Chronic Venous Insufficiency

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Chronic Venous Insufficiency (CVI)

Definition

Prevalence

Anatomy/Physiology

Risk Factors

Signs and Symptoms

Classification and Staging

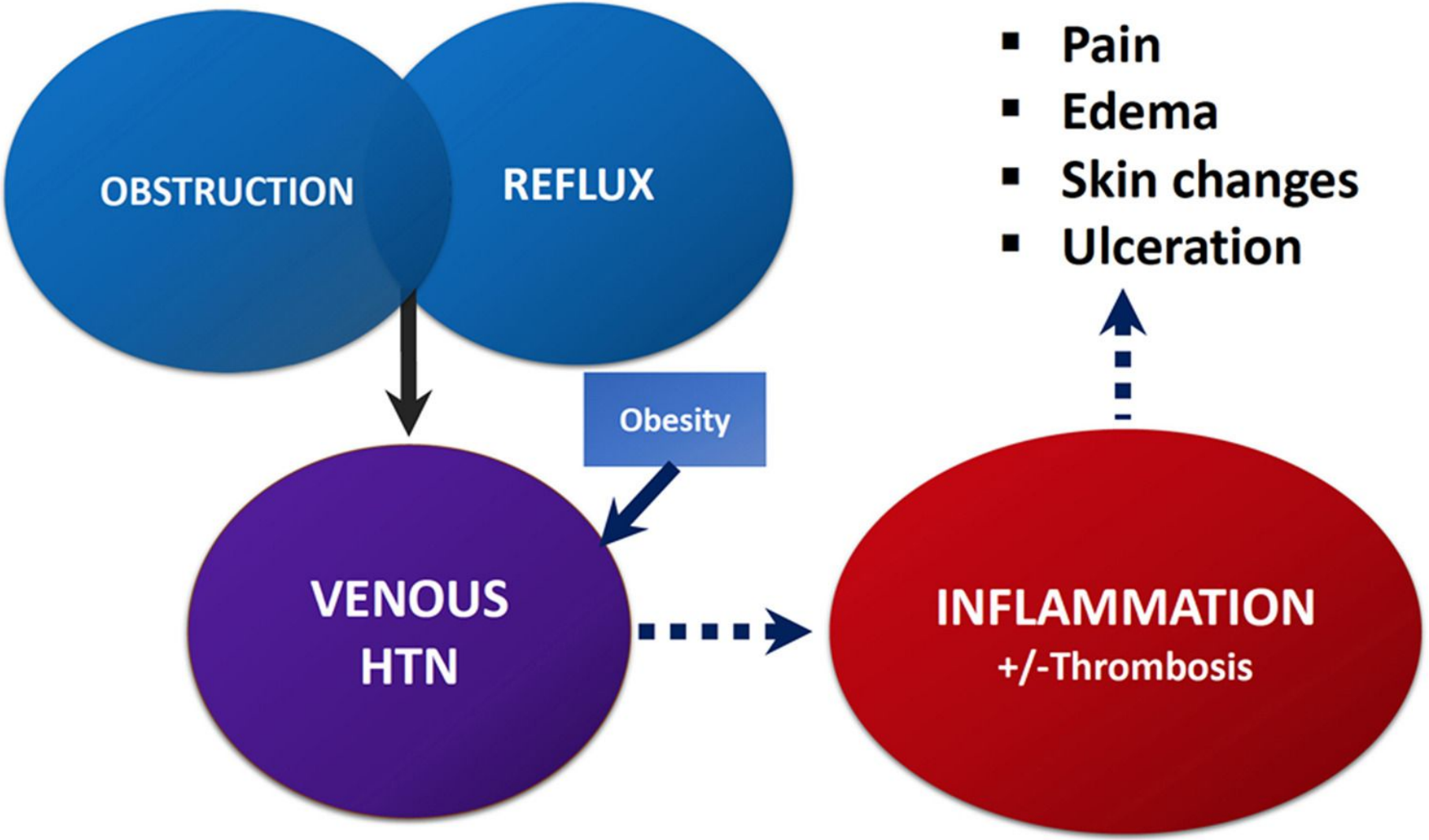
Diagnosis

Treatment

CVI Definition

Chronic Venous Insufficiency is a multi-factorial disorder whereas anatomic Superficial Venous Reflux, or Deep Venous Reflux, or Deep Venous Obstruction, alone or in combination, along with risk factors such as obesity or age, lead to Venous Hypertension in the lower extremities.

Venous Hypertension then leads to→Chronic Inflammation of Veins→
Pain/Edema/Color Changes/Skin changes/Ulceration


OBSTRUCTION
REFLUX
Obesity
VENOUS HTN
INFLAMMATION
+/-Thrombosis
Pain
Edema
Skin changes
Ulceration

CVI: Prevalence

In the United States more than 25 million adults have CVI.

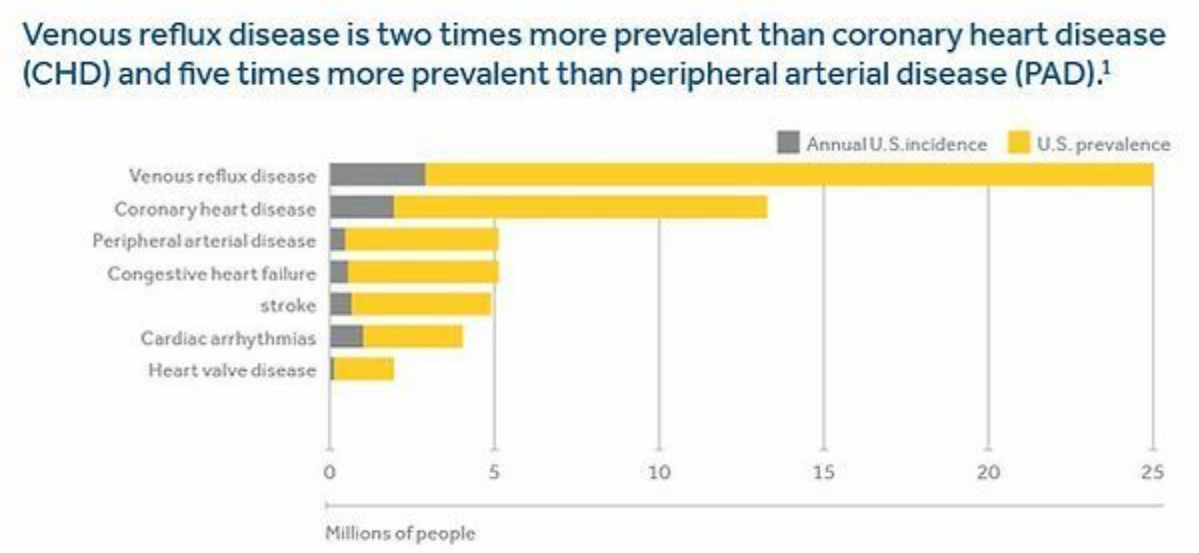
CVI is more prevalent than Peripheral Arterial Disease or Coronary Artery Disease.

The prevalence of varicose veins is as high as 57% in men and 73% in women. The prevalence of venous ulcers is 2% of the population.

Venous ulcers are the most common etiology of lower extremity ulcers in general.

Venous insufficiency direct costs are $1-2 billion annually with 2 million work days lost a year.

CVI: Prevalence



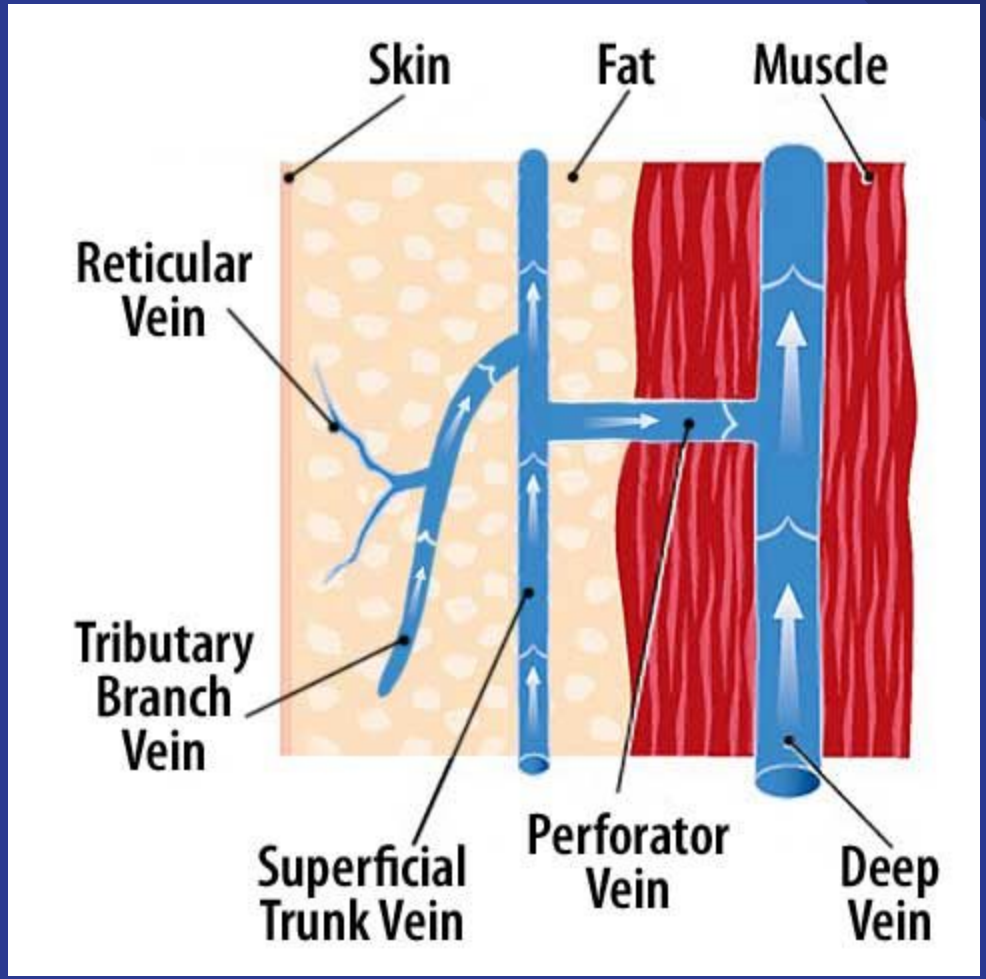
CVI: Anatomy

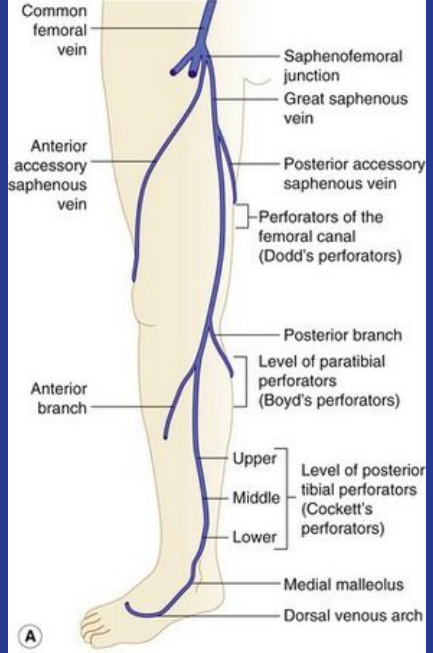
Anatomy:

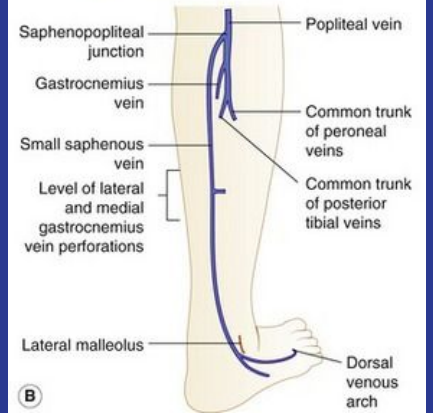
The veins of the lower extremities are divided into Superficial, Deep and Perforating Veins.

These veins all collaborate together to draw the total venous volume into the iliac veins in the pelvis and onward to the Inferior Vena Cava (IVC).

The superficial veins course above the fascia between the dermis and deep fascia, draining cutaneous microcirculation, while the deep veins are located beneath the muscle fascia. Perforator veins connect the superficial and deep veins by penetrating through the muscular fascia.


Skin
Fat
Muscle
Reticular Vein
Tributary Branch Vein
Superficial Trunk Vein
Perforator Vein
Deep Vein





CVI:Anatomy

Perforators are aligned on the medial aspect of the leg

Historical names are numerous for the various thigh and calf perforators

The Greater Saphenous Veins (GSV) have anterior and posterior accessory veins

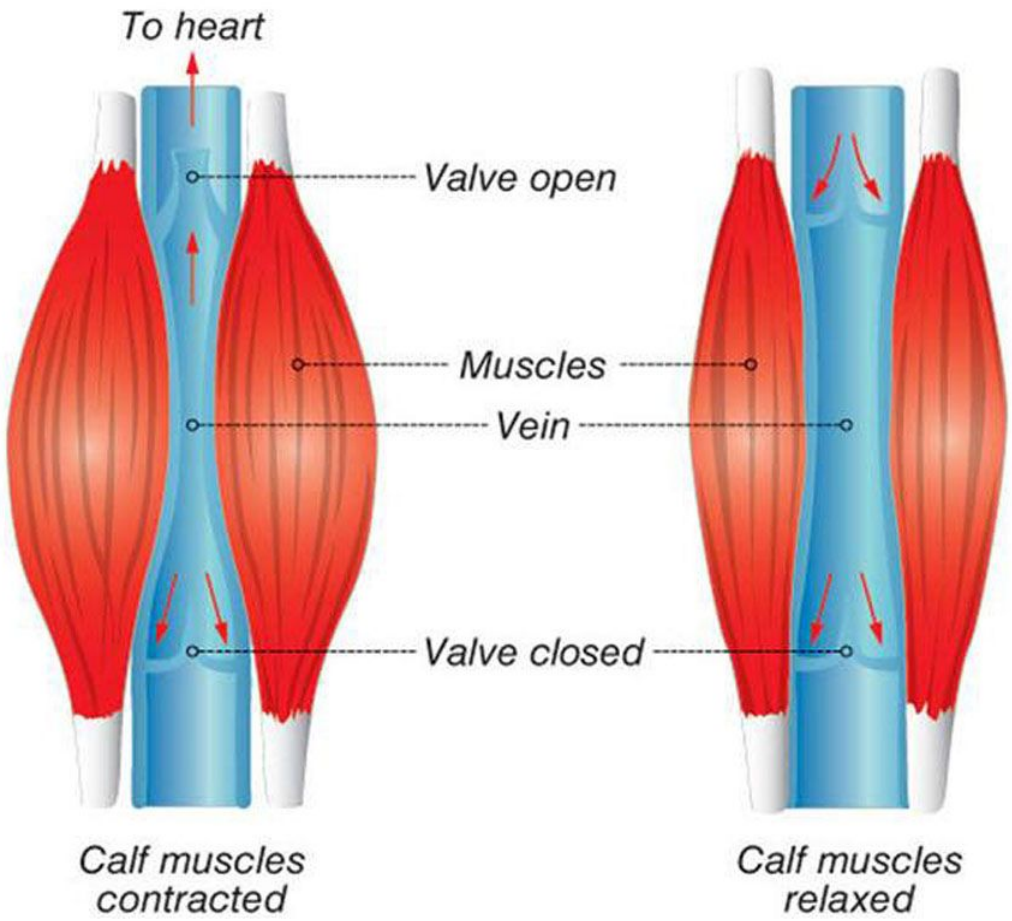
A vein that connects the GSV with the Small Saphenous Vein in the posterior thigh is called the Vein of Giacomini.

CVI: Physiology

Blood flows from the superficial veins to the perforator veins into the deep venous system. The movement of blood against gravity while in the upright position requires the assistance of one-way valves and muscle contraction to pump and return blood to the heart.

The one-way bicuspid valves located throughout the superficial, deep and perforator veins ensure that blood flows in one direction towards the heart while limiting backflow and pooling of blood in the lower extremities.

Together with the valve system, contraction of lower extremity muscles, such as the gastrocnemius, helps propel the blood upwards. Upon muscle contraction, the external venous wall is squeezed inwards forcing blood anterograde. Upon muscle relaxation, the vessel opens and physiologic reflux of blood occurs, closing the sinuses of the bicuspid valve.



Calf muscle contraction is the “pump” or the second heart in the legs to propel the venous return to the heart forward and upward.

This process does not work UNLESS the venous valves are competent. If INCOMPETENT, the blood returns via gravity to the legs.

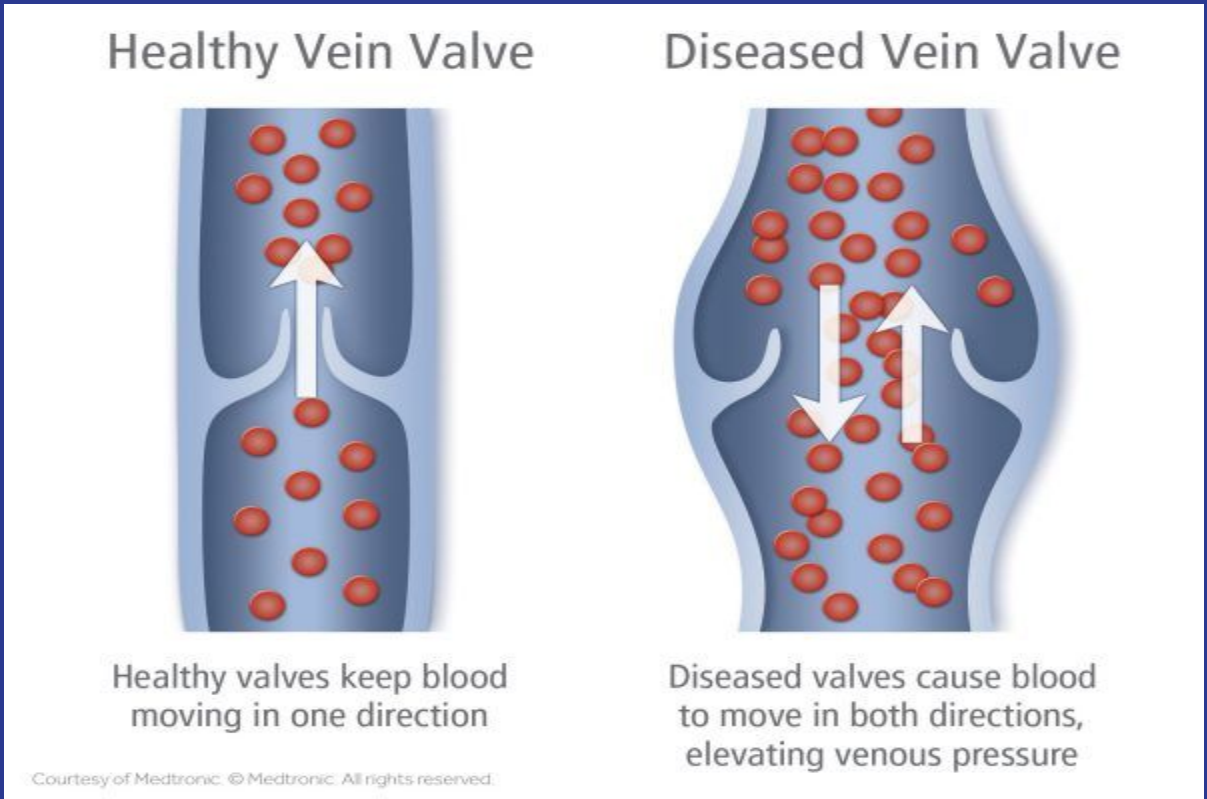
CVI:Pathophysiology

Primary vs Secondary CVI.

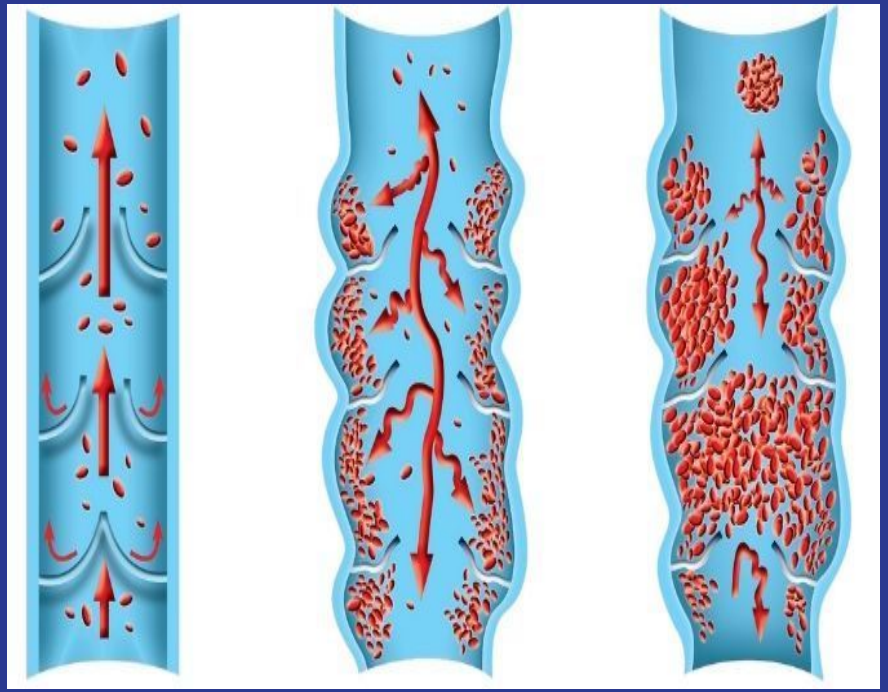
Primary CVI or idiopathic CVI corresponds to non-thrombotic causes, with structural and functional problems in the vein wall or bicuspid valves. Mostly superficial reflux.

Secondary CVI is a result of obstruction or post-thrombosis from a prior deep vein thrombosis (DVT) or from iliocaval compression from physiologic compression or thrombosis, or arteriovenous fistulas increasing venous pressures, venous malformations, and congenital deep vein agenesis or hypoplasia. Mostly deep reflux.

CVI: Valvular Reflux



CVI: Valvular Dysfunction

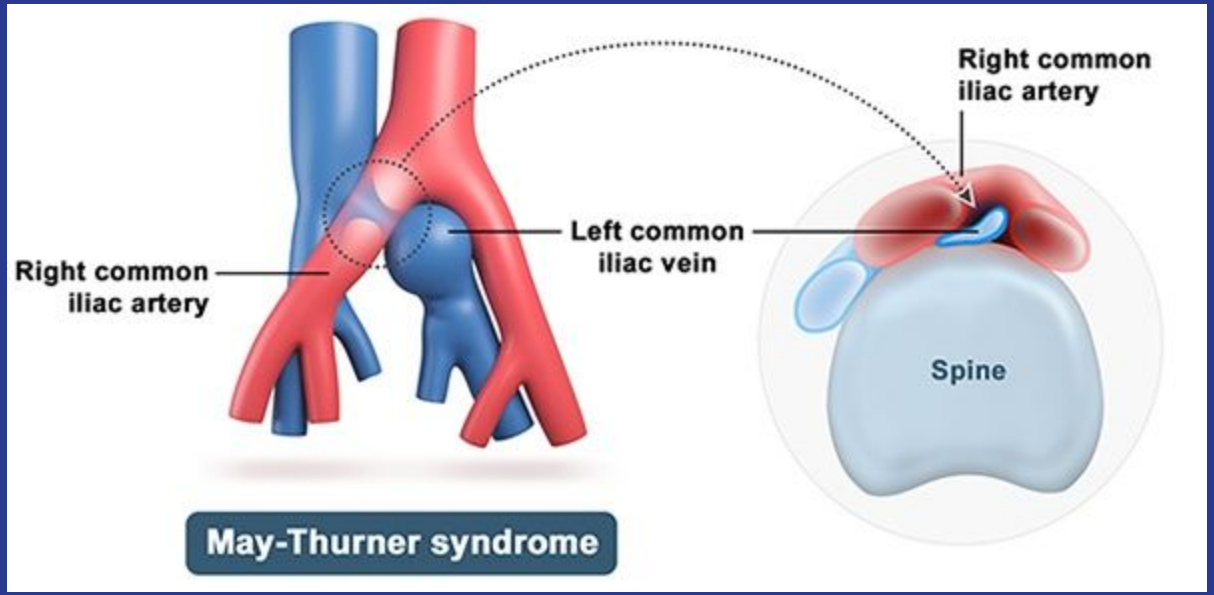


Left side is normal flow.

Right side is during an acute DVT.

Middle picture shows how in chronic DVT changes, the old clot hugs the vessel wall and the leaflets and causes valvular dysfunction.

CVI: External Compression



CVI: Risk Factors

Modifiable factors	Non-modifiable factors
Increased BMI **Smoking** **Sedentary life**	Advanced age Family history of venous disease Ligament laxity Trauma in lower limb Previous venous thrombosis Pregnancy Female gender

CVI:Signs and Symptoms

Symptoms

- None
- Aching
- Heaviness
- Throbbing
- Burning
- Tightness
- Cramps
- Itching
- Swelling
- Tiredness

Signs

Vein

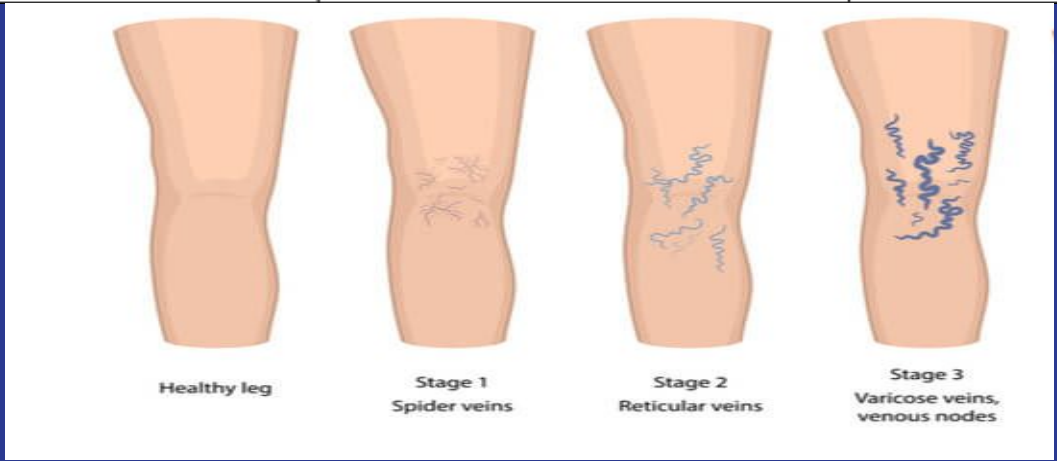
- Venular flares
- Varicosities
- Phlebitis
- Thrombosis
- Haemorrhage

Skin

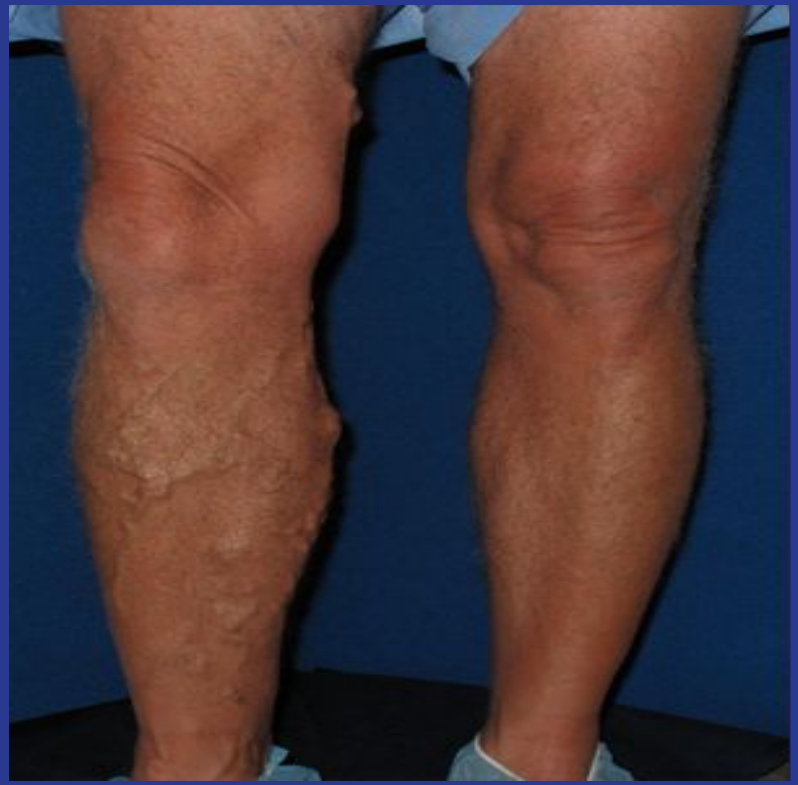
- Dermatitis
- Pigmentation
- Oedema
- Lipodermatosclerosis
- Cellulitis
- Poor wound healing
- Atrophie blanche (pre-ulcer)
- Scarring
- Ulceration
- Malignant transformation (e.g. squamous cell carcinoma)

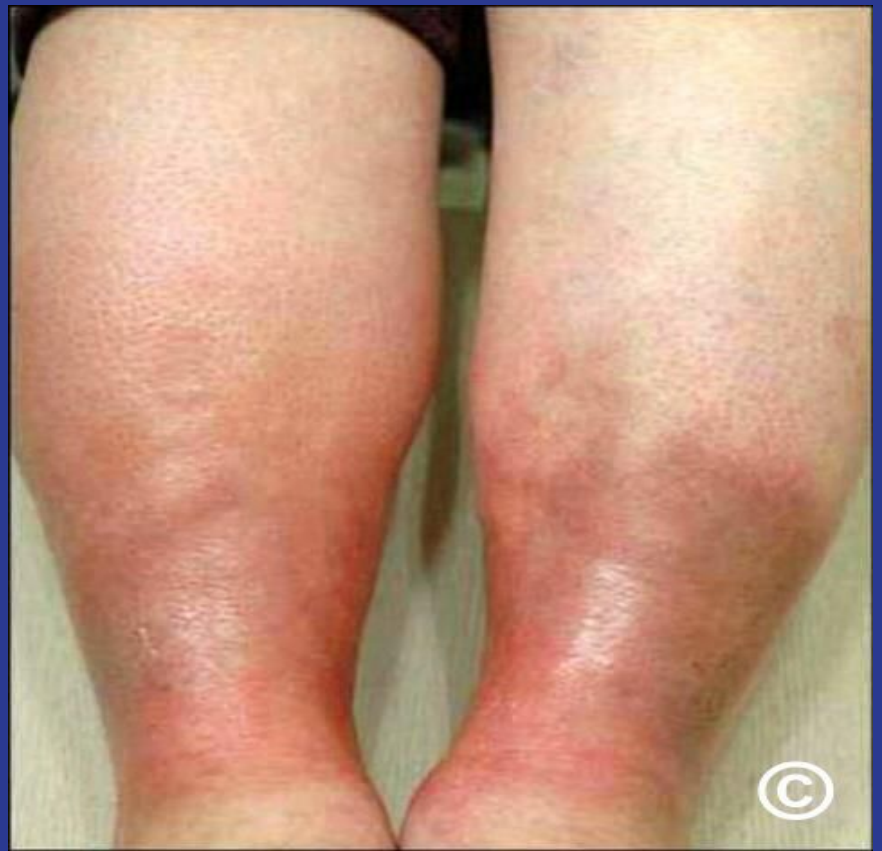
CVI: Signs and Symptoms

Spider veins	Reticular veins	Varicose veins
- measuring about 1-1,5mm; - have a pink, red or purple color; - sometimes accompanied by pain and discomfort in the affected area.	- size about 2mm in diameter; - color varies from green-blue to purple; - often causes burning and itching.	- larger than 2,5mm in diameter; - usually have a dark blue or purple color; - veins often protrude above the surface of the skin and can lead to pain, burning and spasms.

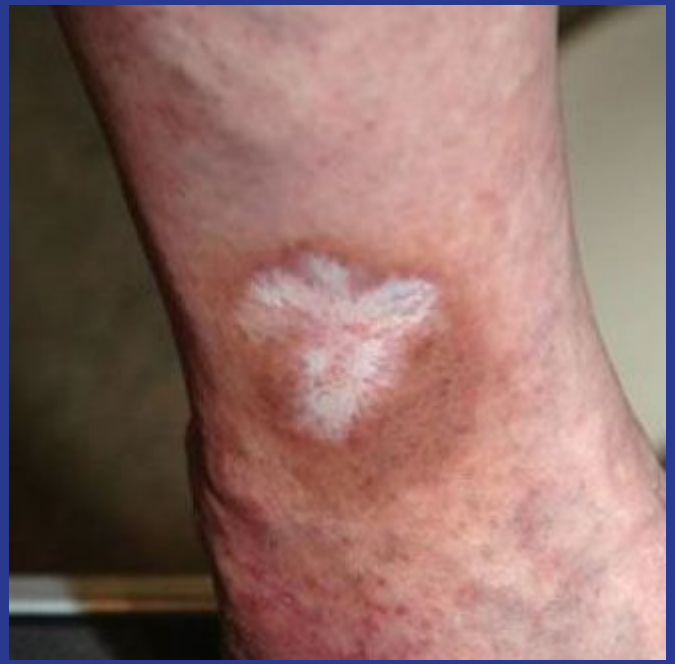


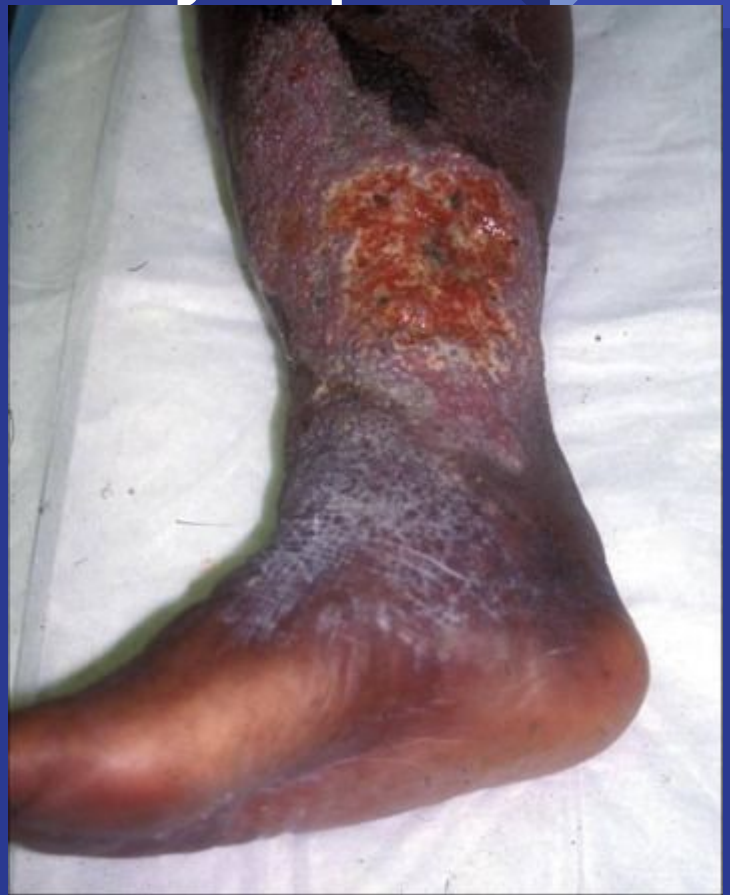
CVI: Signs and Symptoms





CVI: Signs and Symptoms

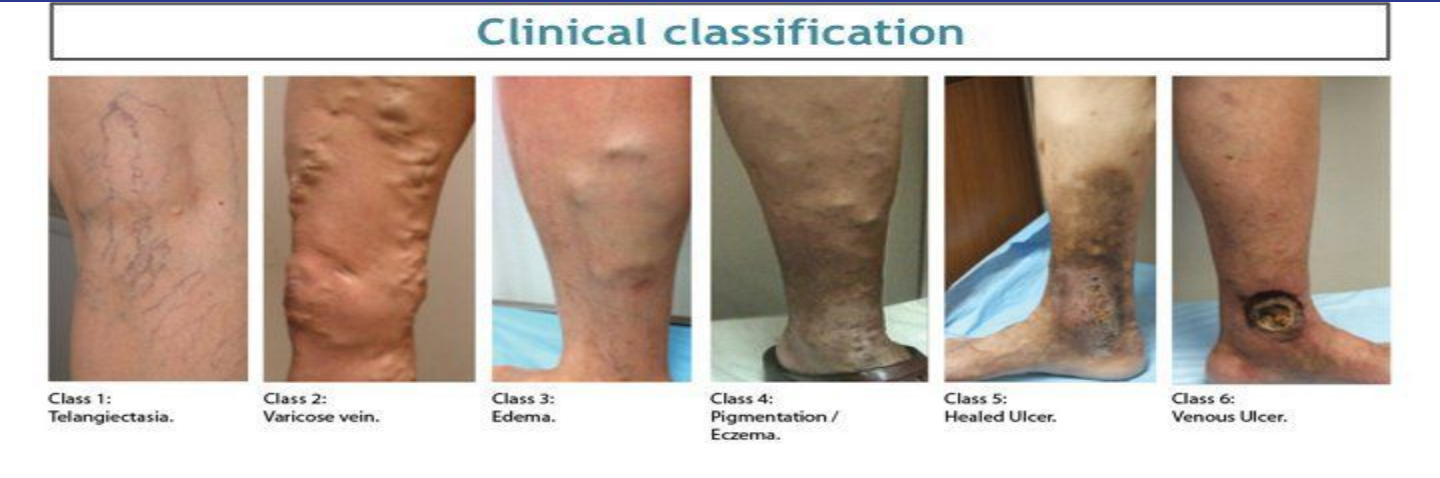




CVI: Classification Systems: CEAP

Clinical		
C0 – no visible or palpable signs of venous disease C1 – telangiectasies or reticular veins C2 – varicose veins C3 – oedema C4a – pigmentation or eczema C4b – lipodermatosclerosis or atrophie blanche C5 – healed venous ulcer C6 – active venous ulcer		
Each clinical class is further characterised by a subscript 's' for 'symptomatic' or a subscript 'a' for 'asymptomatic'. Symptoms encompass aching, pain, tightness, skin irritation, heaviness, muscle cramps and other complaints attributable to venous disease		
Etiology	**Anatomy**	**Pathophysiology***
Ec – congenital Ep – primary Es – secondary En – no venous cause identified	As – superficial veins Ap – perforating veins Ad – deep veins An – no venous location identified	Pr – reflux Po – obstruction Pr,o – reflux and obstruction Pn – no venous patho-physiology identifiable

CVI: CEAP


Clinical classification
Class 1:
Telangiectasia.
Class 2:
Varicose vein.
Class 3:
Edema.
Class 4:
Pigmentation /
Eczema.
Class 5:
Healed Ulcer.
Class 6:
Venous Ulcer.

CVI: Venous Clinical Severity Score (VCSS)

As a descriptive instrument, the CEAP classification responds poorly to change. The VCSS was subsequently developed as an dynamic evaluative instrument that would be responsive to changes in disease severity over time and in response to treatment.10 variables are examined and graded on a scale of 0-3, making the highest score possible is 30.

Attribute	Absent (0)	Mild (1)	Moderate (2)	Severe (3)
Pain	None	Occasional	Daily	Daily with medications
Varicose veins	None	Few	Multiple	Extensive
Venous edema	None	Evening only	Afternoon	Morning
Skin pigmentation	None	Limited, old	Diffuse, more recent	Wider, recent
Inflammation	None	Mild cellulitis	Moderate cellulitis	Severe cellulitis
Induration	None	Focal <5 cm	<1/3 gaiter	>1/3 gaiter
No.of active ulcer	None	1	2	>2
Size of active ulcer	None	<2 cm	2–6 cm	>6 cm
Ulcer duration	None	<3 months	3–12 months	>1 year
Compression	None	Intermittent	Most days	Fully compliant

CVI: Venous Clinical Severity Score (VCSS)

DLQI	Interpretation
0-1	No effect at all on patient's life
2-5	Small effect on patient's life
6-10	Moderate effect on patient's life
11-20	Very large effect on patient's life
21-30	Extremely large effect on patient's life
Score 0=no impact, score 30=maximum impact	

Thus, one can calculate the VCSS at the initial office visit, after venous treatments, and to follow the patient serially for months to years.

By using both the CEAP and VCSS classification systems, one can not only treat the patient, but can on a macro level make treatment comparisons more uniform and improve care.

CVI: Diagnosis

A thorough history and physical exam will be able to narrow the differential diagnosis immensely.
Questions to ask:

1). Do you have edema, pain, swelling, tightness, fullness?
2). Do you have itching, fatigue, weakness, restlessness? Do you have wounds? Where? Color changes?
3). If pain, location/severity/time of day/position of standing vs. sitting?
4). If edema, where and what time of day or position?
5). Do you wear stockings? If yes, how long? How often? OTC vs. prescription? Type of stockings and how much compression in mm Hg?
6). Any history of DVT? Any history of cellulitis? Any history of phlebitis?
7). Any history of claudication or rest pain?
8) Any previous venous studies?
9). Any previous venous treatments? If yes, where, how many, what type of intervention?

Exam:
Examine legs front and back, standing and sitting.
Still examine pulses, as a baseline, to rule out concomitant arterial disease.
Note the type of edema (pitting vs. non-pitting). Characterize the wound specifically and note dimensions.
Note the skin color and texture.

CVI:Venous Reflux

Current gold standard due to the speed of testing, reliability, easy reproducibility, and patient tolerance and it non-invasive nature.

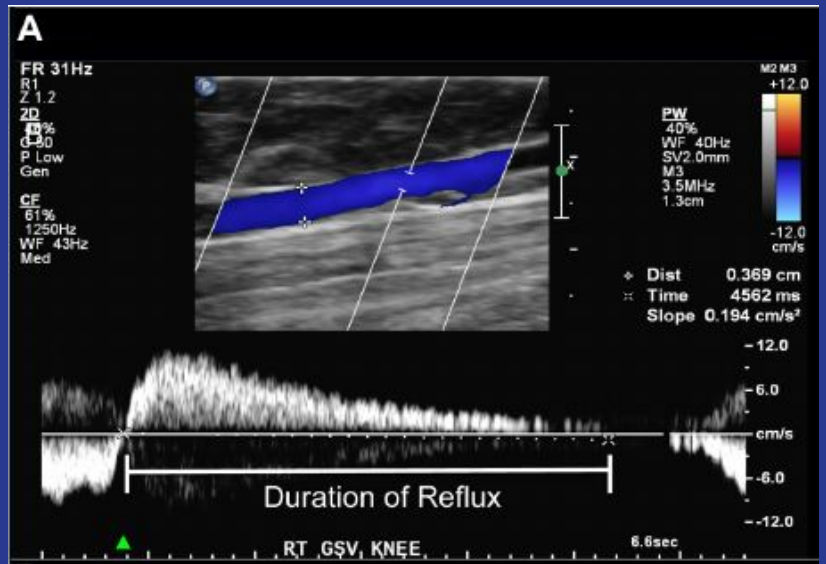
The standing position has been advocated as the best physiologic position for the DUS reflux examination because this position brings on symptoms in patients.

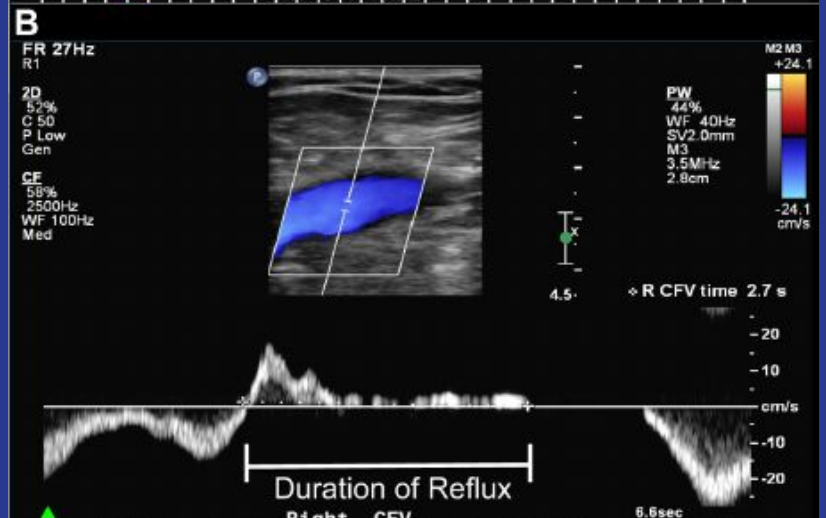
Venous reflux may be elicited by imaging the vein while applying compression to the limb with one of the following methods: release after a calf squeeze for proximal veins or a foot squeeze for calf veins.

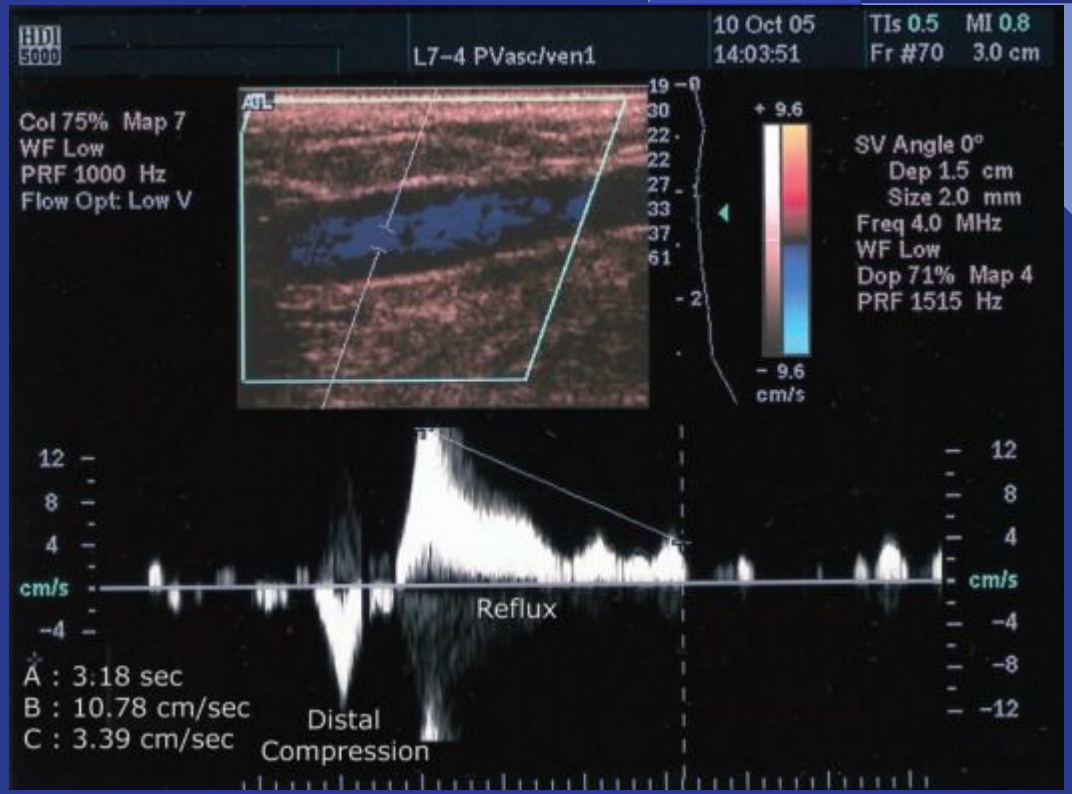
Compression with release (augmentation) distal to the point of insonation is a reliable method of evaluating for venous valvular incompetence (reflux). Compression is abruptly removed, and the presence and duration of reflux are observed.

Ultrasound-derived reflux time (RT) **>0.5 s for the superficial venous system and RT >1.0 s for the deep venous system**, including the common femoral and popliteal veins, have been widely used to indicate the presence of significant reflux

CVI: Venous Reflux Examples







CVI: Venous Reflux Report

Venous Insufficiency Worksheet

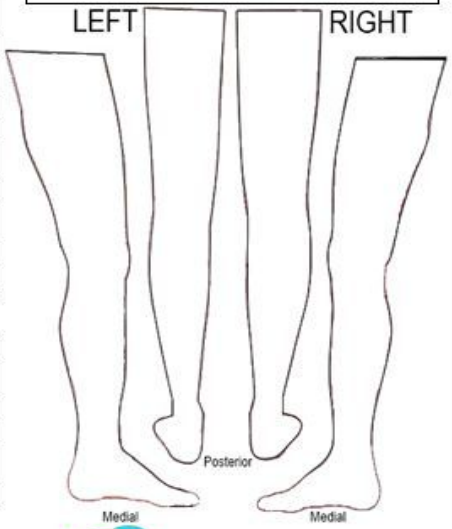
Name:_____ Date:____/____/____DOB: ____/____/_____

Referring Physician:_____Indications:_____Tech:_____

Vein	Diam (mm)	Reflux (msec)
CFV		
SFJ		
GSV Prx		
GSV MT		
GSV Knee		
GSV BK		
GSV Ankle		
Ant Acc GSV		
Post Acc GSV		
Giaco		
Pop V		
SPJ		
SSV Prox		
SSV Calf/Dst		
Ant Arch V		
Post Arch V		

Perforator	Diam (mm)	Reflux (msec)
Dodd's		
Boyd's		
Hunterian		
Cockett's #1		
Cockett's #2		
Cockett's #3		

Comments
DVT: ☐ Negative ☐ Positive ☐ Not Evaluated



All About Ultrasound

CVI: CT Venography

CTV/contrast-enhanced CT provides multiple extra levels of information, including the presence or absence of inferior vena cava (IVC) filters; the condition of the IVC, renal veins, collaterals, and internal iliac veins; potential iliac vein compression or nutcracker syndromes; presence of pelvic varicosities; as well as ovarian vein thrombosis and undiagnosed malignancy.

Potential difficulties with ordering CT venograms as the technicians and radiologists at some centers will just do a **CT angiogram without venous phase. Must explain clearly what you are ordering!**

CTV is an excellent screening tool for Deep Venous Reflux and can help make important clinical decisions on treatment such as focusing on the superficial venous system if the test is negative, or for planning interventional treatments in patients where the test is positive.

However, while it gives good data on extrinsic compression, it can be inaccurate when it comes to intravascular webs or scarring, and it does involve radiation and contrast use. Based on the limitations of CT venogram and clinical suspicion, one may require conventional venogram +/- intravascular ultrasound (IVUS).

CVI: Treatment of Superficial Reflux

Initial treatment in Clinical stage of 1-4 is conservative.

Conservative management:

Compression stockings:

At least 20-30 mm Hg. Wear daily and regularly. Wear at least knee high.
For those with thick ankles, calves or limited mobility, prescribe wrap type or zipper type for better compliance.
Cost may be a factor, as full price is >$80 and some insurances do not cover at all.

Elevation: Use coffee table to Ottoman. Elevate with pillows in bed. Higher the better.

Lifestyle changes: Avoid prolonged standing. Avoid dependent position.

Diet: Weight loss is an important part of venous care. Morbid obesity or severe obesity is a set up for presence of venous insufficiency, and failure of treatment as well.

In the absence of ulcers, should try conservative management for 6 weeks to 3 months. For most insurances, this is actually a requirement prior to approval of invasive procedures.

CVI: Treatment of Superficial Reflux

Former (historical): Vein stripping, high ligation.

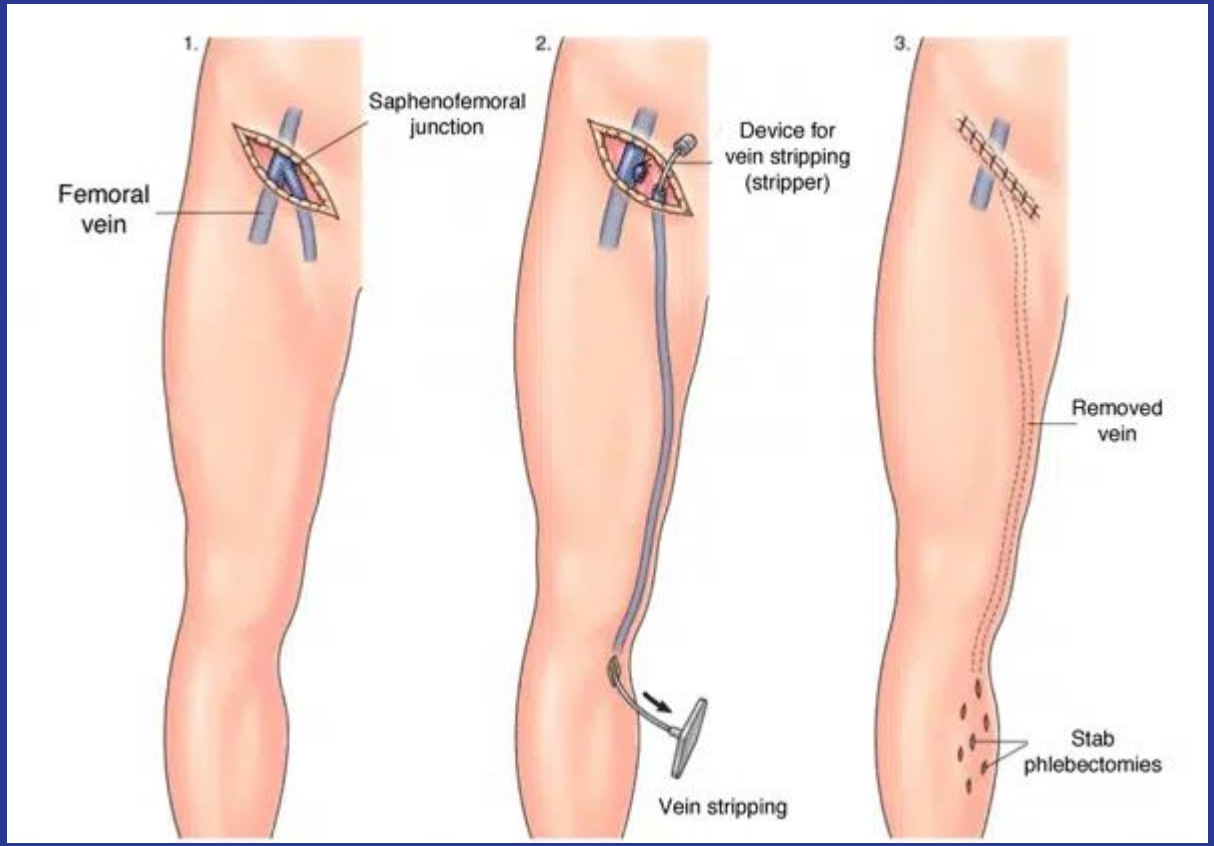
Modern treatments:

Thermal ablation: Laser (EVLT), RF ablation (RFA)

Non-Thermal therapies:

Compounded Foam sclerotherapy (Varithena),
Venaseal (Cyanoacrylate),
Mechanico-Chemical ablation (MOCA)/Clarivein

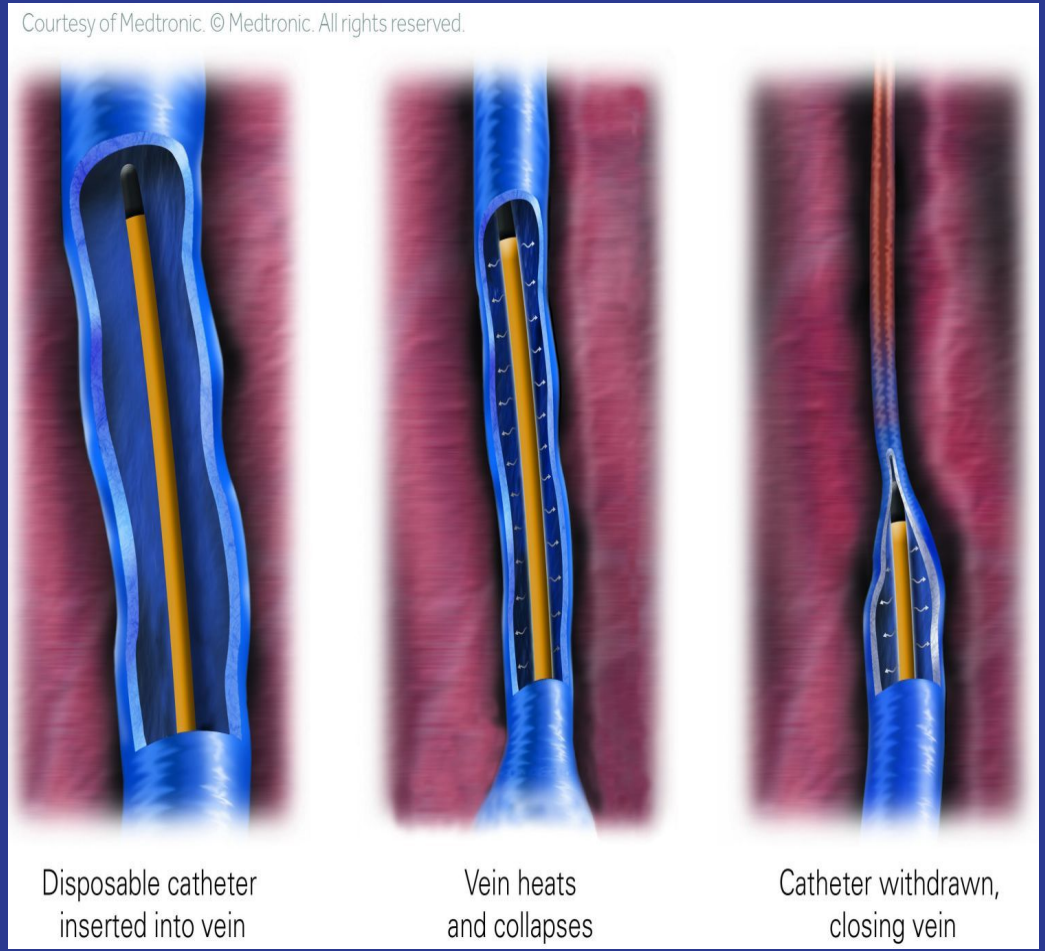
CVI: High ligation and Stripping



Persistent reflux in the below-knee GSV in all patients following stripping to the knee alone and residual visible residual varicose veins in 20% of patients.

Hematoma, cellulitis, edema, or thrombophlebitis can also occur in the residual thrombosed superficial veins, along with neuralgia.

CVI: Thermal Therapies–RF ablation



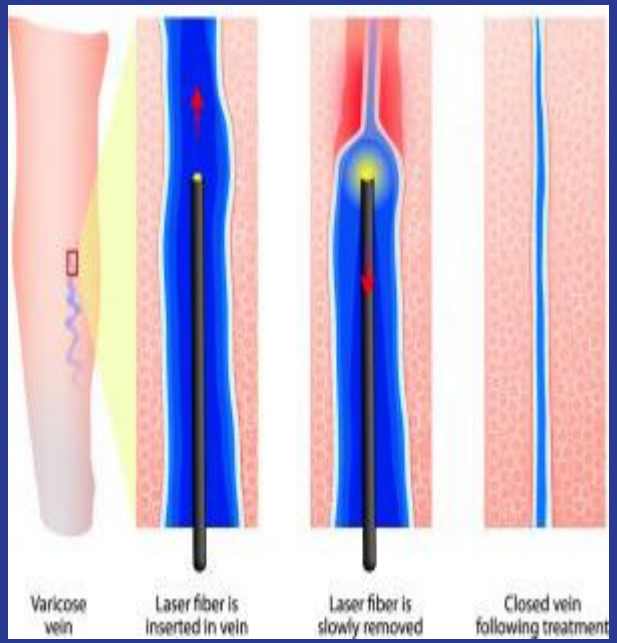
Use a micro-introducer set to place a 7F sheath in the GSV just below the knee.

Advance RF catheter from distal GSV to 2 cm from the SFJ.

Place tumescent anesthetic which is a combination of 0.5 mg adrenaline or epinephrine, 4.2 mg bicarbonate, and 35 ml lidocaine diluted in 500 ml 0.9 percent saline. This serves to insulate the surrounding soft tissue, nerves, deep vessels from heat injury.

The RF generator is then activated, which results in segmental heat energy of 120 degrees Celsius being applied. The RF generator is activated in 20-second intervals until the entire length of the vein is treated.

CVI: Thermal ablation–EVLT

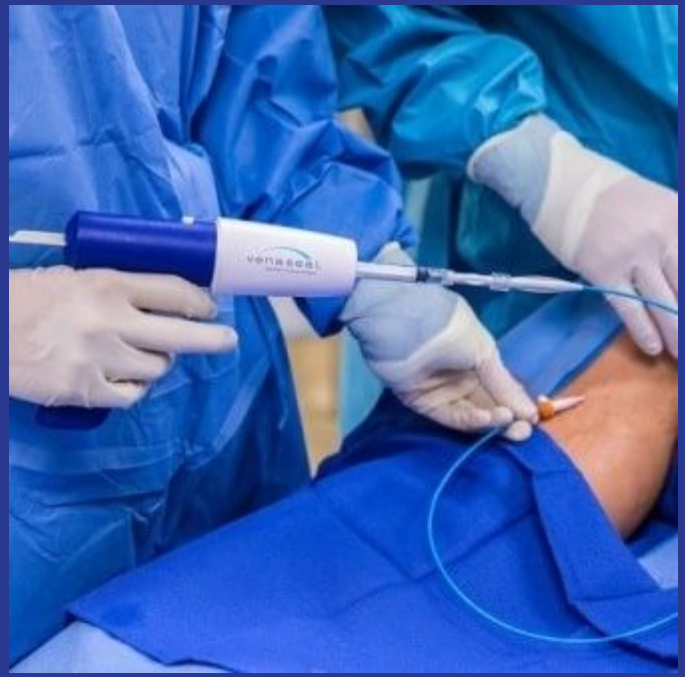


Same general technique as RF ablation in terms of access, tumescent anesthesia, placement of tip of laser catheter to 2 cm below SFJ.

Slow withdrawal of the catheter from superior to inferior and out the sheath.

Laser energy used is 1470-nm diode laser

CVI: Non-Thermal Therapies–Cyanoacrylate (Venaseal)

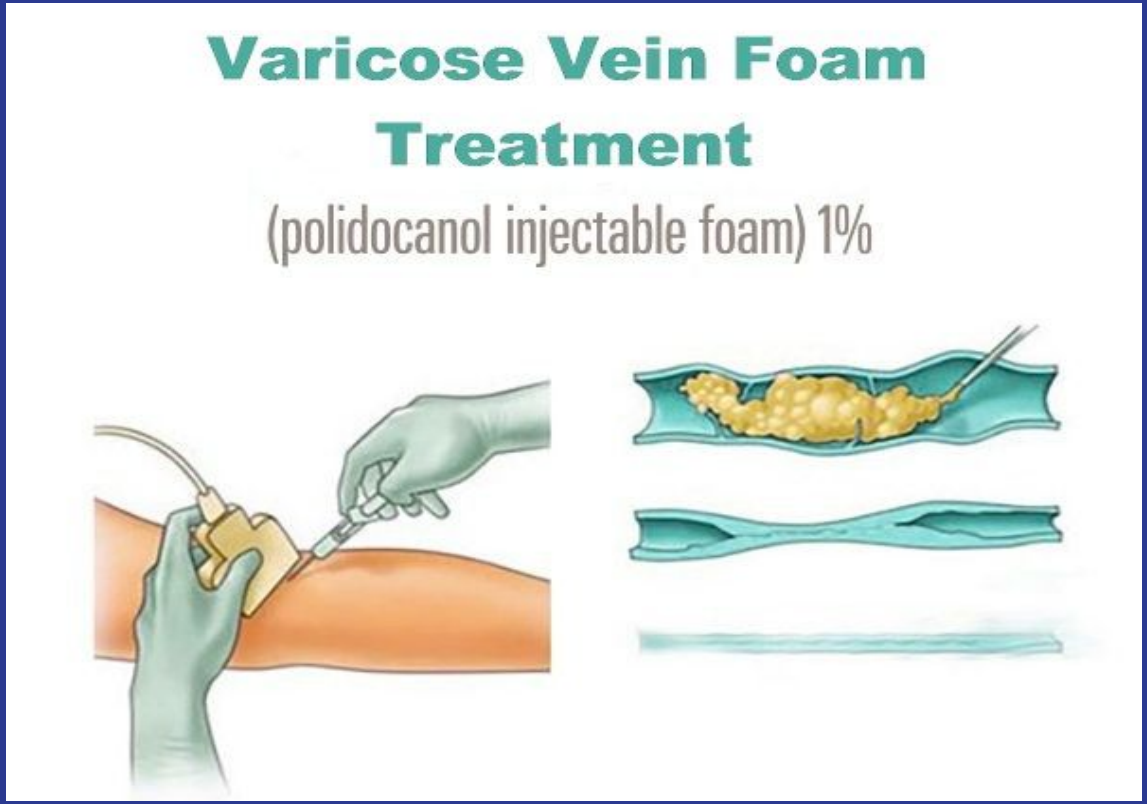


Two injections of approximately 0.10-mL cyanoacrylate glue were each given 1 cm apart at this location, followed by a 3-minute period of local compression with the right hand. Then, repeated single injections and 30 seconds of compression for every 3 cm distally.

Advantages: Only one access, no tumescent required. Cuts time in ½.Less bruising.

Disadvantages:
Allergic reaction in 5-10% with erythema/pain
Severe inflammatory reaction with wound infection or extrusion of glue through skin (rare, 1%). Given NSAIDS and Benadryl for 2-3 days proactively.

CVI: Varithena 1% Polidocanol



- O2:CO2 (65:35) gas mixture with <0.8% nitrogen
- Reliably small bubbles (median diameter <100 µm; all ≤500 µm)
- 7:1 gas: liquid ratio enhances blood displacement to allow for longer dwell time in the vessel.

The microfoam advances and fills 100% of the lumen. The foam then causes endothelial destruction and collapses the vein. The microparticles have a short dwell time and are quickly swept away by the venous system.

CVI: Mechano-Chemical Ablation (MOCA/Clarivein

The ClariVein system is the first venous ablation technique to employ a hybrid (dual-injury) technique built into 1 catheter-based delivery system.

Endomechanical abrasion is produced by the tip of the catheter's rotating wire (mechanical component); and endovenous chemical ablation (EVCA) is via simultaneous injection of sclerosant over the rotating wire (chemical component).

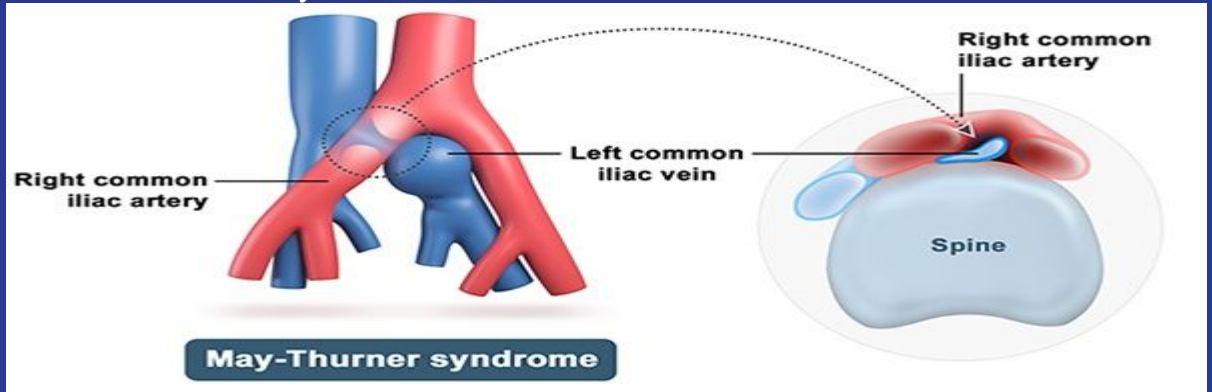
Unilateral treatment with MOCA in the short term resulted in less postoperative pain but more hyperpigmentation compared with RFA and a faster improvement in VCSS. More anatomic failures were reported after MOCA, mostly driven by partial recanalizations, but both techniques were associated with similar clinical outcomes at 1 year and 2 years.

CVI: Deep Reflux treatment for Iliac Vein Compression and/or Stenosis (Non-Thrombotic)

Deep Venous outflow obstruction may be secondary to iliofemoral deep vein thrombosis (DVT) or nonthrombotic iliac vein lesions (NIVLs, previously termed May-Thurner syndrome).

Nonthrombotic iliac vein lesions (NIVLs) most frequently result from extrinsic compression of various segments of the common or external iliac vein.

NIVLs are by definition extrinsic compression of the iliac vein, most commonly between arterial structures and the spine, without associated thrombosis; the compression results in intrinsic vein stenosis characterized by wall fibrosis or intraluminal webs/spurs. 10 The most common cause of NIVLs are commonly referred to as May–Thurner syndrome (MTS) or Cockett's syndrome, 11 usually due to compression of the left common iliac vein by the right common iliac artery.



CVI: NIVL's

NIVL may be clinically silent. Studies have found iliac vein compression present in approximately 70% of the asymptomatic population.

Patients with NIVLs can present with symptoms of CVI, such as leg discomfort/pain, edema, varicose veins, or ulcers. Females may additionally present with features of pelvic venous insufficiency, including pelvic pain and dyspareunia.

One can suspect NIVL in patient's with continued edema and pain and varicosities AFTER full treatment of superficial reflux by ablations, or with paucity of findings on venous reflux while working up chronic venous insufficiency. History of previous DVT may also cause suspicion.

The advent of intravascular ultrasound (IVUS) has significantly improved the accuracy of diagnosing NIVL. High-resolution imaging of the venous lesion with high probe frequency and close luminal distance can improve the detection of venous lesions such as webs or spurs. IVUS allows for more robust and accurate measurements of vein diameter and cross-sectional area.

Therefore, IVUS has become a critical component of endovascular NIVL management.

CVI: NIVL's

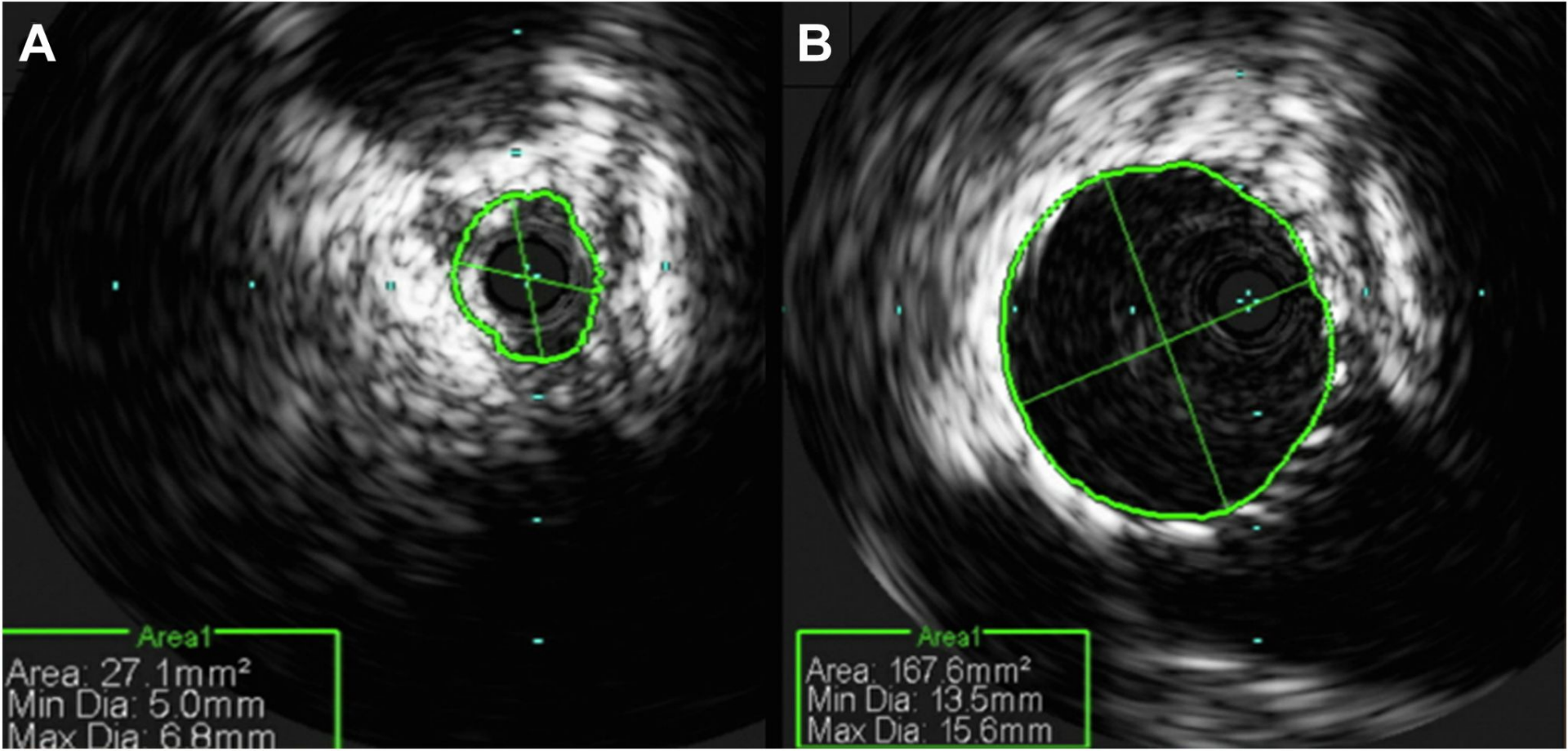
No intervention in asymptomatic individuals.

The goal of treatment is to resolve the obstructive lesion and reestablish good inflow and outflow. Balloon dilation of iliac vein stenosis without stent placement is not sufficient due to recoil of lesions, and stent placement is usually necessary.

Access is generally obtained via the ipsilateral common femoral vein or the Greater Saphenous Vein if available.

Next, a vascular sheath is placed. Venography may demonstrate the presence of collaterals, extrinsic compression, and prestenotic dilation; however, the findings can be relatively subtle. After initial venography, IVUS is used to assess the obstructive lesion and obtain measurements for stent planning. Subsequently, a self-expanding stent, sized based on adjacent normal segment (typically the normal ipsilateral external iliac vein in our practice), is deployed across the obstructive lesion and into the external iliac vein and sometimes into the common femoral vein if the lesion extends that far. If this known prior to venography and IVUS, one may want to do ipsilateral popliteal access.

CVI: NIVL's


A
Area1
Area: 27.1mm²
Min Dia: 5.0mm
Max Dia: 6.8mm
B
Area1
Area: 167.6mm²
Min Dia: 13.5mm
Max Dia: 15.6mm

CVI: NIVL's

Results on carefully selected symptomatic patients are excellent.

Edema relief rate of 89.1% and recurrence-free ulcer healing rate of 82.3% at a median of 4 years.

A systematic review reported primary patency of 94.1% at 12 months and 88.9% at 36 months.

Access-site complication presents the most common reported complication, and includes bleeding, hematomas, pseudoaneurysms, and arteriovenous fistulas. However, with ultrasound-guided puncture, the access-site complications are rare.

Up to 15% of patients may experience back pain, particularly during angioplasty or stent placement. Back pain is largely self-limited and treated readily with analgesics. With careful planning and 100% use of IVUS, stent migration and undersizing should be extremely rare.

If stents extending into distal IVC, to avoid "jailing" the contralateral side, may wish to consider "kissing" iliac vein stents to avoid this complication.

CVI:

THANK YOU.