Assessment and Management of Lower Extremity Vascular Disease

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The Role of the Nurse Practitioner

- As the population ages, nurse practitioners will play a vital role in primary care management of modifiable risk factors and early detection of lower extremity vascular disease.
- Aggressive risk factor modification and early detection along with an on-going established therapeutic relationship with the NP will assist patients in risk-factor avoidance and modification



Role of the Nurse Practitioner

- Early diagnosis and treatment
- Modify risk factors
- Management chronic disease
- Prevent complications, amputations

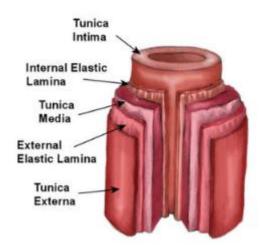


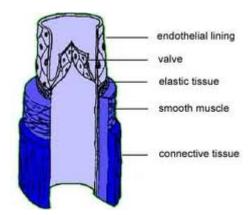


Incidence and Etiology

Arterial Disease

Venous Disease







Differentiation between arterial and venous disease

- Characteristics of arterial
 - Pain (walking or at rest)
 - Foot cool or cold
 - Weak or absent pulses
 - Absence of leg hair
 - Skin shiny, dry, pale
 - Thickened toenails
 - Ulcer location: below ankle
 - ABI less than .5 (note is diabetic it can be greater than 1.0)
 - History of DM, Hypertension, smoking, Claudication
 - History of foot trauma.

- Characteristic of venous disease
 - Foot Warm
 - Edema
 - Brawny skin pigment changes
 - Varicose veins
 - Ulcer location, usually above ankle (medial malleolus)
 - Not painful
 - 100% Granulated, but with drainage.
 - History of trauma, deep vein thrombosis.

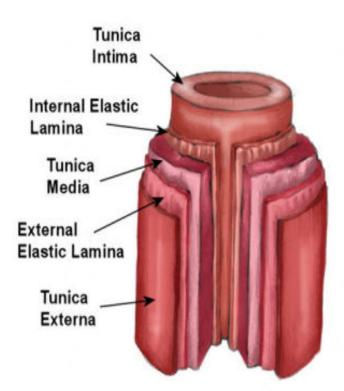
Etiology

 Basis of most arterial diseases can be considered atherosclerosis Atherosclerosis was defined by the World Health Organization as a combination of changes in the intima and media. These changes include focal accumulation of lipids, hemorrhage, fibrous tissues, and calcium deposits



Arterial Disease

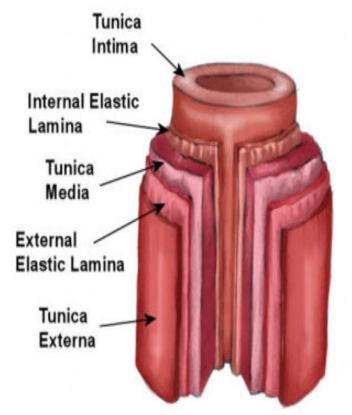
- Arterial disease is the leading cause of death and significant morbidity in the United States and throughout the world
- 36.3% of Americans have cardiovascular disease
- 864,500 deaths annually





Arterial Disease

- Tunica Intima the innermost layer; monolayer of endothelial cells with a thin underlying matrix of collagen and elastic fibers
- Tunica Media relatively thick middle layer of varying amounts of smooth muscle, collagen, and elastic fibers
- **Tunica Externa** outermost layer of artery. Key element in the total strength of the arterial wall.





Atherosclerosis

Atheroma (Greek):
 "gruel" or "porridge"

 Sclerosis (Greek) – "hardening" or "induration"



Atherosclerosis

- Complex process linked to a combination of factors
- These include genetic predisposition (e.g. familiar hyperlipidemia and diabetes), mechanical factors (e.g. hypertension and shear stress), environmental factors (e.g. tobacco and fatty food intake)



Theories of Atherogenesis

- Response to injury hypothesis
- Lipid hypothesis
- Thrombogenic hypothesis

- Mesenchymal hypothesis
- Monoclonal hypothesis



Pathophysiology of PAD

- Decreased delivery of oxygen
- Tissue ischemia
- Tissue loss
- Decreased ability to fight infection
- Decreased healing



Clinical Manifestations of Peripheral Arterial Disease (PAD)

- Major cause of acute and chronic illness
- Associated with decrements in functional capacity and quality of life
- Cause limb amputation
- Increase risk of death



Assessment



Chief Complaint/Introduction

- Noninvasive vascular testing has been use by the clinician as an indicator for arterial occlusive or venous disease.
- These types of quantitative test are use to evaluate the healing potential of ulcers wounds , an as aid in determining wound care management plans,
- These types of test are used as guides in determining which patients need referral to a vascular surgeon for further evaluation.
- The first step in a vascular evaluation is to obtain a complete medical history from the patient. The patient history provides a guide in determining the etiology of wound.
- They should include the following.

- Chief complaint is the combination of symptoms that made the patient visit your office. Focus on the symptoms that concern the patient and then ask the patient to describe hose symptoms.
- A detailed pain history is essential in determining the urgency and type of treatment the patient will receive. Investigate the type of pain, spending time listening to the patient is as important as the physical examination.
- When and where the pain starts and goes.
- What factors aggravate or relive the pain:
 - Elevation?
 - Dependency?
 - Walking?
 - Resting?
 - Standing?

Diagnosis of PAD



ADA Consensus Statement

- Screening should include history & physical exam
- An ankle brachial index
- Treatment should include:
 - Smoking cessation
 - Antiplatelet therapy
 - Lowering blood glucose (HgbA1-C < 7.0)
 - Lowering blood pressure (< 130/80 mmHg)
 - LDL cholesterol < 100 mg</p>



ADA Consensus Statement

- Issued by the American diabetes Association (ADA) November 21, 2003
- Recommends screening for any diabetic patient over age 50
- Additionally any patient with diabetes who is younger than 50 should be considered for screening if they have other risk factors:
 - Smoking
 - High blood pressure
 - High cholesterol
 - Diagnosis of diabetes > 10 years



Vascular Review of systems

- Any exertional limitation of the lower extremity muscles or any history of walking impairment?
- Any poorly healing or nonhealing wounds of the legs or feet?
- Any pain at rest localized to the lower leg or foot and is association with the upright or recumbent positions.



History and Physical

- Measurement of blood pressure in both arms and notation of any interim asymmetry
- Palpation of carotid pulses and notation of any bruits
- Auscultation of abdomen and flank for bruits

- Palpation of abdomen and notation of aortic pulsation
- Palpation of pulses at the brachia, radial, ulnar, femoral, popliteal, dorsalis pedis, and posterior tibial sites.
- Auscultation of both femoral arteries for the presence of bruits



History and Physical

- Remove shoes and socks and inspect the feet. Note color, temperature, integrity of the skin and intertriginous areas evaluated, and the presence of ulcerations
- Additional findings suggestive of severe PAD, including distal hair loss, trophic skin changes, and hypertrophic nails.



Past Medical History/ Physical exam

- Risk factors for PVD
 - Cardiac History
 - Heart disease (cardiac catherization? Results)
 - Heart attack
 - Stroke
 - Chest pain

Hypertension (severity, medications, age at onset, highest BpHyperlipedemia (LDL, AGE)Smoking history

Diabetes (number of years)

- Risk factors for venous disease
 - Trauma (type, date)
 - Deep vain thrombosis (date, thinners)
 - Prolonged inactivity
 - Pregnancies
 - Family history of venous disease
 - Obesity (FAT)
 - Clotting Disorders (Sickle cell)

Past surgical history

Vascular surgery (date of procedure) Angiogram/venogram (dates, indication

General surgery (date of procedure

Systemic nature of PAD

- Contributes to development of concomitant disease of the arteries to heart and brain
- Increased risk of cardiovascular ischemic events

- Myocardial infarction (MI)
- Ischemic stroke
- Death



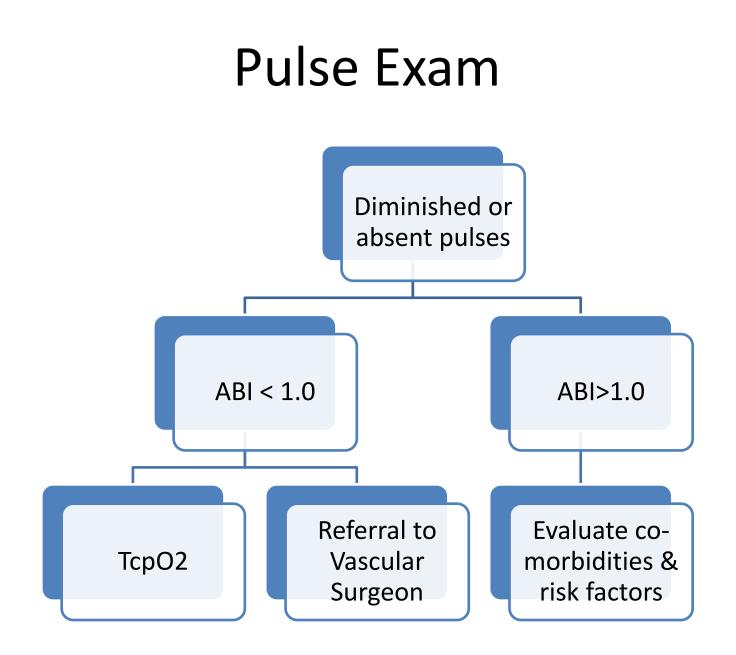
Pulse exam

- The next step in the physical examination is the pulse exam. This will include location and grading of the following.
 - 1. Femoral, Popliteal, Dorsalis pedis, Posterior tibailis
- The grading system is as follows
 - 0 = no pulse
 - 1+ = barely felt
 - 2+ = Diminished
 - 3+ = Normal pulse (easily felt)

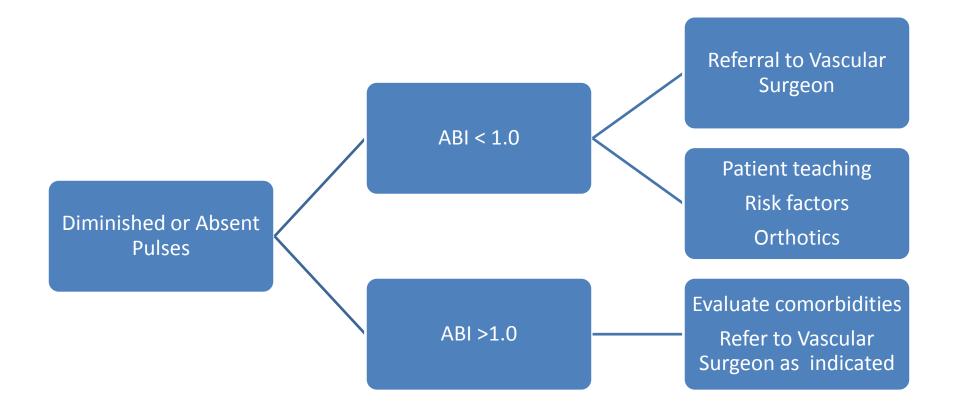
The Pulse Exam

- Monophasic: abnormal; one sound; "Nebraska in Winter"
- Biphasic: abnormal; two sounds. Loss of elastic recoil of artery
- Triphasic: normal, three sounds. Two sounds related to cardiac cycle, additional sound secondary to elastic recoil of artery





Pulse Exam



Tips and tools Severe Arterial insufficiency

- Elevating the leg while the patient is supine will cause the patients leg to become pale. (Elevation pallor), pale color is due to "duh reduce blood flow"
- Hanging the leg over the side of the gurney will make the leg color change to a deep red or purple color. (Dependent rubber) this is due to also reduce blood flow and vasodilatation of the arterioles.
 - Note: Do not confuse severe arterial insufficiency with cellulites, the leg will not become pale with elevation.
- Pain is big indicator, if your patient complains when you simply just look at their wound, then just add all the clues you have been collecting.
- Location of wounds and shape. Arterial insufficiency wounds are located at lower aspects of the foot, below the ankle. The shapes are round.

Signs and Symptoms of PAD

- Thin, atrophic skin
- Dependent rubor
- Pallor with elevation
- Absence of pedal pulses
- Non-healing wounds (cutaneous gangrene & ulceration)
- Muscle wasting
- Hair loss
- Hypertrophic nails



Peripheral arterial occlusive disease

- PAD
- Excludes the functional (vasoreactive) or aneurysmal disorders that affect the noncoronary arteries
- "Lower extremity arterial disease"
- Does not include diseases of the aorta, carotid, upper extremity, or visceral arteries



Rest Pain

- Worse at night
- Usually in forefoot
- "Hanging foot sign"



Associated Symptoms of PAD

- Nocturnal leg cramps
- Edema
- Pain
- Pain typically one level below occlusion

- Exercise-induced leg pain
- Buttock pain
- Rest pain
- Positional-induced pain



Associated conditions with PAD

- Coronary disease
- Carotid artery occlusion
- Abdominal aortic aneurysm
- Congestive heart failure
- COPD
- Hypertension Diabetes mellitus

- End-stage Renal disease
- Hyperparathyroidism
- Hypercholesterolemia & Hypertriglyceridemia
- Coagulopathies
- Homocystenemia



Noninvasive Imaging Techniques



PAD: Diagnostic Tests

- Ankle Brachial Index (ABI)
- Lower extremity arterial study
- Arterial duplex
- Magnetic resonance arteriogram (MRA)
- Arteriogram
- CT angiography



Ankle Brachial Index

- Also known as "ABI"
- "A"nkle pressure/ "B"rachial pressure = "I"ndex (ABI)
- Many clinicians feel that this should now be the "sixth" vital sign



ABI

- A ankle brachial index provides objective information as to the presence and severity of arterial occlusive disease, sometime referred to as a API index.
- All ABI consist of ratios. These ratios are the arterial pressures, speaking in terms of peripheral blood flow to that compared of closer attachments to the pump.
- As you move further away from the heart Bp drops in a linear fashion.

ABI

- There is a strong correlation between decreased distal peripheral blood pressure and ulceration
- These physiologic state of lower blood flow is one of the main etiologic factors to lower limb amputations, chronic non healing wounds, ulcers, and PVD. There is a direct alteration of the immune system, metabolic system, and the nervous system as well.
- The patient becomes compromise to infections. All this leads to a complicated cascade of events to hard to explain cause it's 1:00 AM now k.

Ankle Brachial index procedure

- 1. Lay patient flat.
- 2. Apply BP cuff around the patient's arm
- 3. Apply ultrasound gel
- 4. Hold Doppler probe at a 45 angle and place over brachial pulse.
- 5. Identify the arterial signal and inflate the cuff until the signal disappears.
- 6. Slowly deflate the cuff until the arterial sound returns, the first sound that is hear is the systolic brachial pressure
- 7. Obtain brachial pressure in both arms and record the highest.
- 8. To obtain ankle pressures, place the cuff above the ankle and place Doppler probe over the dorsalis pedis or the posterial tibial artery

ABI= Highest ankle Doppler pressure

Highest brachial Doppler prssure

Significance of Ankle brachial index values

- ABI: of less than .5 referral to a vascular specialist (no compression or surgical debride)
- ABI= .5-0.8 (referral to a vascular specialist. Intermittent claudication means occlusive disease.
- ABI= .8-1.0 (mild PVD compression therapy with caution.)
- ABI= of greater than 1.0 if diabetic referral to a vascular specialist, due to calcified vessels false reading.

Risk of PVD with ABI Findings

> Or = 1.0	Normal	No/Low Risk
.9099	Borderline	No/Low Risk
.7089	Mildly Abnormal	Low/Moderate
.5069	Abnormal	Mod/High
< .49	Severely abnormal	High risk



Correlation of ABI's to Severity of Arterial Ischemia

ABI	Clinical Status
.95 or >	Normal
.70 +/- 0.1	Intermittent claudication
.50 +/- 0.1	Rest pain
.30 +/- 0.1	Impending tissue necrosis



Modifiable Risk Factors

- Smoking
- Diabetes
- Hyperlipidemia
- Hypertension
- Metabolic syndrome



Novel Risk Factors: Molecular Biomarkers

- Homocysteine levels
- High-sensitivity Creactive protein
- Fibrin-degradation products

- Microalbuminuria
- Stem cell research



Criteria for Referral to Vascular Surgery

• ABI

- >0.8: routine referral
- 0.6 0.8: semi-urgent referral
- <0.5: urgent referral
- Gangrene present: urgent referral
- Exposed bone or tendon: urgent referral
- Gross infection or cellulitis: urgent referral

- ABI > 1.0 with diminished or absent pulses: semiurgent referral
- Non-healing wounds despite 3+ pulses: semiurgent referral



CLI: Assessment

- Clinical history and exam
- CBC, platelet count, fasting blood glucose, Hgb A1-D, creatinine, fasting lipid profile, urinalysis (for glycosuria and proteinuria)
- Rest EKG

- Ankle or toe pressure measurements (ABI)
- MRA of lower limb arteries to decided endovascular vs open approach
- Duplex scan of carotids
- Cardiac risk stratification



Tissue Ischemia

- Poor venous outflow
- Smoking
- Radiation
- Diabetes
- Vasculitis
- Fibrosis





CLI Assessment

- Pain
- Paralysis
- Paresthesias
- Pulselessness
- Pallor
- Poikylathermia

- Compare to contralateral side
- Capillary refill



CLI: Clinical Presentation

- Limb pain at rest
- Trophic skin changes or tissue loss
- Discomfort worse when supine and may lessen when limb is maintained in dependent position
- Heavy use of narcotics

- Pain disturbs sleep
- Often unable to walk



Critical Limb Ischemia

- Associated with very high intermediate-term morbidity and mortality
- Patients with lower extremity PAD have 3 – 5 times overall greater risk of cardiovascular mortality
- Clinicians must recognize the cardiovascular ischemic burden



Critical Limb Ischemia

- Defined as limb pain that occurs at rest or impending limb loss that is caused by severe compromise of blood flow to the affected extremity
- If left untreated major limb amputation within 6 months

- Usually caused by obstructive atherosclerotic arterial disease
- Can also be caused by embolic disease, vasculitis, in situ thrombosis related to hypercoagulable states, thromboangitis obliterans, cystic adventitial disease, popliteal entrapment, or traums



The 1980's and Improvement in Revascularization

- Multi-center review of 2,829 patients
- All with limb-threatening ischemia
- Compared trends in treatment
- Primary amputation rate decreased 52% in 1974 to 11% in 1989
- Amputation rate now increasing due to increase in DM pts.



Factors that contribute to CLU

- Syndromes that are known to reduce blood flow to the microvascular bed
- Diabetes
- Severe low cardiac output states
- Vasospastic disease

- Conditions that accelerate or compound CLI
- Infection
- Skin breakdown
- Traumatic injury



Diagnostic Procedures

• Invasive

- Noninvasive
 - Doppler Wave forms
 - Pulse volume recordings



Natural History

 For most patients, atherosclerosis is diffuse and slowly progressive The nurse practitioner can select patients who are most likely to benefit from medical, radiologic, and surgical intervention



Medical Management

- Aspirin
- Antiplatelet therapies
- Risk factor modification
- Statins
- Beta-blockers
- ACE inhibitors

- Glucose control
- Smoking cessation
- Preventititve foot care
- Weight reduction
- Exericse program



Recommendations

- Supervised exercise training
- Program should be a minimum of 30 – 45 minutes at least 3 times per week for a minimum of 12 weeks
- Minimal data support the efficacy of the informal "go home and walk" advice
- Supervised hospital or clinic-based program which ensures that patients are receiving a standardized exercise stimulus in a safe environment is effective



Recommendations

- Statins: target LDL <70
- Efficacy of fibric acid derivatives in PAD not known
- BP control: <140/90 (nondiabetics); <130/80 (diabetics and CRF)
- B-blockers not contraindicated

- ACE inhibitors reasonable
- Control diabetes (Hgb A1-C <7%)
- Smoking cessation
- Homocysteine-lowering drugs (i.e. folic acid and and B12 efficacy not well established)



Surveillance and Secondary Prevention

- Annual physical
- Regular carotid duplex for everyone >60 years old with one or more risk factors for atherosclerosis
- Screening aortic ultrasound

- Ankle-Brachial Index
- EKG and Stress Echo are diagnostic not screening tools. May predict need for angiography or surgery



- UCLA program
- Focuses on employing secondary prevention measures while patients are in the hospital in order to improve clinical outcomes



- Aspirin 81-162 mg daily should be initiated if no contra-indications.
 Consider other antiplatelet therapies (i.e. Plavix or combination therapy)
- Statin therapy
 - For all diabetic patients regardless of their lipid profile
 - Everyone else if no contraindications

- Statin therapy
 - LDL <70mg/dL</p>
 - HDL>40 mg/dL
 - Triglycerides <150mg/d/



- Angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARB) should be started in absence of contraindication irrespective of the blood pressure or cardiac ejection fraction
- Beta blockade should be prescribed for all patients in the absence of contraindication



- Fish oil or omega-3 fatty acids should be commenced with dietary instruction for all patients
- Aerobic exercise programs that involve 30-60 minutes of moderately intense exercise at least 5 times a week should be prescribed



 Smoking cessation, including access to formal smoking cessation programs Before hospital discharge and at six week follow-up, cardiovascular lipid profile and liver enzymes should be checked and at routine intervals thereafter.



Recommendations

- Antiplatelet therapy
- Aspirin in daily doses (81 mg – 325 mg)
- Plavix 75 mg daily
- Warfarin



Warfarin (Coumadin)

- Keep INR 2.0 3.0 unless contraindicated
- Combination of oral anticoagulation plus aspirin may reduce risk of cardiovascular events but is associated with an approximately 2-fold increased risk of bleeding



Pharmacology Treatemnt for Claudication

- Pletal (cilostazol 100 mg po bid) in the absence of heart failure
- Trental (400 mg tid) improves walking distance



Other proposed medical therapies: not effective

- L-arginine
- Carnitine
- Ginkgo biloba
- Prostaglandins
- Vitamin E
- Chelation



Surgical Management

• Open bypass

- Endovascular
 - Angioplasty
 - Stent



Surgical Interventions

- Endarterectomy cleaning the plaque out
- Direct angioplasty adding a little extra piece to an artery
- Bypass rerouting around a blockage



Indications for Lower Extremity Amputations

- Complications of
 60 80%
 diabetes
- Nondiabetic infections with ischemia
- Ischemia without infection
- Chronic Osteomyelietis
- Trauma

• 15 - 25%

- 5-10%
- 3-5%
- 2-5%



Survival Rates After Amputations

- After 1 major lowerextremity amputation
 - 3 year survival rate is
 50%
 - 5 year survival rate is 40%

- Contralateral amputation
 - 42% of patients within 1
 to 3 years after first
 amputation
 - 56% of patients within 3
 to 5 years after first
 amputation



Indications for Lower Extremity Amputations

Complications of Diabetes	60 - 80%
Nondiabetic infections with ischemia	15 – 25%
Ischemia without infection	5 - 10%
Chronic osteomyelitis	3 – 5%
Trauma	2 – 5%
Miscellaneous	5 - 10%



ACC/AHA 2011 Practice Guidelines for the Management of Patients with Peripheral Arterial Disease (lower Extremity, Renal, Mesenteric, and Abdominal Aortic)

A Collaborative Report from the American Association for Vascular surgery/Society for Vascular surgery, society for Cardiovascular Angiography and Interventions, society for Vascular Medicine and biology, society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines

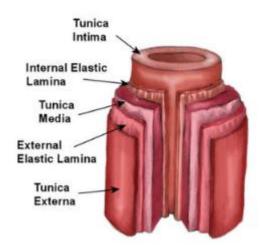


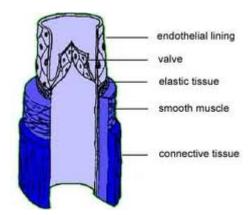


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 - Ulcer location, usually above ankle (medial malleolus)
 - Not painful
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- The treatment of chronic venous insufficiency (CVI and varicose veins has undergone a radical transformation in the last decade.
- The rapid adoption of endovenous ablation, utilizing heat generated by either a radiofrequency generator or a laser, has led to the movement away from operating room management to a treatment paradigm with office-based procedures.



Signs and Symptoms of Chronic Venous Insufficiency

- Cramping, heaviness, and "tiredness" of the limbs
- Leg swelling
- Large varicosities
- Hyperpigmentation
- Venous ulceration



Risk Factors for CVI

- Multiple pregnancies
- Family history
- Obesity
- Standing profession
- History of leg trauma
- Previous DVT
- Female estrogen can cause venous relaxation



Lipodermatosclerosis

- Progressive replacement of skin and subcutaneous tissue by fibrous tissue
- Inflammatory response to increased pressure in veins
- Acute phase
 - Skin painful, thickened, red, hot, tender
- Chronic phase
 - Skin thick, hard, tight
 - "inverted champagne bottle"
- Treatment: pulse dose steroid (50mg prednisone q 8h or Medrol dose pack x 2 with topical steroid

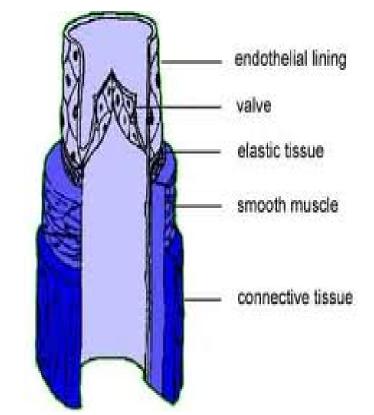


Venous Ulceration

- Skin dies faster tan can be replaced with fibrous tissue
- Tissue becomes necrotic
- Trauma leads to ulceration
 - Any minor injury can precipitate death of skin

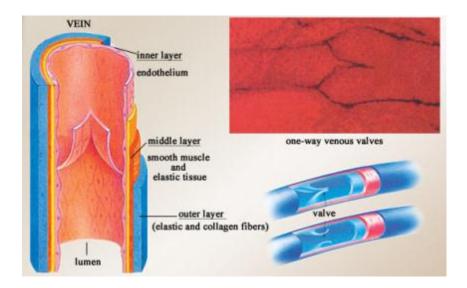


- Valves play the central role in most venous disorders
- Normal venous valves are bicuspid, opening so that blood flows toward the heart and closing so as to prevent reflux



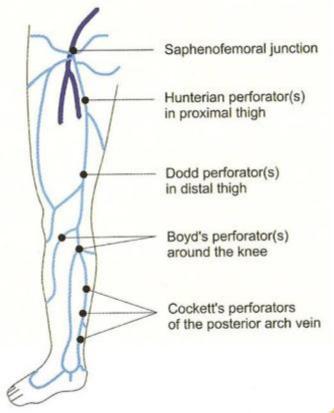


 The greatest number of valves are located in the lower leg





- Veins can be divided into a superficial and deep system
- The superficial vein most commonly diseased is the greater saphenous veins
- Deep veins are thinner and accompany arteries





Diagnosis of CVI



Vascular Physical Exam



History and Physical

- Symptoms progess as patients on their feet more during the day
- Subjective complaints: "heaviness"; "fatigue"
- Objective findings: swelling and engorged varicosities
- Pedal pulses must be checked and documented



Hemosiderin staining

- Occurs in long-standing CVI
- Typically in a distribution around the medial malleolus



Duplex ultrasound

Specify why duplex ordered

- Not a rule out DVT study
- Study should include examination of the greater and lesser saphenous veins
- Study should include presence of enlarged or refluxing perforator veins
- Note size and distance from the ankle

Reflux

- Reflux typically defined as reversal of flow for greater than 0.5 seconds
- Perform studies in standing position
- Note if reflux is spontaneous or elicited (squeezing or Valsalva)



Treatment of CVI

- Leg elevation
- Compression stockings
- Multi-layer compression therapy
- Manual lymphatic drainage
- Lymphedema pneumatic pumps
- Topical therapy



Surgical Options

- Saphenous vein stripping
- Sclerotherapy
- Skin grafting
- Deep vein reconstruction (Valvuloplasty & Axillary vein transfer)
- Open subfascial perforator ligation
- Stab phlebectomy



Treatment of branches and perforators



Diagnosis

- D-dimer blood test (98.4% sensitive)
- Duplex ultrasound (95% sensitive)
- Contrast venography is gold standard but potential risks of contrast must be weighed
 - Costly
 - Less readily available
 - Allergic reaction
 - Renal dysfunction



Venous thromboembolism

Deep Vein Thrombosis

- Vessel wall injury, stasis, hypercoagulability
- 50% will develop CVI
- Occurs in deep veins of the lower extremity
- Unilateral leg swelling, pain, pitting edema, blanching

Pulmonary embolism

 Vessel wall injury, stasis, hypercoagulability



Table 1. Wells Scoring System	To Predict DVT
Finding	Score
Active cancer	+1
Paresis, paralysis, or recent plaster cast immobilization of lower extremity	+1
Bedridden >3 days or major surgery within four weeks	+1
Localized tenderness over distribution of deep veins	+1
Entire leg swollen	+1
Calf swelling >3cm compared to contralateral side	+1
Pitting edema (greater in symptomatic leg)	+1
Collateral superficial veins (non varicose)	+1
Alternative diagnosis as likely or greater than DVT	-2



Treatment of DVT

- Goal: minimize risk of pulmonary embolism, prevent propagation of thrombus, and facilitate resolution of the clot itself to prevent longterm complication
- Anticoagulation: 5-7 days of unfractionated heparin
 - Usually IV
 - 5000 U as loading dose (or 80U/kg)
 - At least 30,000 U every
 24 hours
 - Goal: ptt between 50-80



Coumadin

Warfarin (Coumadin)

- Keep INR 2.0 3.0 unless contraindicated
- Combination of oral anticoagulation plus aspirin may reduce risk of cardiovascular events but is associated with an approximately 2-fold increased risk of bleeding





Absolute Contraindications of Oral Anticoagulants

- Known esophageal varices
- Significant thrombocytopenia (platelet count <50 x 10/L)
- Documented hypersentitivity
- Acute clinically significant bleed

- Decompensated liver disease or hypercoaguable state (INR>1.5)
- Pregnancy or within 48 hours post-partum
- Severe renal impairment



Relative Contraindications

- Previous history intracranial hemorrhage
- Recent major extracranial bleed within last 6 months where cause has not been identifed or treated
- Recent documented peptic ulcer within 3 months

- Recent history recurrent iatrogenic falls in patient with high bleeding risk
- Dementia or marked cognitive impairment with no reliable caregiver
- Chronic alcohol abuse

