CVD have reflux.\(^5\) Despite the diversity of signs and symptoms associated with chronic venous disease, it seems likely that all are related to venous hypertension. In most cases, venous hypertension is caused by reflux through incompetent valves, but other causes include venous outflow obstruction and failure of the calf-muscle pump.\(^4\) Reflux may occur in the superficial or deep venous system or in both. Valve damage may occur as the result of aging, extended sitting / standing or a combination of aging and reduced mobility. In many patients varicose veins will be present in conjunction with chronic venous insufficiency, but this is not always the case. There are many patients with typical changes of chronic venous insufficiency, but no obvious problem with their superficial veins. These patients may have abnormalities in the deeper veins which will only be apparent on special scans. The transmission of high venous pressures to the dermal microcirculation causes extravasation of macromolecules and red blood cells that serve as the underlying stimulus for inflammatory injury.\(^3\)

### Symptoms
- Ankle swelling
- Discomfort - tightness in calves, burning, itching, dull ache, or heaviness in affected calves or legs.
- Tired, restless legs
- Pain while walking or shortly after stopping (Pain that gets better when legs are raised).

### Signs
- Swelling of the ankles and/or calves
- Change in skin color, usually around the ankle
- Rash
- Signs Telangiectasia
- Reticular veins
- Varicose veins
- Pigmentation
- eczema
- Thickening and hardening of the skin on the legs and ankles (lipodermatosclerosis)
- Atrophie blanche
- Skin ulcer
**CEAP classification**

The CEAP classification was developed at a 1994 international consensus committee meeting sponsored by the American Venous Forum. Venous disease of the legs can be classified according to the severity, cause, site and specific abnormality using the CEAP classification. Use of such a classification improves the accuracy of the diagnosis and improves communication between specialists.

In 2000, Rutherford proposed a new tool to measure the severity of venous disease⁶. Its aim was to quantify the progression and treatment of chronic venous disease. These scores were mainly developed for severe forms of CVD according to the CEAP clinical classification (grades C4, C5, and C6), ie, for cases designated traditionally in terms of chronic venous insufficiency (CVI). These severity scores for CVD grade clinical severity, anatomo-pathophysiologic

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### Table-I

*CEAP Classification (Revised)*

<table>
<thead>
<tr>
<th>Clinical classification (C)</th>
<th>C0: no visible or palpable signs of venous disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C1: telangiectasias or reticular veins</td>
</tr>
<tr>
<td></td>
<td>C2: varicose veins (&gt;3 mm diameter)</td>
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<tr>
<td></td>
<td>C3: edema</td>
</tr>
<tr>
<td></td>
<td>C4: skin and subcutaneous tissue changes</td>
</tr>
<tr>
<td></td>
<td>C4a: pigmentation or eczema</td>
</tr>
<tr>
<td></td>
<td>C4b: lipodermatosclerosis or atrophie blanche</td>
</tr>
<tr>
<td></td>
<td>C5: healed venous ulcer</td>
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<tr>
<td></td>
<td>C6: active venous ulcer</td>
</tr>
</tbody>
</table>

Each clinical class is further characterized by a subscript for symptomatic (S) or asymptomatic (A)

<table>
<thead>
<tr>
<th>Etiologic classification (E)</th>
<th>Ec: congenital</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ep: primary</td>
</tr>
<tr>
<td></td>
<td>Es: secondary (postthrombotic)</td>
</tr>
<tr>
<td></td>
<td>En: no venous cause identified</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Anatomic classification (A)</th>
<th>As: superficial veins</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ap: perforator veins</td>
</tr>
<tr>
<td></td>
<td>Ad: deep veins</td>
</tr>
<tr>
<td></td>
<td>An: no venous location identified</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pathophysiologic classification (P)</th>
<th>Pr: reflux</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Po: obstruction</td>
</tr>
<tr>
<td></td>
<td>Pr,o: reflux and obstruction</td>
</tr>
<tr>
<td></td>
<td>Pn: no venous pathophysiology identifiable</td>
</tr>
</tbody>
</table>
severity and disability. Without a doubt, these severity scores deserve to be tested and evaluated by their potential users.

Diagnostic evaluation
Diagnostic evaluation of CVI can be logically organized into 1 or more of 3 levels of testing, depending on the severity of the disease:

- **Level I:** History and clinical examination, which may include use of a hand-held Doppler scanner.
- **Level II:** Noninvasive vascular laboratory testing, which now routinely includes duplex color scanning
- **Level III:** Invasive investigations or more complex imaging studies, including ascending and descending venography, venous pressure measurements, computed tomography (CT), venous helical scanning, or magnetic resonance imaging (MRI).

Measurement of ambulatory venous pressure remains an important invasive diagnostic tool and identifies the standing pressure nadir as well as the rapidity of emptying and refill. In practice, these examinations are often supplanted by more patient-friendly, noninvasive plethysmographic studies, which provide an objective measure of volume changes related to posture and to function of the calf muscle pump. However, as duplex ultrasonographic diagnosis has assumed an increasingly dominant diagnostic role, disease classification is often assigned on the basis of duplex scanning information alone. Magnetic resonance venography (MRV) offers yet another alternative for clarification of proximal diagnostic questions. The evolution of new imaging techniques in MRV and computed tomography venography (CTV) holds as much or more promise for elucidation of venous disease than of arterial disease. Both modalities require only venepuncture for image optimization.

Treatment
A. Conservative treatment
- Avoiding long periods of standing or sitting
- Intermittent elevation of the legs during the day and elevation of the legs at recumbency
- While sitting, leg should be above the thigh
- Maintaining ideal body weight
- Beginning a walking program
- Avoiding crossing the legs
- Compression therapy - Compression therapy using pumps, bandaging, and/or graded compression stockings is the mainstay of treatment
- Micronised purified flavonoid fraction (Diosmin + Hesperidin)

B. Vein ablation treatments
- Sclerotherapy-(Sodium tetradecyl sulfate, Sodium morrhuate, Hypertonic solution of sodium chloride, Polidocanol, Sodium iodide, Ethanolamine oleate)
- Radiofrequency ablation
- Laser ablation

C. Surgical procedures
- Vein stripping
- Ambulatory phlebectomy
- Bypass surgery
- Valve repair
- Valve Transplantation
- Valve Transposition
- Valve Substitutes
- Linton’s operation
- Endoscopic ligation of perforators
- Stenting

Many patients without symptoms or signs of lipodermatosclerosis or ulceration simply require reassurance. Others presenting for treatment are concerned about the cosmetic appearance of their limbs. Patients should be given a trial of graded compression stockings and further instructions to avoid prolonged standing and to elevate the feet above the heart while lying down as well as several times during the day. Stockings in the lower pressure range (class 1, 20 to 30 mm Hg) are sufficient to control edema, but higher pressures (class 2, >30 to 40 mm Hg; or class 3, >40 mm Hg) are recommended to control venous dermatitis or ulcers. In a prospective randomized double-blind controlled trial, micronised purified flavonoid fraction did not change the symptoms of CVI except for nighttime cramping. Surgical therapy for CVI should be considered in patients who have failed less invasive treatments. Patients with varicose veins and superficial venous reflux can undergo saphenous vein high ligation and stripping or other endovascular techniques along with branch varicosectomy to remove the offending reflux. Alternatively, sclerotherapy or other minimally invasive techniques can be used in some cases. Surgical correction of superficial venous reflux in addition to compression bandaging does not improve ulcer healing but reduces the recurrence of ulcers.

Prevention
Certain factors such as genetic predisposition, gender and increasing age are unavoidable. Weight control, adequate
physical exercise, avoidance of smoking, avoidance of sedentary activities and control of hypertension may all be significant in preventing the development of varicose veins.

References: