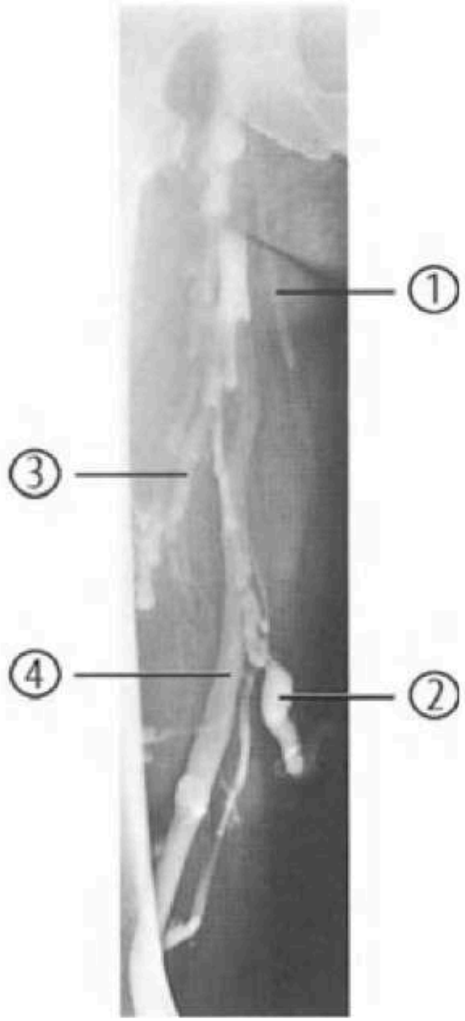
Fig. 10-23. Phlebography showing a stage III varicosity of the greater saphenous vein with involvement of the accessory veins.
① Greater saphenous vein



10-24 10-25

Fig. 10-24. Phlebography showing massive varicosities of the greater saphenous vein with aneurysmal enlargement in the proximal thigh.
① aneurysm of the greater saphenous vein; ② greater saphenous vein; ③ superficial femoral vein; ④ popliteal vein

Fig. 10-25. Phlebography showing complete stage IV varicosities of the greater saphenous vein. The accessory veins are widened and the valves incompetent. These changes in the accessory veins are an integral part of varicose changes; they have been described as atony of the veins.
① atony of the superficial femoral vein; ② atony of the popliteal vein; ③ greater saphenous vein



10-27

Fig. 10-27. Phlebography showing widened perforating veins of the greater saphenous vein. In a complete varicosity, the saphenofemoral junction valves are incompetent. In an incomplete varicosity, the problem begins with the perforating veins and accessory veins. Here the perforator is in the middle of the thigh and is a DODD vein.

① Greater saphenous vein
② Dilated DODD perforating vein
③ Deep femoral vein
④ Superficial femoral vein

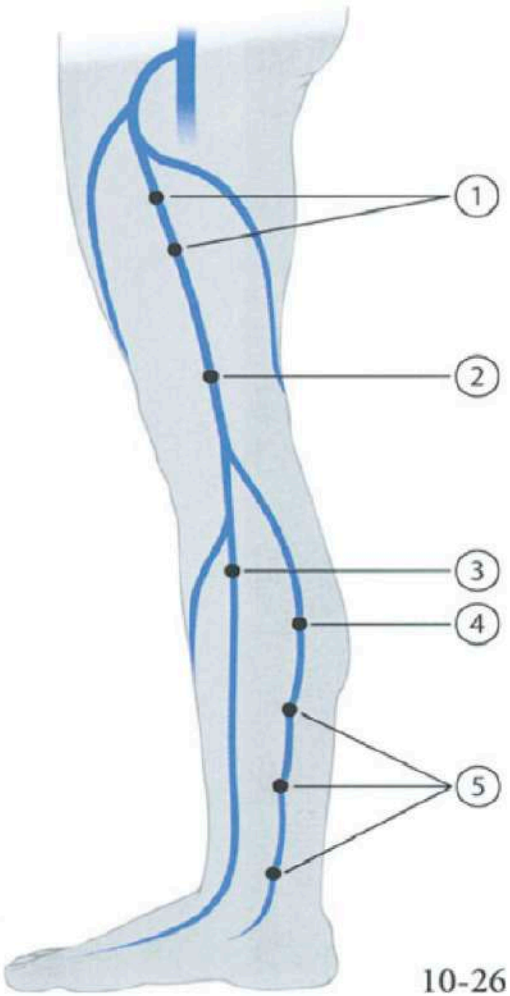
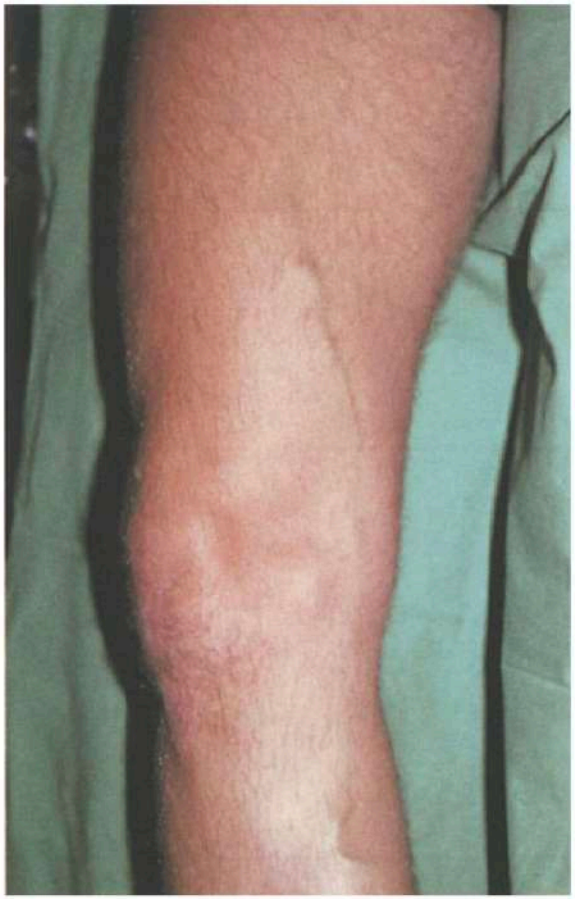


Fig. 10-26. The clinically important perforating veins on the medial aspect of the leg. The perforating veins are the veins that connect the deep and superficial veins, passing through the fascia. There are more than 100 perforating veins in each leg. In a healthy leg, the flow goes from the superficial to the deep venous system. (After HACH-WUNDERLE 1997)
① DODD veins, ② HUNTER vein, ③ BOYD veins, ④ SHERMAN vein, ⑤ COCKETT veins

Figs. 10-28 – 10-30. Various degrees of severity of incomplete varicosity of the greater saphenous vein with DODD perforating veins



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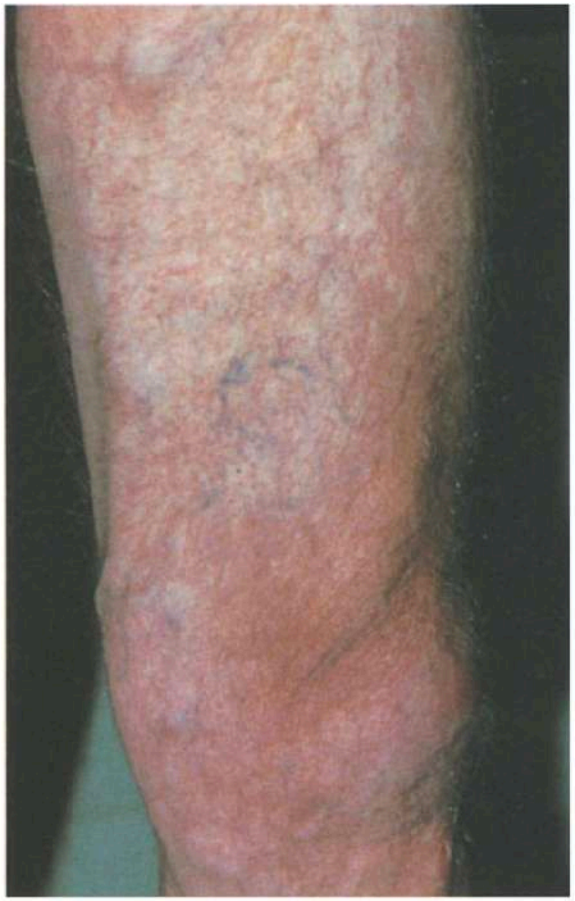

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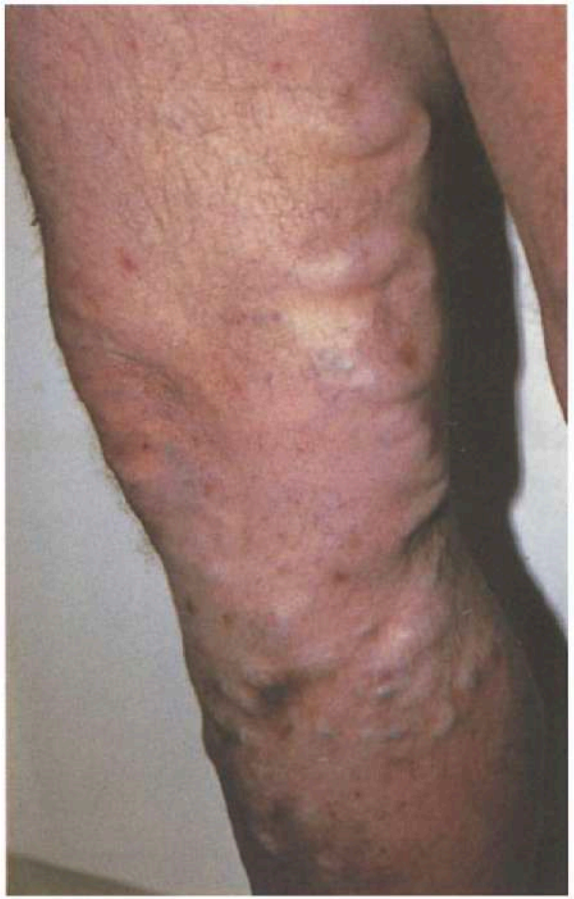
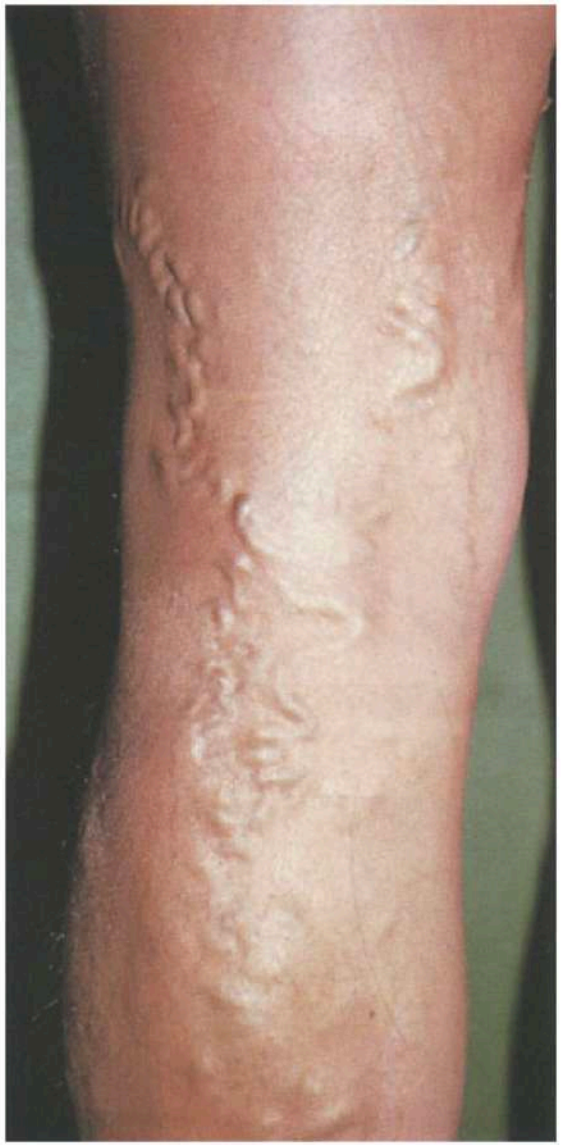


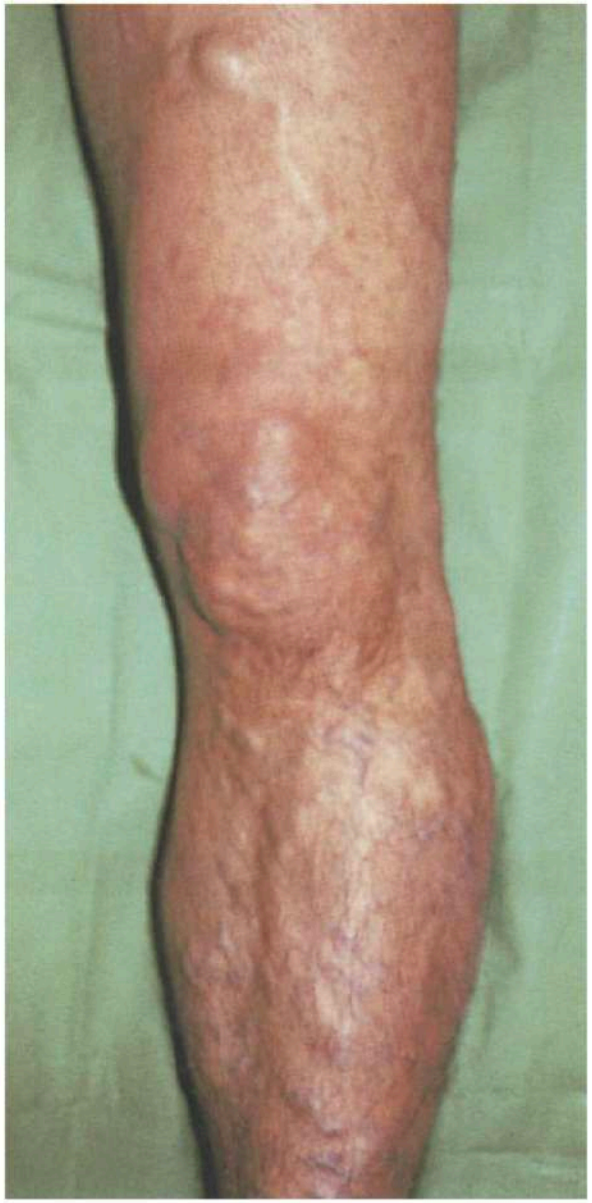
10-30

Fig. 10-31. Incompetent HUNTER vein with varicosities of the accessory veins following removal of the greater saphenous vein. The distal perforating veins of the DOFF group, are found at the end of the adductor or HUNTER's canal (JOHN HUNTER, 1728–1793, British anatomist and surgeon) and are thus called HUNTER veins



10-31

Figs. 10-32, 10-33. Atypical course of an incomplete varicosity of the greater saphenous vein with a blow-out aneurysm on the anterior surface of the thigh



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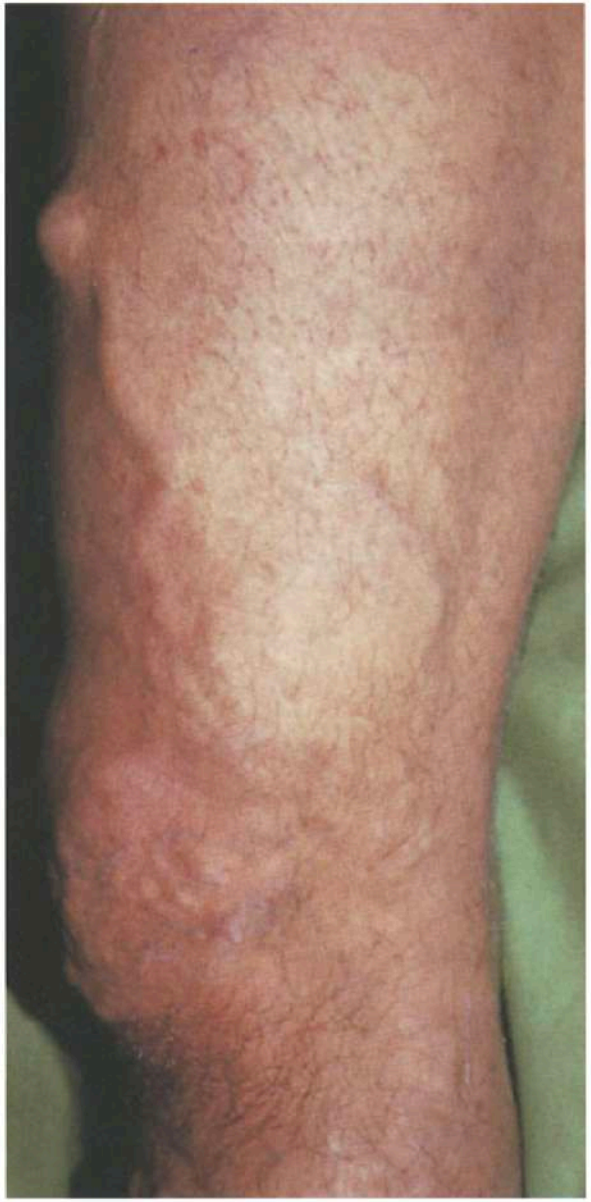

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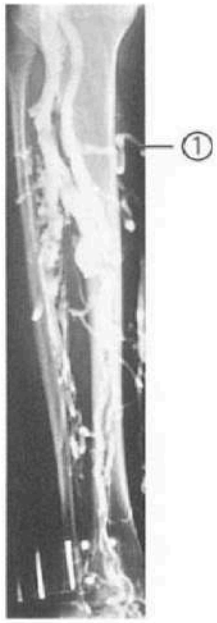

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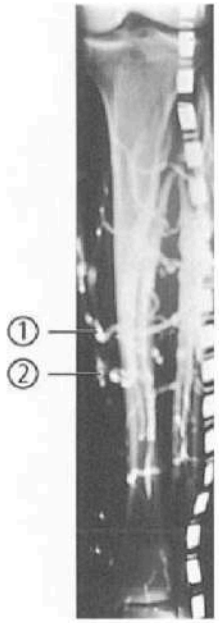
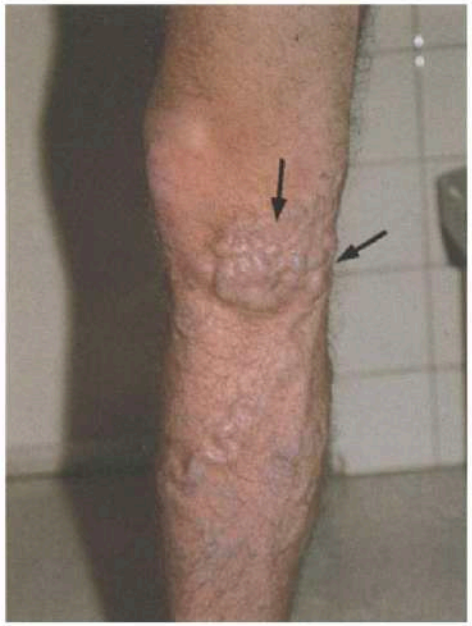


10-35

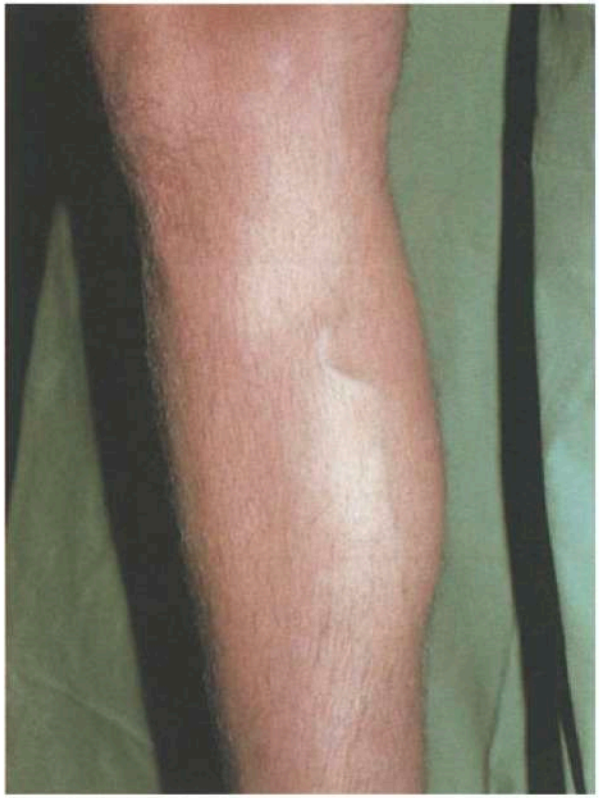
Fig. 10-34. Phlebography showing incompetent perforating vein on the medial aspect of the lower leg and ankle ①. The BOYD (A.M. BOYD, contemporary British phlebologist) perforating veins connect the greater saphenous vein with the posterior tibial veins at the level of the tibial tuberosity

Fig. 10-35. Phlebography showing the COCKETT veins along LINTON's line. They connect the dorsal venous arch with the posterior tibial veins.
①, ② COCKETT perforating veins



10-36

Fig. 10-36. Clinical appearance of an incompetent BOYD perforating vein with varicosities of the accessory veins



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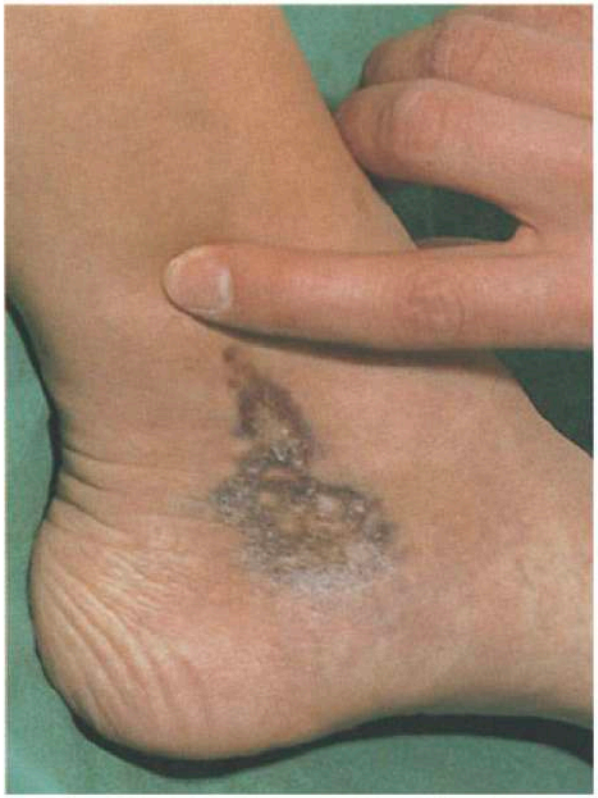
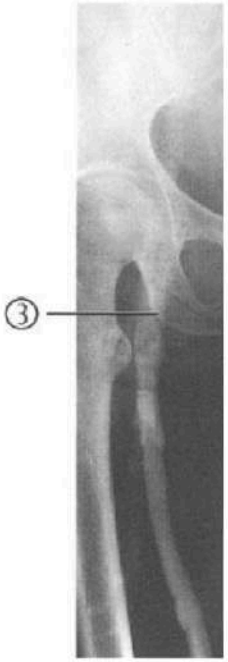


10-38

Fig. 10-37. Moderate incompetence of the SHERMAN perforating vein

Fig. 10-38. Incompetent COCKETT veins with signs of chronic venous insufficiency over the medial malleolus, including hyperpigmentation and focal areas of atrophie blanche. The defect in the fascia is easily palpated with the finger

10-46



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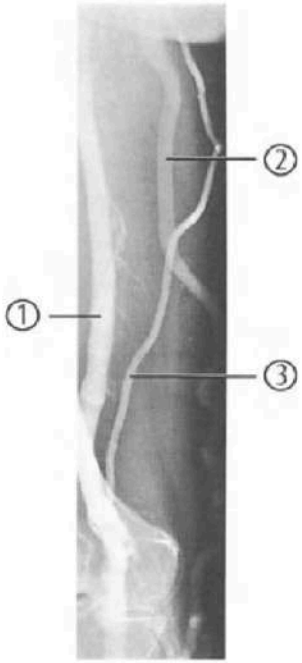


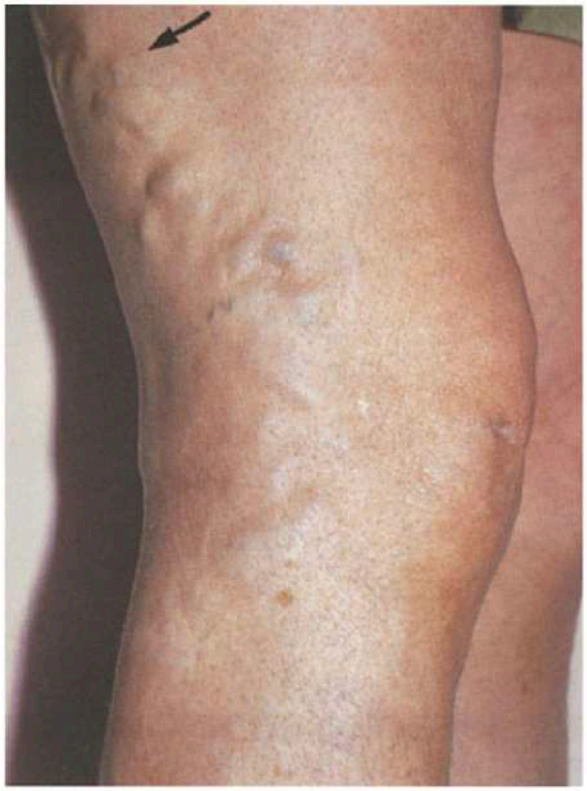
10-48

Figs. 10-46, 10-47. Phlebography showing incomplete greater saphenous vein varicosity of the dorsal type, caused by an incompetent GIACOMINI anastomosis. Varicosities of the medial accessory saphenous vein are often associated with incompetent femoro-popliteal vein and their GIACOMINI anastomosis. In this way, connections between varicosities of the greater saphenous vein and lesser saphenous vein can develop.

① Proximal level of incompetent valves
② Giacomini anastomosis
③ Intact sapheno-femoral junction

Fig. 10-48. Phlebography showing complete greater saphenous vein varicosity of the dorsal type, caused by an incompetent GIACOMINI anastomosis.

① Superficial femoral vein
② Greater saphenous vein
③ GIACOMINI anastomosis



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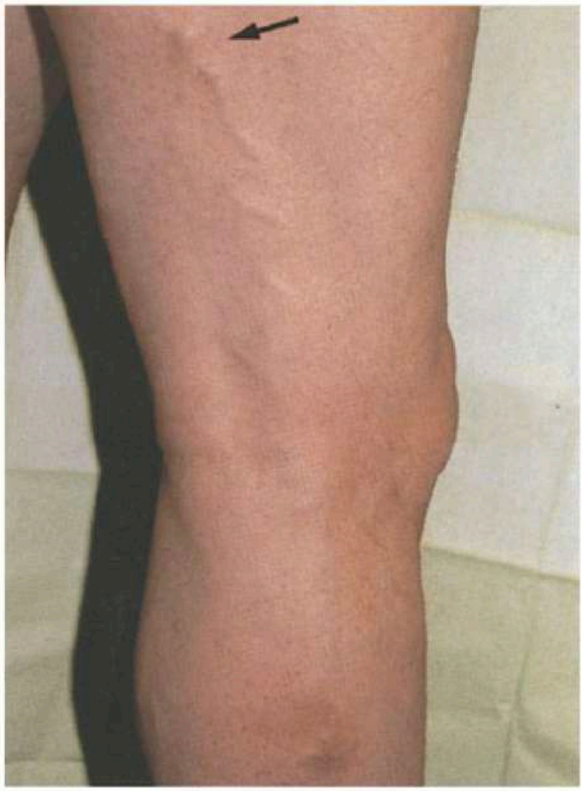
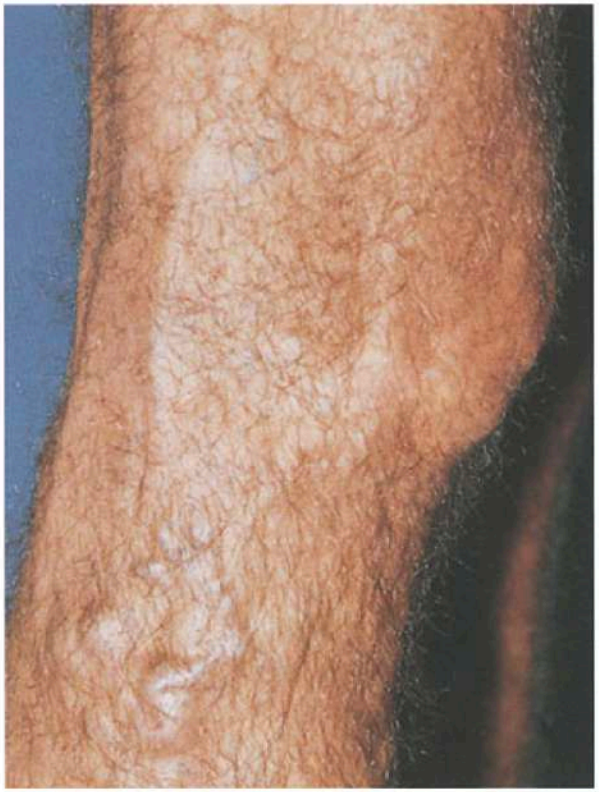


10-50

Figs. 10-49, 10-50. Varicosities of the deep perforating veins (after HACH). Because of the incompetent perforating veins to the deep femoral artery, varicosities develop on the lateral surface of the proximal thigh. The fascial defect is easily seen (*arrows*)

Figs. 10-55, 10-56. Clinical appearance of persisting marginal veins in a 33 year old man. Some persisting marginal veins (lateral posterior veins) are associated with KLIPPEL-TRENAUNAY syndrome. Other such veins drain directly into the superior gluteal veins and thus into the pelvic veins



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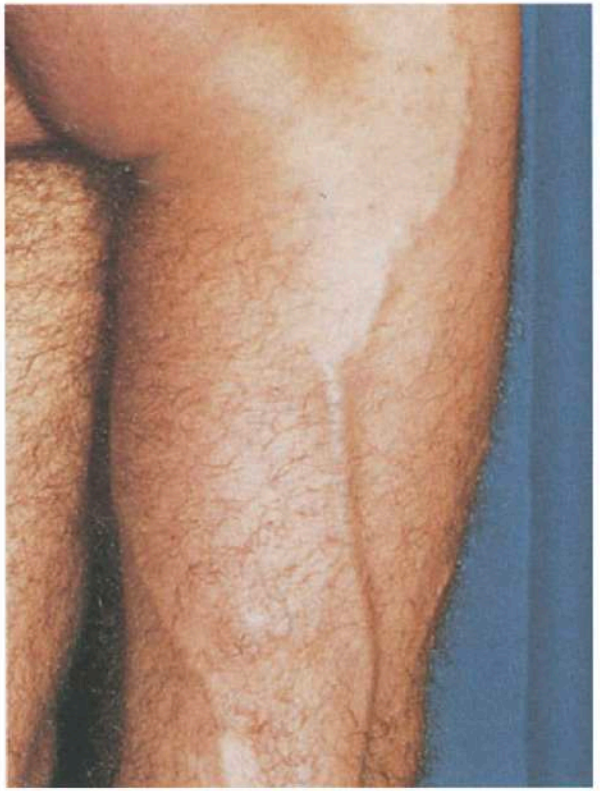

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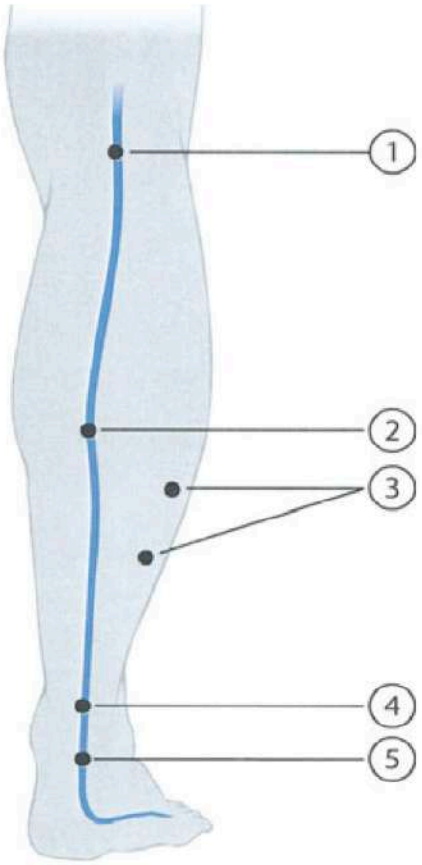

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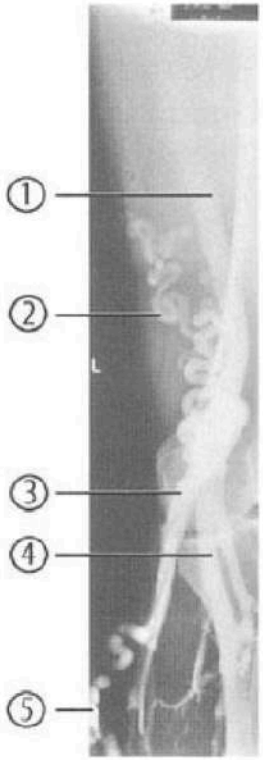

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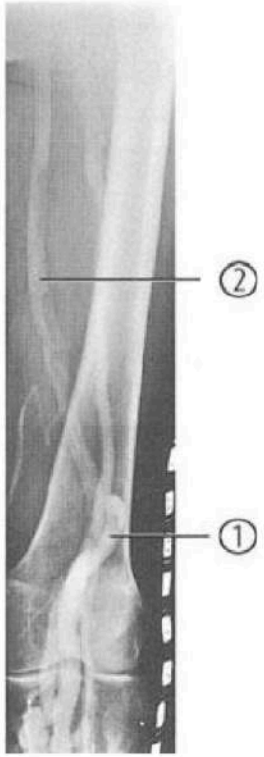
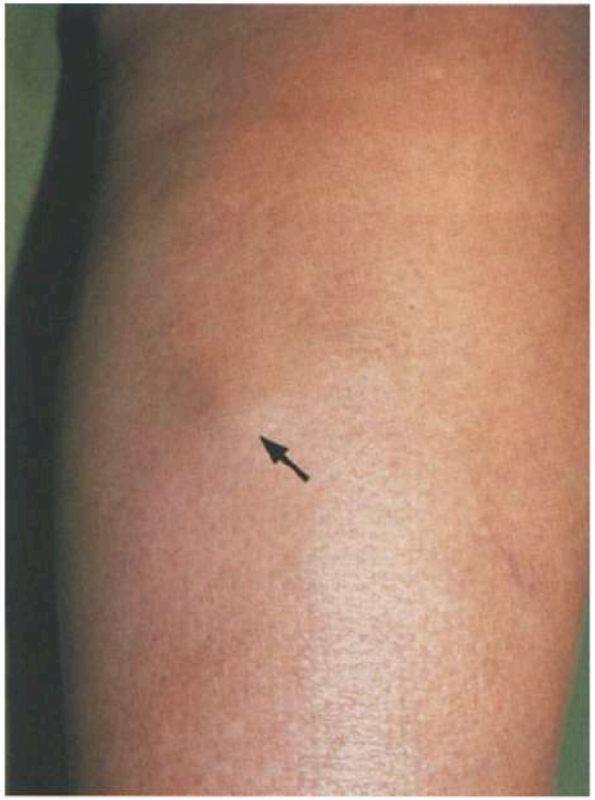


10-59

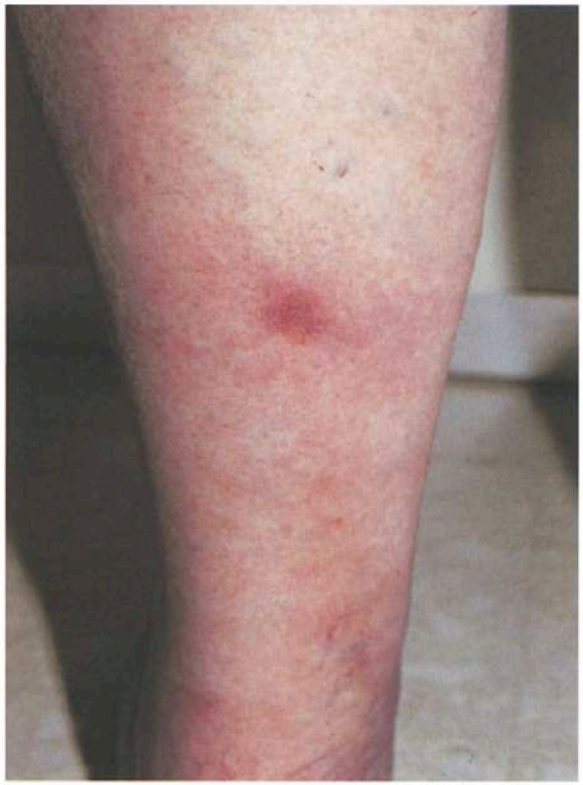
Fig. 10-57. The lesser saphenous vein and the clinically important perforating veins on the latero-dorsal aspect of the calf (after HACH and HACH-WUNDERLE 1997). The MAY (ROBERT MAY, 1914 – 1984, Austrian phlebologist) perforating vein connects the lesser saphenous vein and the gastrocnemius veins in the middle of the calf. The connections between the lesser saphenous vein and peroneal veins occur at the level of the lateral malleolus as BASSI (GLAUCO BASSI, born 1914, Italian phlebologist) perforating veins at 9 cm, as well as at 12 cm.

① popliteal perforating vein;
② May perforating vein;
③ lateral perforating vein;
④ 12 cm perforating vein;
⑤ Bassi perforating vein (9 cm)

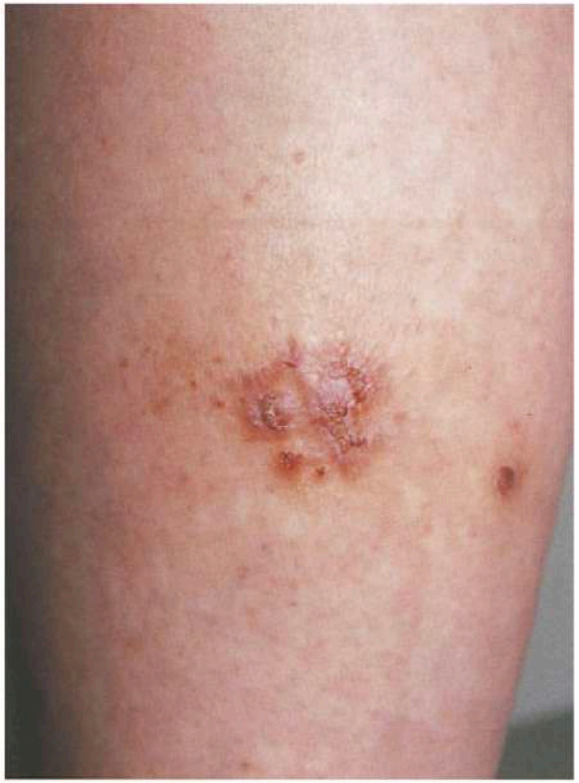


10-64

Fig. 10-64. Incompetent perforating vein of the calf with blow-out (*arrow*) that is easily seen



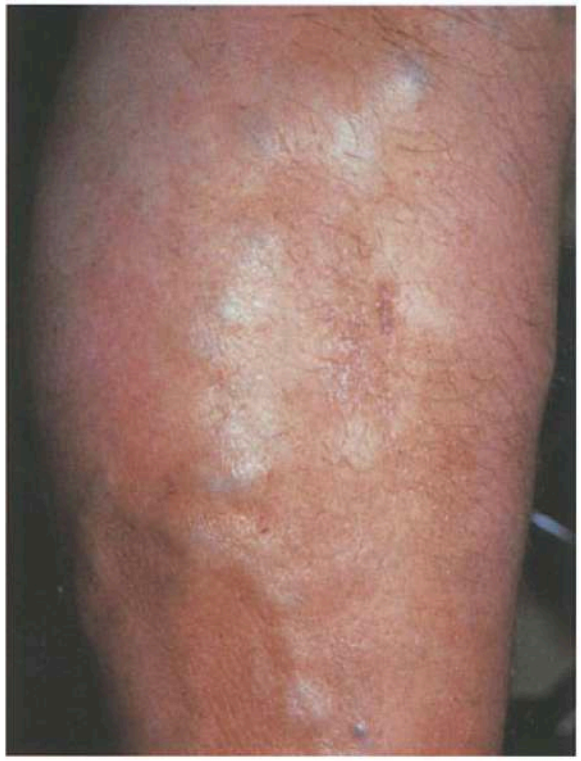
10-65



10-66

Fig. 10-65. Thrombosis of a perforating vein with local phlebitis at the pressure point of the soleus veins

Fig. 10-66. A focal area of chronic dermatitis directly over an incompetent perforating vein of the calf



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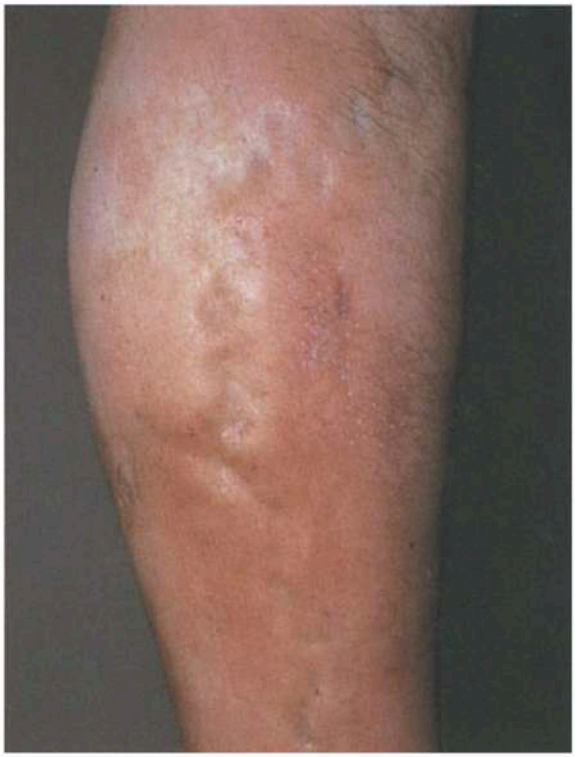


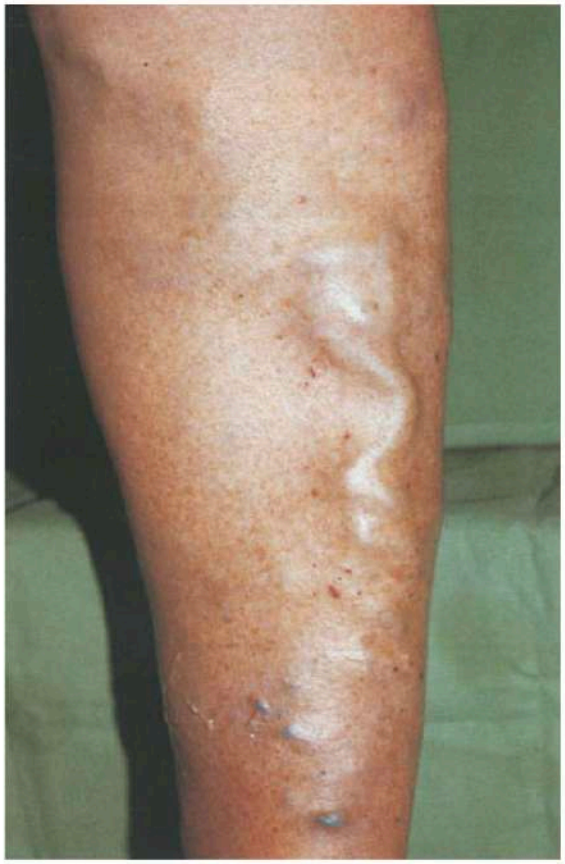
10-68

Fig. 10-67. Filled canyon varicosity

Fig. 10-68. Empty canyon varicosity, showing the derivation of the name: A canyon varicosity is a thin-walled superficial vein which makes a dent or canyon in the sclerotic dermis associated with chronic venous insufficiency. They are difficult to treat, as both surgery and sclerotherapy yield poor results

Fig. 10-69. Pretibial varicosity, involving the dorsal venous arch, an accessory vein of the greater saphenous vein. These varicosities are prominent over the tibia and often painful or easily injured

Fig. 10-70. Ruptured varicosity with bleeding is a feared complication for patients with varicose veins. Here is a bleeding lesion following injury to the tibial surface. Often the bleeding does not stop spontaneously and a firm compression bandage and elevation of the leg are required



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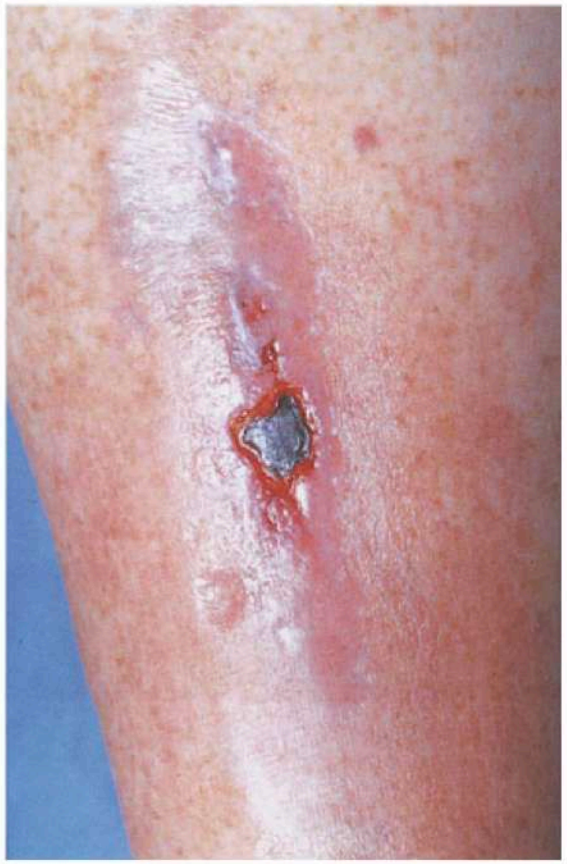
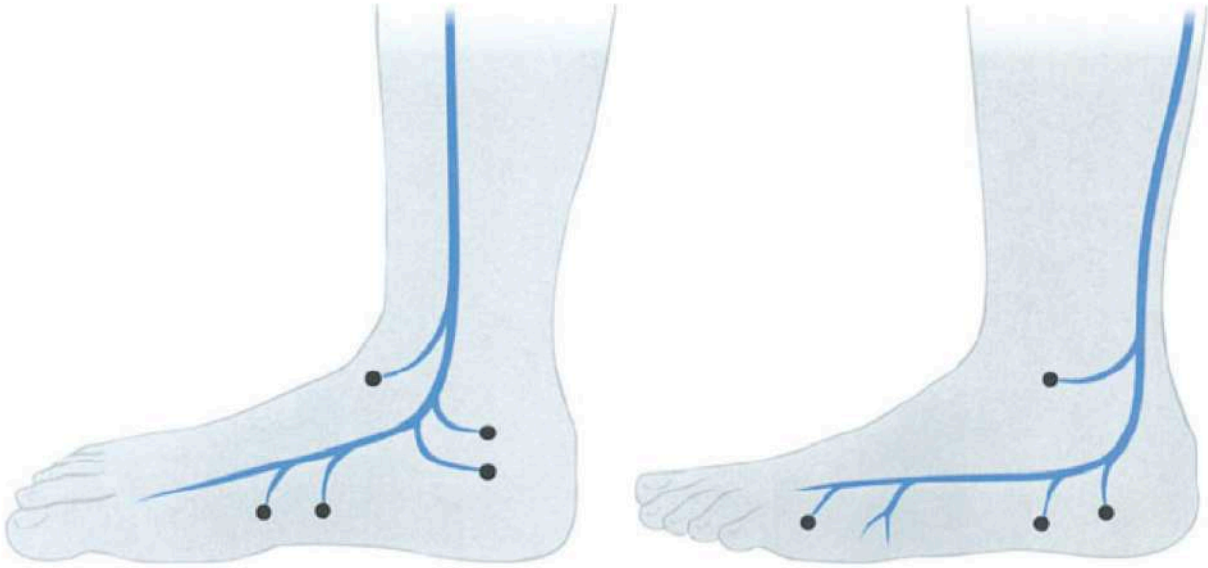


10-70

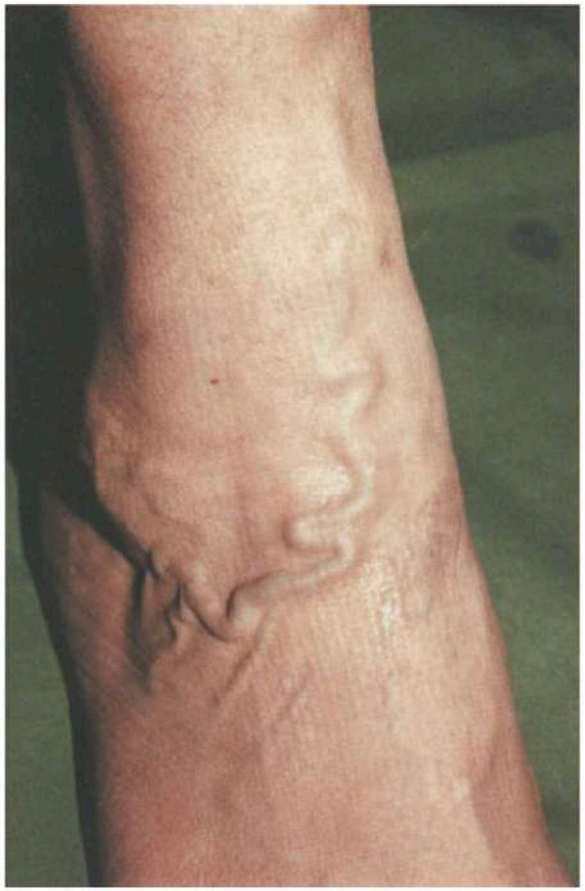
Fig. 10-71. Perforating veins about the ankle. The KUSTER perforating veins are found about the medial and lateral malleoli and along the edge of the foot. (After HACH und HACH-WUNDERLE 1997)



10-71

Fig. 10-72. Varicosity of accessory vein on dorsal aspect of ankle with pretibial varicosity

Fig. 10-73. Scratch marks over a distal varicosity of the lesser saphenous vein. In the early stages of stasis dermatitis, pruritus is often a complaint



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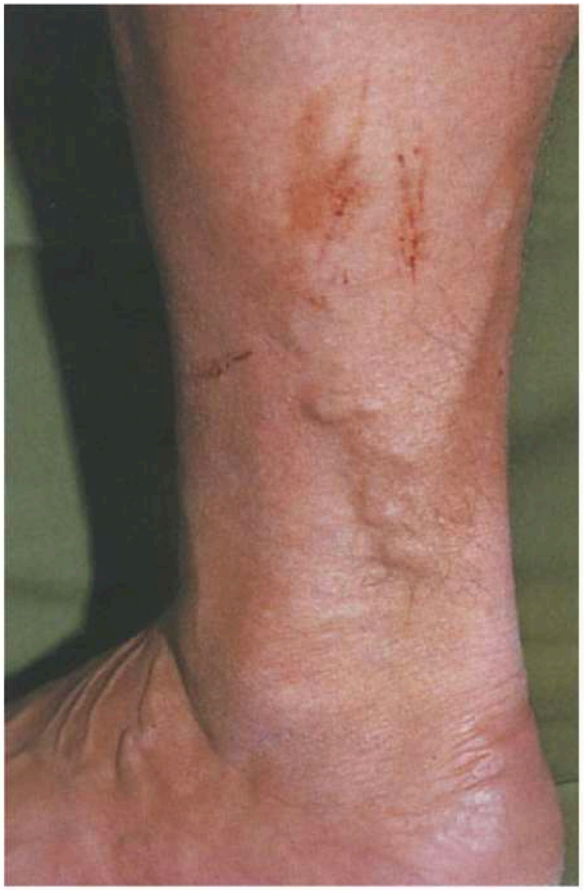

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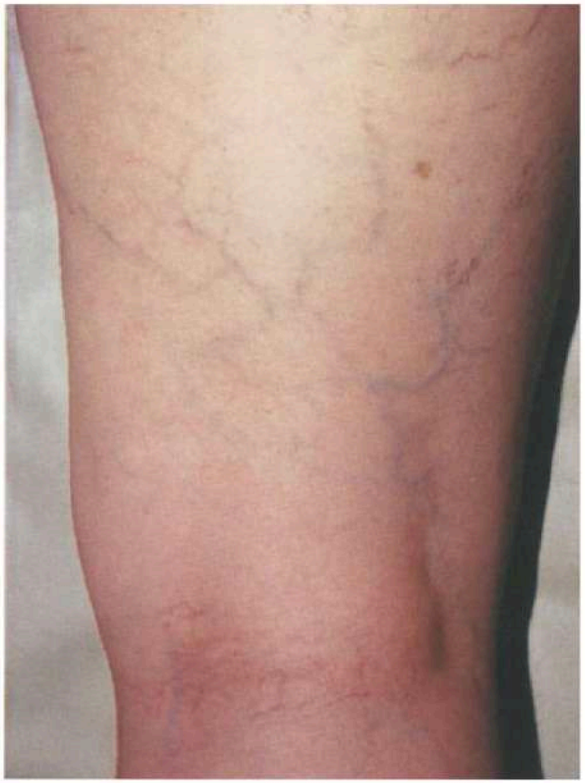
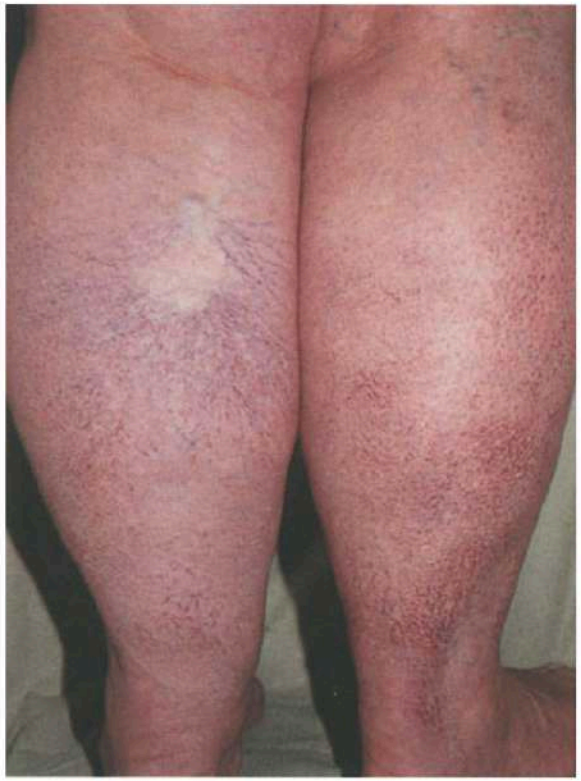


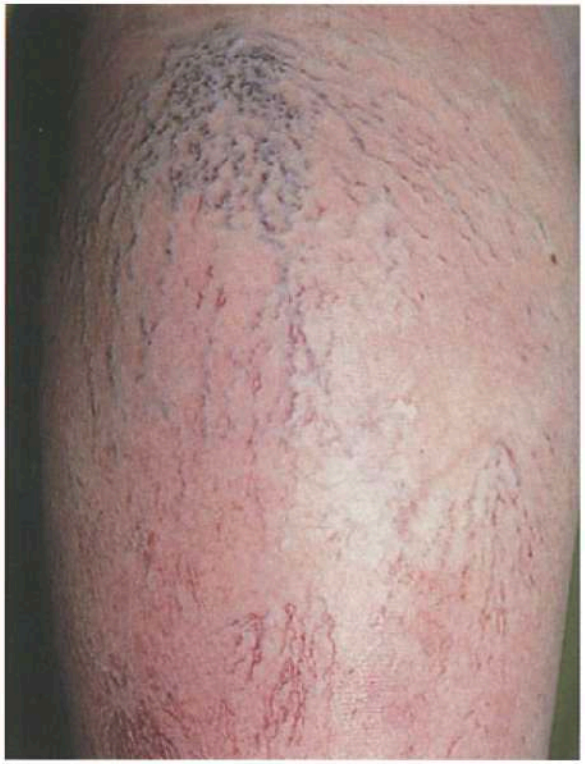
10-74

Fig. 10-74. Reticular varicosities on the dorso-medial aspect of the thigh. These dilated veins lie in the uppermost layers of the subcutaneous layer and show through the skin, with a net-like pattern. They have no direct connection to the infrafascial venous system. Although reticular varicosities have no hemodynamic significance, they are a cosmetic problem. Sclerotherapy is the treatment of choice



10-75

Fig. 10-75. Numerous spider veins on both legs of a 41 year old woman. These lesions are tiny intradermal micro-varicosities. They can be single, multiple or confluent. In extreme cases, wide areas of the skin are covered. Again spider veins have no hemodynamic significance. Some patients complain of pain and heavy legs, especially in the pre-menstrual phase



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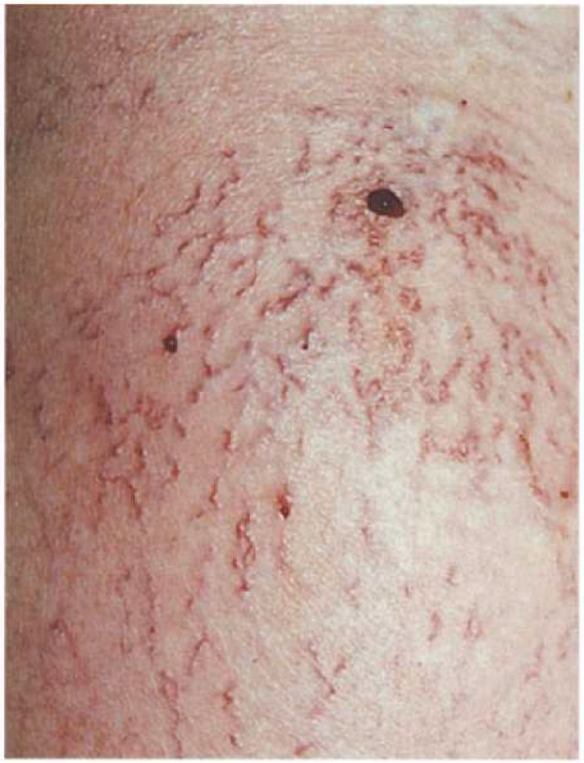
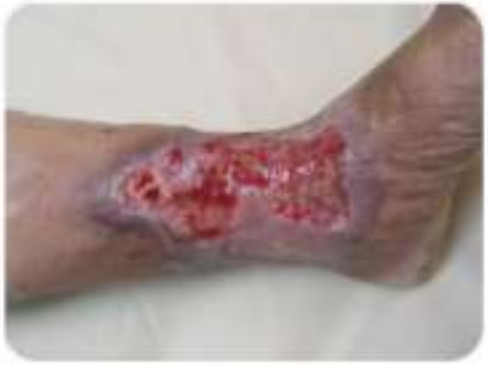


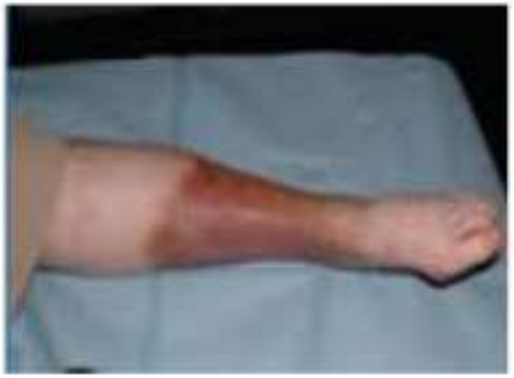
10-77

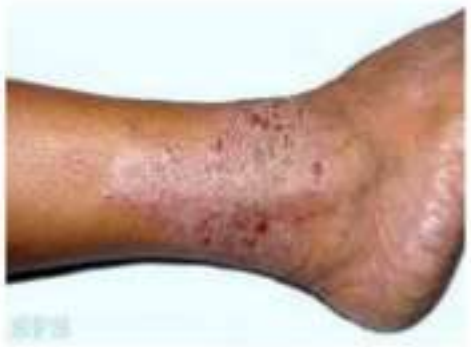
Fig. 10-76. Both red and blue spider veins in the same patient. The blue lesions are best treated with sclerotherapy, while a variety of laser systems seem best for the red variant

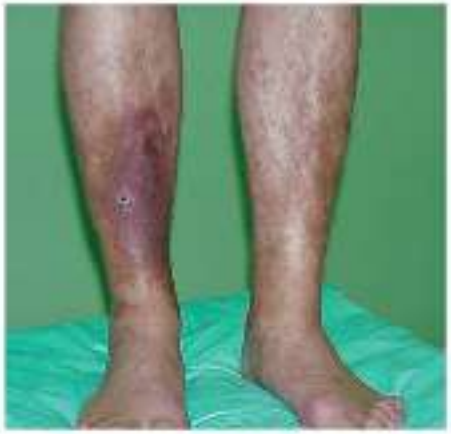
Fig. 10-77. Rupture of a spider vein in the same patient.

Venous disease – Venous hypertension



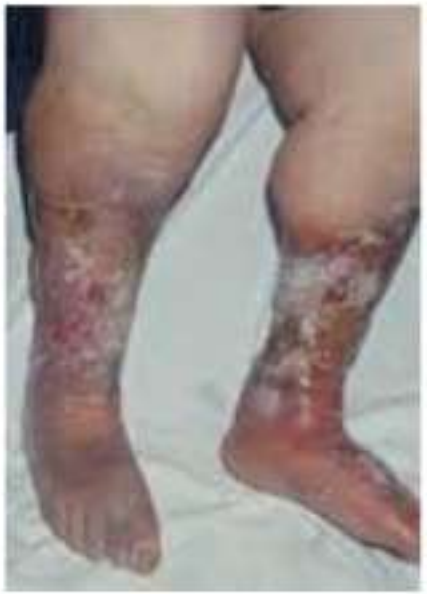






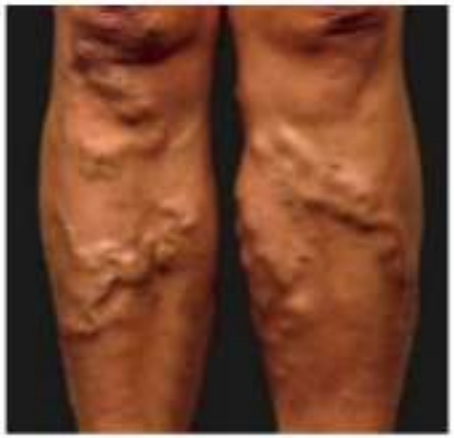
Inspection

- Look for signs of chronic venous disease:
- ankle oedema
- brown pigmentation / venous eczema
- “inverted champagne bottle leg”
- pale patches / ulceration
- “cayenne pepper” petechiae



Varicose Veins

- Site, extent and size of veins
- Best examined with patient standing



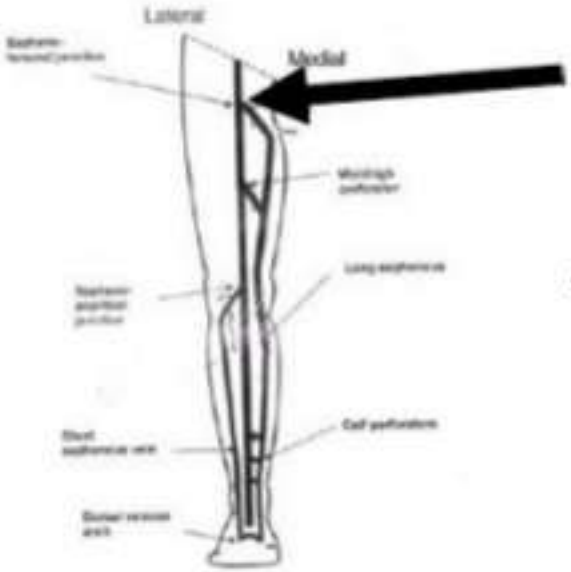
Palpation

- Skin texture for lipodermatosclerosis
- Heat and tenderness of superficial thrombophlebitis
- Gently press on the varicosities and release to watch them refill.
- This confirms they are vascular.
- (Hard veins ~ thrombosis - Painful veins ~ phlebitis
- Use the back of your hand to feel the area around the varicosities. Varicosities are warm.

Saphenofemoral Junction

Ask patient to lie down.

•Find the femoral pulse (midway between the ASIS and the pubic tubercle).



•The SFJ is located approx 2cm medial and 2cm inferior to the femoral pulse.

Tests for incompetencies

Tap Test

- Ask the patient to stand up.
- Place one hand on the SFJ, the other on the
- varicosities.
- Tap the SFJ and feel for a thrill over the varicosities
- If a thrill is felt, it means there is backflow between the
- SFJ and the varicosities (i.e. incompetent valves).

Cough Test

- Ask the patient to stand. Place fingers over SFJ and ask the patient to cough. If a thrill is felt, it suggests incompetence.

Trendelenberg/Tourniquet test

- Empty superficial veins by raising the leg with patient lying flat
- Apply tourniquet high in the thigh
- Ask the patient to stand
- If the veins below tourniquet fill up, this implies that the incompetent perforators are below that level
- If veins remain empty, perforator is above that level

Perforators/level of incompetencies

- Perforators connect deep and superficial systems, allowing the superficial blood to flow into the deep veins where the muscle pump helps to aid venous return
- Perforator valve incompetence lead to increased pressure within veins, which is the usual underlying abnormality

1. Sapeno-femoral junction – extensive vv over medical calf and thigh, sometimes with saphena varix
2. Mid thigh perforator (Hunter's vein) – vv over medial calf
3. Short sapheno-popliteal junction – vv over lateral calf
4. Medial calf perforators (Cockett's veins) – vv over medial calf

Treatment

Aimed at relieving the swelling and decreasing the pressure in the veins,

- **Leg elevation** — above heart level for 30 minutes 3-4 four times per day can reduce edema and improve blood flow in the veins.

- **Exercises** — Foot and ankle exercises are often recommended. Pointing the toes and feet down and up several times throughout the day can help to pump blood up into the legs.

- **Compression Therapy** - Compression stockings gently compress the legs, improve blood flow in the veins by preventing backward flow through the veins of the legs.

- **Dressings —** Ulcers are usually covered with special dressings before putting on compression stockings or compression bandages. Dressings are used to absorb fluid oozing out of the wound, reduce pain, control odor, remove dead or infected cells, and help new skin cells to grow.

- **Compression bandages —** These are multilayer wraps placed on the leg after any ulcers are covered. The bandages are usually changed once or twice a week and must stay dry. The bandages should be removed and replaced if they become wet, either from water or from wound drainage.

Surgical therapy

- involves extensive ligation and stripping of the greater and lesser saphenous veins
- reserved for patients who are very symptomatic, suffer recurrent superficial vein thrombosis, and/or develop skin ulceration
- may also be indicated for cosmetic reasons.

Surgery

Vein ablation treatments are treatments designed to destroy superficial veins with abnormal valve function.

- Sclerotherapy — Sclerotherapy is a chemical ablation in which a substance is injected into the vein, causing the vein to collapse.

- Radiofrequency or laser ablation — Radiofrequency and laser ablation involve putting a special device into the abnormal vein. High energy is applied, which destroys the vein from the inside. The vein is not removed but it no longer functions.

- Vein ligation/stripping — Vein ligation or stripping are surgical are removed with many small incisions.

NB Compression stockings are generally recommended following these ablation procedures

Medications

- Diuretics - Venous disease is caused by a problem in the veins of the legs rather than an overload of fluid throughout the body but patients with severe edema) may sometimes benefit from a few days of diuretics.

- Aspirin (300 to 325 mg/day) may speed the healing of ulcers.

- Antibiotics are only recommended when there is an infection.

- Stasis dermatitis (irritation of skin resulting from swelling, pooled blood and increased pressure in the veins) usually responds to moisturisers. Sometimes, a steroid cream or ointment is needed to help with itching.

- Scented lotions should be avoided because there is a risk of developing an allergic rash (contact dermatitis).

- Treatment of contact dermatitis — Contact dermatitis is common in people with chronic venous disease treated with topical therapy.

- **CHRONIC VENOUS INSUFFICIENCY**

  - - may result from deep vein thrombosis and/or valvular incompetence
  - - often complain of a dull ache in the leg that worsens with prolonged standing and resolves with leg elevation
  - - PE demonstrates increased leg circumference, edema, and superficial varicose veins
  - - erythema, dermatitis, and hyperpigmentation develop along the distal aspect of the leg
  - - skin ulceration may occur near the medial and lateral malleoli
  - - Cellulitis may be a recurring problem

LYMPHATIC DISORDERS

- lymphatic circulation is involved in the absorption of interstitial fluid and in the response to infection

LYMPHEDEMA

- prevalence of primary lymphedema is approximately 1 per 10,000 individuals
- Primary lymphedema
    - secondary to agenesis, hypoplasia, or obstruction of the lymphatic vessels
    - may be associated with Turner syndrome, Klinefelter syndrome, Noonan syndrome, the yellow nail syndrome, the intestinal lymphangiectasia syndrome, and lymphangiomyomatosis
    - women are affected more frequently than men
    - Three clinical subtypes:
        - » Congenital lymphedema (Milroy's disease) - which appears shortly after birth
        - » lymphedema praecox (Meige's disease) - which has its onset at the time of puberty
        - » lymphedema tarda - which usually begins after age 35

- Secondary lymphedema - resulting from damage to or obstruction of previously normal lymphatic channels
- Filariasis - most common cause of secondary lymphedema
- generally a painless condition, but patients may experience a chronic dull, heavy sensation in the leg, and most often they are concerned about the appearance of the leg
- gradually progresses up the leg so that the entire limb becomes edematous
- evaluation should include diagnostic studies to clarify the cause
- Abdominal and pelvic ultrasound and computed tomography can be used to detect obstructing lesions such as neoplasms

- MRI may reveal edema in the epifascial compartment and identify lymph nodes and enlarged lymphatic channels
- Lymphoscintigraphy and lymphangiography - rarely indicated
- can be used to confirm the diagnosis or to differentiate primary from secondary lymphedema.
- Primary lymphedema, lymphatic channels are absent, hypoplastic, or ecstatic
- Secondary lymphedema, lymphatic channels are usually dilated, and it may be possible to determine the level of obstruction.

TREATMENT

- must be instructed to take meticulous care of their feet to prevent recurrent lymphangitis
- Skin hygiene is important, and emollients can be used to prevent drying
- Prophylactic antibiotics are often helpful
- should be encouraged to participate in physical activity
- frequent leg elevation can reduce the amount of edema
- Physical therapy - to facilitate lymphatic drainage
- graduated compression hose to reduce the amount of lymphedema that develops with upright posture
- Diuretics are contraindicated
- Recently, microsurgical lymphatico-venous anastomotic procedures have been performed to rechannel lymph flow from obstructed lymphatic vessels into the venous system.