Chronic Venous Insufficiency

VENOUS DISEASE:
UNDERSTANDING EDEMA, ITS CAUSES AND
WHAT WE CAN DO AND WHERE WE NEED TO BE CAUTIOUS

Marlene Reid, DPM FACFAS, FASPS, FACFAOM
Lower Extremity Venous Disease

- By age 50, nearly 40% of men and 20% of women have significant venous problems. Venous Disease Foundation (2012)

- 600,000 DVT/yr in US with 50% to PE. WebMD LLC (2013)

- Up to 1/3 of people with venous insufficiency cannot work outside of the home. USF “Understanding Compression” 2001

- 30 million Americans affected by varicose veins or CVI, only 1.9 million seek treatment annually. Gloviczki P, et al., JVS; May 2011

- “70% of us practice veins but only 20% of us had training.”
CVC Vascular Surgeon, Chicago 2012
Chronic Venous Disease

- **Etiology**
  - Venous Obstruction (DVT)
  - Valve Incompetence
  - Post Thrombotic Syndrome

- **Risk Factors**
  - Heredity and age
  - Prolonged Standing
  - Heavy Lifting
  - Multiple pregnancies
  - Ligamentous Laxity
  - Obesity
  - Diabetes??
  - PAD risk factors ??
  - Inc. in circulatory fluid volume
  - Dec. in skeletal muscular activity

- **Manifestations**
  - Recurrent Edema
  - Hyperpigmentation
  - Venous Stasis Dermatitis
  - Lipodermatosclerosis
  - Varicose Veins
  - Telangiectasias & reticular veins
  - Corona Phlebectatica
  - Venous Ulcerations

- **Presentations**
  - Superficial Venous Disease
  - Deep Venous Disease

- **Course**
  - Progressive - Stages: I, II, III
  - Preventable
  - Manageable
Stages of Venous Disease

I. Varicose veins
   I. Heavy Foot Syndrome

II. Ankle/leg edema
   II. Intermittent edema

III. Stasis dermatitis
     III. Persistent edema and skin changes

IV. Lipodermatosclerosis

V. Venous stasis ulcer
     IV. Ulceration

I. Edema and Corona Phlebectatica
   II. Dermatological changes

III. Ulceration
CEAP Classification of Venous Disease

- Clinical
  - C0: No visible evidence of venous disease
  - C1: Superficial spider veins (telangiectasias or reticular veins)
  - C2: Simple varicose veins only
  - C3: Ankle edema of venous origin
  - C4a: Skin pigmentation in the ankle area and/or dermatitis/eczema
  - C4b: Lipodermatosclerosis
  - C5: Healed venous ulcer
  - C6: Open (active) venous ulcer
  - S: Symptomatic
  - A: Asymptomatic

- Etiological
  - Ec: Congenital
  - Ep: Primary
  - Es: Secondary
  - En: No venous etiology

- Anatomical
  - As: Superficial veins
  - Ap: Perforating veins
  - Ad: Deep veins
  - An: No venous anatomy involved

- Pathophysiologic classification:
  - Pr: Reflux
  - Po: Obstruction
  - Pr, o: Reflux and obstruction
  - Pn: No venous pathology
Venous Disease in Podiatric Practices

Hyperpigmentation and Venous Stasis Dermatitis and Ulceration
Skin loses elasticity and becomes dry and shiny
Lipodermatosclerorsis (LDS)

- LDS literally means scarring of the skin and fat
- Chronic panniculitis with lipomembranous changes
- Found with long standing venous insufficiency
- Skin becomes brown, smooth, indurated and painful
- Proximal to the ankle, usually medial
- Tapering proximal to the ankles “inverted Champagne bottle”
- Skin is permanently and irreversibly damaged
Commonly Seen Abnormal Veins
Caused by Superficial Vein Reflux

- **Telangiectasia (spider veins)**
  - Confluence of red dilated venules
  - Intradermal <1 mm in caliber
  - 15% of men and 25% of women
  - Family hx, sun exposure, pregnancy
  - Can be caused by Reticular Veins

- **Reticular Veins (blue veins)**
  - Also called intradermal varices.
  - Dilated bluish subdermal vein
  - 1 mm to less than 3 mm in diameter
  - Usually Tortuous but nonpalpable
  - Called “Feeder Veins” if associated with Telangiectasia veins
Corona Phlebectatica (CP)

- CP is classically described as the presence of abnormally visible cutaneous blood vessels at the ankle with four components: "venous cups," blue and red telangiectases, and capillary "stasis spots."
- Fine visible vessels too numerous to delineate
- Dilation and elongation of venules in the dermis of the ankle and foot
Varicose Veins

- Subcutaneous Dilated Veins > 3mm in diameter
  - Twisted, elongated and palpable – est. 29 million suffer
  - Irreversible damaged walls and valves between deep/superficial
  - Also known as a varix, varices and varicosities – palpable
  - Ant/Pos/Med/Lat thigh and legs and med/Lat ankles as well as female genitalia
  - Blood pools in stagnant segments and becomes inflamed causing chronic phlebitis, aching, pain, itching, burning and cramping
Venous Ulcerations

- VLUs affect 1% of the adult population and may be primary or secondary Johnson and Rodgers, Podiatry Management August 2011
- Primary VLU are due to valvular defects and hypertension in the superficial venous system
Risk Associated with Venous Ulcers

• Underlying Disease Process

Wound Care and Compression alone often fail to prevent recurrence without treating underlying reflux and venous hypertension Barwell, et al., Lancet 2004; 363:1854-1859.

• Recognition of etiology

The plaintiff maintained that the defendants were negligent in treating the lesions as venous stasis ulcers, failing to note or record evidence of chronic venous insufficiency, failing to perform proper venous tests, failing to diagnose and treat arterial insufficiency, and failing to timely perform vascular surgery.
Venous Anatomy of the Lower Extremity

• Subcutaneous superficial system not paired with arteries

• Deep system within the muscular fasciae paired with corresponding arteries

• Connecting or Perforating veins

• Dysfunction, mainly of the valves, may occur in each system and in combination
Anatomy of a Vein

- Intima > valves
- Media
  - Smooth muscle
  - Collagen
- Adventitia

- Larger superficial veins – thicker media more muscular
- Smaller superficial veins – thinner media, less strength
- Deep veins – more collagen, more strength and sheath, function with the muscular calf pump
Superficial Venous System

Small (Lesser) Saphenous Vein (SSV)

- Courses lateral to posterior
- Posterior to lateral malleolus
- Superficial to the deep fascia
- Midline to upper calf
- Between two heads of the gastrocnemius
- Usually joins the Popliteal
- 1/3 time feeds into GSV via communicating veins
Superficial Venous System

Great Saphenous Vein (GSV)

- Anterior to medial malleolus
- Crosses posteriorly to lie medial
- Anteromedially AK
- Superficial to the deep fascia
- Passes thru foramen ovale
- Feeds common femoral vein at saphenofemoral junction

Common Femoral Vein
Saphenofemoral junction

- Other veins that feed into it around the junction:
  - the superficial inferior epigastric
  - the superficial external pudendal
  - the superficial circumflex iliac

- Above the knee
  - Anterior and Posterior Circumflex

- Below the knee
  - Anterior tributary
  - Posterior Arch Vein
Superficial Venous Supply of the Foot

- Dorsal Venous Arch
  - Dorsal Digital veins
  - Intercapitular (plantar)
    - Via perforators
  - Common digital Veins form Dorsal Venous Arch
  - Medial Marginal to GSV
  - Lateral Marginal to SSV
  - Perforating Veins to GSV

- Dorsal Superficial Venous Structures –
  - Distally, more organized than plantar
Perforator Veins

• Direct from superficial to deep veins
• Bicuspid valves and unidirectional
• High variability with some consistent groupings
  • Mid thigh - Hunterian
  • Distal thigh - Dodd
  • Knee - Boyd
  • Ankle and distal medial calf - Cockett
  • Foot - multiple
Perforating Veins of the Foot

- Medial more developed than Lateral
- Perforator extending across the 1st IM space extends from the superficial venous arch dorsally to the (deep) pedal vein (dorsalis pedis vein) which leads to the anterior tibial vein
Deep Venous System

- Eventual return to the Right Atrium
- Valvular
- Intramuscular

- External Iliac Vein
- Common Femoral Vein
  - Deep Femoral Vein
    - Short course with deep muscle tributaries
  - Femoral Vein
    - Courses through the adductor canal
  - Popliteal Vein

- Peroneal Vein
  - soleal and gastrocnemius intramuscular venous plexi join mid calf from sinusoid venous network

- Anterior Tibial
- Posterior Tibial
Deep Venous Supply of the Foot

• Deep Plantar Venous Arch
  • “Plantar Venous Pump”
  • Plantar digital veins ??
  • Send intercapitular veins to superficial dorsal venous arch
  • Metatarsal plantar veins
  • Deep plantar venous arch
  • Medial/Lateral plantar veins
  • Posterior Tibial Vein

• Dorsal Deep Veins
  • less prominent than Plantar
  • Dorsal digital veins are superficial
  • Deep veins begin at digital clefts
  • Pedal Vein (Dorsalis Pedis Vein)
    • Along with the 1st IM space perforator of the superficial venous arch
  • Anterior Tibial Vein
Venous Return

- **Primary** – Left Ventricular cardiac activity
  Normal inflow to the lower extremity veins is purely via arterial inflow

- **Secondary**
  - Valves - preventing reverse flow
  - Venous Tone
  - Skeletal Muscle Venous Pump (musculovenous pump)
    “peripheral heart”
  - Respiratory Pressure – negative intrathoracic pressure
**Venous Pump**

- Cyclic rhythm of contraction and relaxation during stance
- Deep veins are refilled from superficial veins during the relaxation phase
- Contraction forces proximal valve to open
- Works to decrease distal capillary pressure and preserving the integrity of the microcirculation
Venous Valves

- Valves of the Leg – approximate distribution

Deep Veins: More distally
- Popliteal vein 2 - 4
- Anterior tibial veins 9 -11
- Posterior tibial veins 9 -19
- Peroneal veins 7

Valves present at junctions
- GSV/Femoral
- SSV/Popliteal

Superficial Veins have fewer:
- GSV and SSV 7-9
- Valves near tributary terminals
- Flow is slower due to lack of muscular pump

- Valves in the LE outnumber the UE due to gravity

- Major Perforating veins have 1-3
- Absent in smaller perforating veins
- Perforators in the foot are absent of valves or valves lead to superficial veins
Valvular Incompetence

Venous Insufficiency – Increased SVP

- Primary Cause of Chronic Venous Insufficiency (CVI)
- Venous Reflux Disease results
- Venous Outflow Obstruction
- Venous insufficiency also tends to be progressive
- Superficial venous reflux can cause spider veins, varicose veins, and lead to edema, hyperpigmentation, and venous ulcers
- Perforator reflux can cause edema hyperpigmentation and ulcers
- Most patients with venous dysfunction have incompetent valves in both superficial and perforator veins
- Symptoms: pain, swelling, leg heaviness and fatigue
- Many cases can be treated surgically
- Reflux extends to superficial vein trunk and increased superficial venous pressure (SVP) leading to varicosities and ulcers
# Medically Significant Reflux

<table>
<thead>
<tr>
<th>Age</th>
<th>Backward Flow %</th>
<th>Medical Problem %</th>
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<tbody>
<tr>
<td>10</td>
<td>3</td>
<td>0</td>
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<tr>
<td>20</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>65+</td>
<td>75</td>
<td>30</td>
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Chronic Venous Insufficiency
Deep Veins

- Deep Vein Incompetence usually the result of a DVT
- Deep venous insufficiency may result from increased pressure from superficial insufficiency
- Post phlebitic syndrome may develop
  - pain, edema, pruritis, hypohydrosis, eczema and eventually ulceration
- Venous Claudication may occur
  - deep incompetency and increased venous pressure during muscle contraction
- DVT recurrence rate is about 30% within 8yrs of patients who receive only 3-6 months of initial anticoagulation
- Deep venous disease is treatable but not curable
- Venous thromboembolism (VTE) includes DVT and PE
DVT Risk Factors

DVT recurrence rate is about 30% within 8yrs of patients who receive only 3-6 months of initial anticoagulation

Post knee arthroscopy, 2006 Virginia. 8 days p/o, DVT of the gastrocnemius veins of the left lower extremity. He prescribed Lovenox and Coumadin. Patient remained on anticoagulation therapy for three months with no complications.

In 2008 the same surgeon performed an arthroscopy with an anterior cruciate ligament reconstruction on the decedent’s same knee. He was not placed on anticoagulation prophylaxis following the surgery. Six days post-op he acutely developed difficulty breathing with choking, chest pain and syncope and later died.
DVT

- Risk Factors
- Inheriting a blood-clotting disorder...
- Prolonged bed rest, such as during a long hospital stay, or paralysis...
- Injury or surgery...
- Pregnancy...
- Birth control pills (oral contraceptives) or hormone replacement therapy...
- Being overweight or obese...
- Smoking...
- Cancer
DVT Risk Factors

- Paralysis
- Paresthesia or recent cast immobilization of lower extremities
- Major surgery requiring regional or general anesthetic in the past 12 weeks or was recently bedridden for greater than 3 days
- Recent long-distance travel
- Localized pain along distribution of deep venous system
- Swelling of entire leg and/or calf greater than 3 centimeters
- Pitting edema
- Collateral superficial veins
- Previously documented DVT or PE
- Active cancer
Thromboprophylaxis

- Provide prophylaxis or not??
- Risk Assessment vs. Bleeding
- Non-medical option:
  - In a study of the efficacy of intermittent pneumatic compression devices in multiple postoperative patient groups versus no use of prophylaxis, Urbankova et al reported that the incidence of DVT was reduced by 60%. However, the use of mechanical means of prophylaxis alone is not effective in moderate or high-risk cases.
- Medical options:
  - A large study performed in Europe, the Pulmonary Embolism Prevention (PEP) study, found that the overall DVT rate was decreased 30% with low-dose aspirin compared with placebo, and the overall pulmonary thrombosis rate was decreased by 40%.
  - Xarelto 10mg QD starting 6-10 hrs s/p Sx x ?? Days – x 12 days for knee
Chronic Venous Insufficiency
Other Etiology and Pathogenesis

- Pathogenesis
  - Defects in venous wall strength
  - Valvular incompetence
  - Venous hypertension
  - Cellular problems
- 70% hereditary females > males – hormonal relaxation of venous walls
- Inflammatory changes in the veins
- Inflammation causes ischemic and trophic injury to skin
Symptoms of CVI

- Pain, throbbing or cramping
- Burning or itching, especially around a vein
- Heaviness, aching or fatigue especially late in the day or after periods of dependency
- Night Cramps
- Restless Leg Syndrome
- Same symptoms found with pes valgus or PTTD
- DDx - can be found with periods of sitting or NWB
- Eczema and hair loss to lower legs

Course
  - Progressive
  - Preventable
  - Manageable
Superficial Venous Disease

- Defect in superficial valves
- Phlebitis, spider veins
- Symptoms of superficial venous reflux:
  - pain, swelling, leg heaviness and fatigue
- Varicose Veins
  - 60% of women
Varicose Veins

• Primary – SVD
  • Degeneration of venous wall
  • Starts distally
  • Inflammation
  • Venous hypertension
  • Collagen breakdown
  • Intima remains intact
  • Very common
    • 20-40% adult population
    • Progresses slowly

• Venous Stasis Syndrome
  Caused by Primary or secondary
  Post Phlebitic Syndrome only
  one cause

• Secondary:
  • Deep Reflux
  • Post Thrombolytic Syndrome
    (Post Phlebitic Syndrome)
  • Clot causes inflammation
  • Obstruction > collaterals
  • Damage to the intima
  • Much less common
    • 20-50% of DVT > VV
    • Most common with Iliofemoral clot
    • Onset often 6 months post DVT
    • Progress quicker – severe within 5 years
Capillaries: Fluid exchange within tissue
Between the intra- and extravascular space

- Starling principle of microvascular fluid exchange
  - Arterial End: hydrostatic > osmotic
  - Venous End: osmotic > hydrostatic

- Exchange takes place at the capillary bed
- Exchange takes place across the capillary wall
- Capillaries are normally impermeable to plasma proteins
- Capillaries are freely permeable to water and low molecular-weight solutes
- Normally, a relatively stable interstitial fluid volume (IFV) is maintained
- 10% fluid is left in the tissue and picked up by the lymph system
Hydrostatic Pressure

- Dependent on the gravitational forces in a standing position as well as the distance of the vein from the heart
- Superficial venous pressure (SVP) at the ankle is normally:
  - 12mmHg when supine
  - 104mmHg when in stance
- During gait, ambulatory venous pressure (AVP) is:
  - 30mmHg
- With venous disease, pressure backs up and increases the venous capillary pressure (VCP)
- Increases in VCP causes increased transcapillary filtration which results in edema
- Venous reflux increases SVP and VCP increases and pressure in skin
Edema

- Clinical state characterized by an accumulation of fluid in the interstitial or intracellular space

- Edema is the result of excess fluid in the interstitial space and occurs when there is a breakdown in this pressure gradient
  - either high rate of transcapillary filtration into the tissue
  - low lymphatic drainage rate
Etiology by Transcapillary Pressure Gradient

- **Increase in Capillary Pressure**
  - CHF
  - Venous Congestion
  - Increase in blood volume
- **Decrease in Blood Osmotic Pressure**
  - Hypoalbuminemia
  - Thermal injury
- **Increase in Tissue Osmotic Pressure**
  - Retaining waste and metabolites
  - Thermal injury
- **Increase in Capillary Permeability**
  - Inflammation - gaps in the endothelium allows leakage of proteins
Edema by Classification

- **Acute Edema**
  - DVT
  - Trauma
  - Cellulitis
  - Septic Arthritis
  - Allergic Reaction

- **Chronic Edema**
  - **Regional**
    - Varicose Veins
    - Obstruction of venous return
    - Lymphedema
      - usually involves the foot – venous edema does not
  - **Systemic**
    - CHF
    - Nephrotic Syndrome
    - Myxedema
Edema by Classification

- **Pitting**
  - Bilateral
    - Hypoalbuminemia
    - CVI
    - Venous Hypertension
    - CHF, kidney disease
  - Unilateral
    - CVI
    - DVT
    - Compartment syndrome
    - Cellulitis
    - Baker’s Cyst, tumors, trauma

- **Non-Pitting**
  - Lymphedema
    - Usually Unilateral
    - May pit early before fibrosis
  - Myxedema
    - Non-fluid
    - mucopolysaccharides in dermis
Medications that Commonly Cause Edema

Meds diabetics may be on
• Pioglitazone (Actos) and Rosiglitazone (Avandia)
• Gabapentin (Neurontin)
• Pregabalin (Lyrica)
• Amlodipine (Norvasc)
• Prednisone
• NSAID’s (Ibuprofen, Naproxen, Celecoxib)
• MAO inhibitors
• Anti-psychotics

Anti-hypertensives:
• Hydralazine
• Diazoxide (Hyperstat)
• Minoxidil
• Methyldopa
• Calcium Channel Blockers (e.g. Amlodipine)
• Beta Blockers
• Clonidine (Catapres)
• Acyclovir (Zovirax)
• Pramipexole (Mirapex)
• Oral contraceptives
Pitting Edema

- **1+**: 2mm depression-barely detectable
  Immediate rebound

- **2+**: 4mm deep pit
  A few seconds to rebound

- **3+**: 6mm deep pit
  10-12 seconds to rebound

- **4+**: 8mm: very deep pit
  >20 seconds to rebound

- **1+**: Barely detectable impression

- **2+**: Slight indentation
  15 seconds to rebound

- **3+**: Deeper indentation
  30 seconds to rebound

- **4+**: >30 seconds to rebound


Post Op Edema

Pre Op Considerations??
Other Venous Conditions

Lipedema  Lymphedema

[Images of affected legs]
Other Venous Conditions

- **May Thurner Disease**
  - “Iliac Compression Syndrome”
  - 30% population have anatomical variant that causes compression/obstruction on of the left common iliac vein via right common iliac artery
  - Entire left leg is edematous
    - can be seen in the right with different crossover location
  - Collateralization causes varicose veins

- **Pelvic Congestion Syndrome**
  - “Pelvic Varicosities Pain Syndrome”
  - Chronic pelvic pain
  - 10% of the general female pop
  - Pelvic varicosities
  - Varicose veins of the perineum, buttocks, or lower extremities
Diagnosing CVI

- Duplex Ultrasound
  - Provides visual image of vein and valve damage and possible venous obstruction
  - Measures speed and blood flow
- Photoplethysmography (PPG)
  - Multimethod venous refill tests
  - Venous outflow testing
  - Measures time and records wavelengths for refill and outflow
- Venogram

“Phlebology”
Field of Medicine that treats venous disease
Treatment Options for CVI

- Chemical sclerotherapy
- Vein stripping
- Bypass surgery
- Valve repair
- Angioplasty with or without stenting of a vein
- Minimally invasive endovenous ablation procedures such as Laser and Radio Frequency Ablation.
Podiatric Treatment of Superficial Veins
Treating CVI and Edema with Compression

- Bandaging
  - Unna’s Boots
  - Casts
  - Ace wraps
- Intermittent Pumps
  - Refractory Edema or Venous Dysfunction
  - Used in conjunction with stockings
  - Does not involve muscle pump
  - Adjustable pressure
- Compression Stockings
  - Ready to Wear or Custom
  - Must remove (>20mmHg) at night!
Why Compression is Important

- Helps prevent VTE – Venothromboembolism: DVT/PE
- Helps alleviate edema and discomfort
- Reduction of superficial venous pressure
- Decreases the progression of both venous and lymphatic disease
- Decreases the progression of post thrombosis syndrome and other venous dermatitis
- Helps soften woody edema
- Helps prevent skin breakdown
- Helps prevent and heal venous ulcers
What does Compression Actually Do??

- **Therapeutic Effect:**
  - Decreases pathologic venous capacity
  - Increases (or supports) the function of the insufficient valves
  - Increases fibrinolytic activity of the blood
  - Increases reabsorption of fluid into the capillaries

- Decreases Retrograde Flow
- Increases Venous Return
- Counters the affects of Ambulatory Venous Hypertension
The Accepted Mechanism of Action of Gradient Compression

- Reduces Edema
  - Allows the dermis to engage the superficial capillary network to improve function of O2 and waste exchange

- Capillary Action: Improves absorption and decreases leakage
  - Increases interstitial pressure
    - Decreased capillary leakage of fluids and solutes
    - Increase absorption of interstitial fluids – lymph system as well
    - Improves local capillary clearance

- Reduces the Diameter of Superficial Veins
  - Reduces the capacitance of these veins
  - Reduces reflux and venous pooling
  - Increase proximal venous flow
Compression of Superficial Veins

- Acts as a “restraint” as superficial veins do not have the muscular and fascial support system of deep veins
- External compression can compensate for both superficial and perforator valvular disease
- Return distended, over dilated veins to a more normal size
- Fabric is an external replacement for lost elasticity and resistance of skin
Indications for Compression Therapy

- Edema
- Chronic Venous Insufficiency
- DVT/PE Prevention
- Prevention of ulcer recurrence
- Management of ulcers
  - Requires 35mmHg
- Varicose Veins
- Pregnancy
- Post sclerotic therapy
- Superficial thrombophlebitis
- Tired, aching legs
- Hypertrophic Scar and dermatitis
- Lymph Edema
Characteristics of Compression Therapy

- **Resistance**
  - Ability to withstand stretch
  - High Resistance has Low elasticity and no applied compression (active force)
  - Unna Boots/casts – in-elastic

- **Elasticity**
  - Allows stretch by yielding to passive stretch
  - More elastic, less resistant and easier to apply garment
  - Short stretch – 40-90%
    (textile elastic)
    Constant tension - high resistance
  - High stretch – 120-200%
    Constant tension - less resistance
Types of Compression Devices

- Bandages
  - Reduce active edema
  - Require skilled clinician
  - Maintain conical shape for natural gradient/uniform compression
    - Pad dorsal ankle and posterior to LM.MM!
  - The more in-elastic, the less tension need to apply
    - Elastoplast
    - Casting
  - Short term (Loss of compression)
  - Loss of ADL

- Compression Garments
  - Long term management
  - Compression held 4-6 mos.
  - Consistent gradience
  - Works with the calf muscle
  - Removable
  - DJD, PAD
  - Neuropathy may dictate less compression
  - Over compress may cause muscle atrophy/compartment syndrome
  - Manual Strength may limit ability - lower compression may provide better compliance
Choosing the Right Compression Garment

- Ankle Anatomy and Sizing
- Measure ankle & calf
- Range of mmHg is given as a range of pressure at ankle based on ankle size
- Abnormal anatomy may dictate custom for greater precision

- Choosing Level of Compression mmHg
- 10-15 - fatigue, aches
- 15-20 - pregnancy, post surgical, travel, mild varicosities, moderate edema, DVT prophalactis
- 20-30 - moderate to severe varicosities, edema with pregnancy DVT, ulcer prevention, CVI, thrombophlebitis
- 30-40 – Lymph edema, severe CVI symptoms, ulcers
Risk of Compression Therapy

- Peripheral Arterial Disease
- Proper use of stockings
- Peripheral Neuropathy
  - Dermatitis
  - Infection
  - Cellulitis
- Ulcers
- CHF
In Summary....

Lower Extremity Venous Disease affects our patients and needs as much attention as peripheral arterial disease for prevention of progression.

Conservative treatment for edema should include elevation, increasing the muscle pump with activity and supportive measures to reduce the subjective and objective symptoms of superficial and deep venous disease.

Thank You