Venous Reflux Disease

About Venous Disease: Understanding Superficial Venous Reflux
In venous insufficiency states, venous blood escapes from its normal upward path of flow and refluxes backward down the veins into an already congested leg. Venous insufficiency syndromes are caused by valvular incompetence in the high-pressure deep venous system, low-pressure superficial venous system, or both. Untreated venous insufficiency in the deep or superficial system causes a progressive syndrome involving pain, swelling, skin changes, and eventual tissue breakdown.
Deep venous insufficiency usually occurs when the valves of the deep veins are damaged as a result of deep venous thrombosis (DVT). With fewer valves to prevent deep system reflux, the hydrostatic venous pressure in the lower extremity increases dramatically. This condition is often referred to as a postphlebitic syndrome.
Superficial venous incompetence is the most common form of venous disease. In superficial venous insufficiency, the deep veins are usually normal, but venous blood escapes from the deep system and flows backwards through dilated superficial veins in which the valves have failed.
The valves in superficial veins can fail for a variety of reasons. Direct injury or superficial phlebitis may cause primary valve failure. Congenitally weak vein walls may dilate under normal pressures to cause secondary valve failure. Congenitally abnormal valves can also be incompetent at normal superficial venous pressures. Normal veins and normal valves may become excessively distensible under the influence of hormones (as in pregnancy).
Most cases of superficial vein valve failure occur after a single point of high-pressure leakage develops between the deep system and the superficial system high up in the groin. High pressure causes secondary valve failure when otherwise normal superficial veins become so widely dilated that the thin flaps of the venous valves can no longer make contact in the lumen of the vessel. Over time, these incompetent superficial veins become visibly dilated and tortuous, at which point they are recognized as varicose veins.
High pressure can enter the superficial veins as a result of the failure of key valves at any point of communication between the deep system and the superficial system. The 2 major sources of high-pressure leakage from the deep veins to the superficial system are junctional valve failure and perforator valve failure.
Junctional high-pressure disease most often results from failure of the primary valve at the junction between the greater saphenous vein and the common femoral vein at the groin (saphenofemoral junction). Vein incompetence then proceeds distally from the groin, and patients perceive that a large vein is growing down their leg. A less common form of junctional reflux results from failure of the primary valve at the junction between the short saphenous vein and the popliteal vein at the knee (saphenopopliteal junction).
Perforator high-pressure disease results from failure of the valves of any perforating vein. The most common sites of primary perforator valve failure are in the midproximal thigh (Hunterian perforator) and in the proximal calf (Boyd perforators). When the primary high-pressure entry point is distal, large clusters of veins are first noticed in the lower leg, with large veins eventually growing up the leg toward the groin.

Pathophysiology: When venous valves are working correctly, every movement of the leg causes blood to be pumped inward and upward past a series of valves. During ambulation, the normal pressure in the venous system of the lower leg is nearly zero. Immediately after ambulation, the early standing pressure in the normal leg remains low. Arterial inflow fills the leg veins slowly, and the only source of venous pressure is the hydrostatic pressure of a column of blood as high as the nearest competent valve. After prolonged standing, the veins are completely filled, and all the venous valves float open. At this time, high hydrostatic venous pressure results from the unbroken column of fluid that extends from the head to the foot.
Failed valves cause the column of standing blood in the vein to remain high even when during ambulation. The hydrostatic pressure increased during and immediately after ambulation.
High venous pressure is directly responsible for many aspects of venous insufficiency syndrome, including edema, tissue protein deposition, perivascular fibrin cuffing, red cell extravasation, impaired arterial inflow, and other locally mediated disturbances.
Not all of the sequelae of venous insufficiency are related to venous hypertension, and not all patients with venous hypertension develop ulceration. Some patients with venous ulceration do not have marked venous hypertension.
The poor clearance of lactate, carbon dioxide, and other products of cellular respiration also contribute to the development of the syndrome. A defect in the clearance of extraneous substances can be quantified: If albumin labeled with a radioactive tracer is injected into the foot tissues, the clearance rate is markedly slowed by deep venous obstruction or by deep or superficial venous incompetence. Although this effect is referred to as venous stasis, the reduced clearance of cellular metabolites is not always due to true venous stasis. In many cases, the venous blood is moving at a normal speed, but a local recirculation of this venous blood upward through normal veins and downward through varicosities prolongs the average transit time for the blood to pass from the heart and lungs through the legs and back to the central circulation.
The time required for an aliquot of radiolabeled blood to pass from the femoral artery through the leg and back to the central circulation is highly correlated with the development of leg ulcers. The aliquot transit time and the clearance time for an extremity are closely related to the volume of retrograde flow through refluxing veins. Superficial varicosities always produce venous recirculation and can result in prolonged clearance that may be localized or affect the whole leg. Experimental evidence shows that if the peak retrograde flows in the greater and short saphenous veins and popliteal vein add to less than 10 mL/s, progressive visible stasis dermatitis and ulceration do not occur. If the sum is greater than 15 mL/s, the incidence of ulceration is high. In some cases, purely superficial local reflux with a

pressure of more than 7 mL/s can cause local ulceration.
Chronic nonhealing wounds of the lower extremity have many different potential causes, but most chronic lower-extremity ulcers are of venous etiology. Most venous ulcers are caused by venous reflux that is purely or largely confined to the superficial venous system. Only a minority are caused by chronic DVT or by valvular insufficiency in the deep veins.
Mortality/Morbidity: The syndromes of venous hypertension and reduced venous clearance are important causes of morbidity and disability in patients with varicose venous disease.
Chronic nonhealing leg ulceration can be debilitating. Approximately 1 million Americans have an ulceration due to superficial venous disease, and approximately 100,000 are disabled because of their condition.
As many as 50% of patients with untreated varicose veins develop superficial thrombophlebitis at some time. This is of grave concern, because unrecognized DVT is present in as many as 45% of patients with what appears to be purely superficial phlebitis. The risk of DVT is 3 times higher in patients with superficial varicosities than in the general population.
Bed rest and intercurrent illness place patients with venous insufficiency at higher risk for DVT. Phlebitis develops in 60% of hospitalized patients with clinically evident superficial venous insufficiency, and in nearly one half of cases, the condition progresses to DVT. Approximately one half of patients with DVT have detectable pulmonary embolism, and the mortality rate in this group exceeds 1 in 3.
Bleeding from lower-extremity varicosities can be fatal. Twenty-three such fatalities were reported in England and Wales in 1971, and although there is no central registry to tabulate the frequency with which it occurs, such cases are not unusual in the United States. Bleeding is not a rare problem, but often is managed incorrectly.