

Workshop: Managing Varicose Veins
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3:30-4:25 PM

The format of this workshop will include a 30 minute presentation of the anatomy and physiology of the LE superficial venous system, a review of the pathophysiology of LE venous insufficiency and its patterns and an introduction to the clinical and imaging diagnosis of the pattern in a given patient. With the remaining 25 minutes we will have an open discussion about issues of interest to the audience. I will also bring some illustrative cases for review if time permits.

I have included some abstract material for the above topics for review.

Epidemiology and Pathophysiology of Lower Extremity Venous Insufficiency

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Introduction:

Lower extremity venous insufficiency is a common condition whose clinical manifestations vary from cosmetically displeasing to symptomatically disabling. Several well defined mechanisms are responsible for varicose veins. In this presentation the pathophysiology of venous insufficiency will be discussed and the common patterns reviewed.

A. Epidemiology:

It is estimated that between 30-60% of adults have some form of true lower extremity varicose veins. This incidence increases with age. If we include telangiectasias and venulectasias as part of lower extremity venous insufficiency, it is likely the some form of this disease is present in all adults at some point in their life. There is a higher incidence in women, which may reflect hormonal factors thought important in varicose vein formation, but may also reflect an under-reporting of the prevalence of the disease by men. Over 50% of patients who present for evaluation of peripheral venous insufficiency do so because of symptoms. Of those who present for cosmetic concerns, the majority also have symptoms when they are specifically sought for through questioning. Venous insufficiency is a predisposing factor in the development of superficial phlebitis as well as the cutaneous changes which can lead to venous ulceration. The careful management of patients with this disorder is essential for the prevention of such serious medical as well as cosmetic complications.

B. Pathophysiology:

In order to understand the pathophysiology of venous insufficiency, a thorough knowledge of the anatomy and physiology of the superficial veins of the lower extremity is required.

1. Anatomy of the lower extremity venous system:

The anatomy of the venous system of the lower extremity is conventionally divided into the deep and superficial venous systems. However, in the context of venous insufficiency they are best thought of as components along with the calf muscles of a complex and efficient vascular reservoir and pump system.

A. Superficial Veins

i. Supra-facial Collecting Veins

- subcutaneous thin walled structures superficial to the fascia
- drain into perforators or into the truncal superficial veins

ii. Truncal (Conduit) Superficial veins

a. Greater Saphenous Vein (GSV) and its tributaries

- 2 major tributaries below and above the knee
- 1/5 are duplicated in the thigh

b. Lesser Saphenous Vein (LSV)

- 2/3 drain into popliteal vein
- 1/3 drain to GSV or deep vein in thigh

B. Perforating Veins

Fairly typical locations

Connect the collecting veins and GSV with the deep system

Contain valves

C. Deep Veins

D. Calf Muscle Pump

2. Physiology of the normal lower extremity venous system:

The propagation of blood from the lower leg back to the heart occurs against substantial hydrodynamic forces. In the erect position the pressure created by the column of blood from the heart down to the ankle is on the order of 90-120 mmHg. Propagation toward the heart depends on compression of the deep veins by calf muscles and functional one way valves. The deep and superficial veins function as venous reservoirs which dilate substantially with minimal increase in their intra-luminal pressure. When the muscles in the leg, (especially the calf), contract the deep veins in the given compartment are externally compressed by extremely strong forces. Competent valves cause the blood to flow only in one direction in the deep veins toward the heart. When the muscles relax, the compartment pressures drop and blood flows into the deep veins from lower deep veins

and from superficial veins via perforators or at either the sapheno-femoral or sapheno-popliteal junctions (SFJ and SPJ). The calf muscles function as a “peripheral heart” with the valves in the perforators, truncal superficial, SFJ, SPJ and deep veins analogous to the cardiac and great vessel valves.

3. Pathophysiology of venous insufficiency:

Venous insufficiency develops when any component of the venous system fails. With failure, the thin walled superficial collecting veins are exposed to higher than normal pressures which results in excessive dilation and elongation.

A. Failure of valvular function.

Valvular failure is the most common cause of varicose veins. When valves fail, the normal refilling of any segment in the venous system is added to by blood refluxing from the segment above. This results in an elevation of the pressure and secondarily dilation in the segment. The contiguous valve apparatus will then dilate and fail. This is then transmitted back to the superficial collecting veins which dilate creating varicosities.

Valvular dysfunction is generally caused by heritable defects in valve (primary venous insufficiency) which can be potentiated by hormonal effects (including pregnancy) and prolonged standing or sitting. Previous thrombosis of the deep veins is a less common cause.

The pressures passed through incompetent valves result from:

- a. hydrostatic forces of blood from the segments above the incompetent valve in the superficial truncal as well as the deep veins, and by
- b. the much stronger hydrodynamic forces produced by the calf muscle pump through incompetent perforator veins.

Reflux leading to varicose veins can occur in (in the order of incidence),

- a. Superficial truncal veins, “*Typical Reflux*”, (approx. 70% of all cases)
 - i. Junctional Incompetence: saphenofemoral or saphenopopliteal junction
-secondary reflux in the valves within the saphenous veins
- b. Reflux in the valves within tributaries of the greater saphenous vein often secondary to SFJ or pudendal incompetence
- c. Perforator Incompetence: resulting in exposure the very high deep vein pressures to the saphenous vein and secondary saphenous incompetence
- d. Atypical patterns such as anomalous drainage of the LSV into the posterior medial saphenous tributary vein (Vein of Giacomini)

e. Deep veins, as a result of prior DVT or valvular trauma, agenesis or secondary deep vein valvular failure from vein dilation

f. Congenital anomalies: e.g.: Persistent vein of Labbe reflux in patients with KTS

B. Deep venous obstruction:

The calf muscle pump functions normally and generates sufficient pressure to move blood. However, the outflow tract is obstructed, resulting in pressure elevations, dilation and secondary valvular incompetence. These forces lead to perforator incompetence and varicose veins.

C. Failure of the Muscle Pump:

Neuromuscular diseases in which the muscles can not generate sufficient pressures to drive blood back toward the heart. This results in elevated venous pressures, venous dilation and valvular incompetence.

Superficial venous insufficiency: clinical patterns and duplex evaluation

Introduction:

The veins of the lower extremity are not passive conduits but rather components of a complex vascular pumping mechanism responsible for actively returning blood back to the heart against a substantial hydrodynamic gradient. This can be achieved only in the presence of functional valves, patent venous outflow tract and a functional venous pump. The failure of valves accounts for the majority of cases of venous insufficiency. In these cases, the pattern of clinical findings will depend on which valves fail. Effective treatment options exist but will only be successful if the appropriate therapy is selected for each patient. With a history and physical supplemented by careful Duplex imaging, the cause in a given patient can reliably be identified.

A. Clinical Evaluation:

In all patients a history should be obtained to identify what symptoms are present as well as to identify clues to the etiology of the problem. These will include a history of pregnancy, possible deep vein thrombosis, significant trauma or hypercoagulable syndrome. 70% of patients with varicose veins will have a family history of this problem. Such a history makes truncal vein insufficiency much more likely. An understanding of any previous therapy is also essential. The types of symptoms reported with venous disease include leg heaviness and tiredness, especially late in the day, night cramps, swelling and burning sensations. The duration and pattern of development of the manifestations of venous insufficiency may provide additional clues to the pathophysiology.

A physical exam should be performed in the erect position and should include the legs, pubic region and lower abdomen. Unfortunately, varicose veins caused by the different mechanisms frequently overlap in appearance. Some patterns are characteristic:

1. Junctional incompetence:
 - a. High saphenous pressures secondary to SFJ or SPJ reflux
 - b. Vein dilation from proximal to distal
2. Perforator Incompetence:
 - a. Most commonly from mid thigh or high calf perforator reflux
 - b. Varicosities start at this level and dilate distal to proximal
3. Lateral subdermal venous plexus related spider and varicose veins
 - a. Related to congenitally anomalous veins
 - b. Varicosities develop on lateral and posterior thigh or around the knee
4. Collateral veins related to chronic ilio-femoral vein thrombosis
 - a. Upper thigh, lower abdomen and pelvic collaterals
5. Pudendal vein incompetence
 - a. Most had vulvar varicies develop during pregnancy

- b. May cause secondary saphenous incompetence
6. Giacomini reflux:
- a. Varicosities form from the popliteal fossa medially to the upper third of the thigh from isolated reflux in this vein or secondary to GSV reflux

B. Imaging evaluation:

Although the location and distribution of varicose veins can occasionally suggest the cause, the appearance is rarely sufficient to make a confident diagnosis. Treatment based on assumptions made by physical examination alone will often be ineffective. Duplex ultrasound examination of the entire superficial venous system is essential. The goal of imaging is to map out abnormal pathways and to identify the source of incompetence. Duplex ultrasound allows identification of venous variants and facilitates diagnosis of atypical causes of reflux.

The Duplex exam is also performed standing with the patient's weight supported on their contra lateral limb. The leg to be examined is flexed and turned slightly outward. The exam begins from the top of the thigh to the level of the lowest varicosities. The SFJ is assessed for competency and the common femoral vein examined for evidence of prior DVT. The course of the GSV and its major tributaries are followed noting their size and whether there is any reflux. Reflux is sought using color Doppler as well as pulse wave Doppler immediately after an abrupt compression and subsequent release of a venous segment below the level being examined. Perforator communication between the GSV and the deep system is identified and their competency assessed. The location of the perforator veins in relation to the varicosities is noted; incompetent segments can be easily traced directly to the varicosities.

The patient is then turned away, and the LSV is examined. Again the knee is slightly flexed. The location of the termination of the LSV is established. The LSV may drain well above the popliteal vein in up to 1/3 of cases. The size and competency of the LSV and its junction are assessed and its relationship to posterior calf varicosities is noted. The popliteal vein is also examined for evidence of prior DVT.