CHRONIC ULCERS OF THE LEG OF VENOUS ORIGIN

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IT is estimated that 600,000 ulcers of the leg are treated annually in the United States—an estimate that is extrapolated from a reported incidence of 15,000 ulcers of the leg treated annually in Denmark.[1] These vast numbers argue a problem, the magnitude of which never has been evaluated in terms of physical disability and economic loss both to the individual and to the community. This study will present the current beliefs and practices in regard to the origin and treatment of the disease.

Classification of ulcers of the lower extremities. On the basis of etiology, six categories of ulcers of the lower extremities are recognized, as follows[2]:

I. Arterial
   A. Organic origin
      1. Arteriosclerosis obliterans with or without diabetes
      2. Thromboangiitis obliterans
      3. Embolic or thrombotic occlusion
      4. Local pressure
   B. Spastic origin
      1. Raynaud's disease
      2. Scleroderma
      3. Frostbite
      4. Local arteriospasm, traumatic or occupational

II. Venous
   1. Varicose ulcers
   2. Phlebitis
   3. Congenital anomalies

III. Specific ulcers
   1. Syphilis
   2. Tuberculosis
   3. Mycosis
   4. Drugs
   5. Vitamin deficiency
   6. Neurotrophic ulcers
   7. Infection
   8. Gout
   9. Regional enteritis
   10. Chronic ulcerative colitis

IV. Posttraumatic ulcers
   1. Occupational
   2. Factitial

V. Blood dyscrasia
   1. Polycythemia
   2. Pernicious anemia
   3. Leukemia
   4. Sicklemia

VI. Malignant ulcers

This study is concerned chiefly with chronic ulcers in the second category: ulcers of the leg of venous origin.

Anatomy and pathogenesis. An understanding of the fundamental anatomy and pathologic physiology is the basis of rational therapy. The venous anatomy of the lower extremity is composed of three systems: a superficial system, a deep system, and one that interconnects between these two by means of perforator or communicating veins. Normally, the three systems contain a varying number of valves that prevent venous reflux of the blood (Fig. 1). The patency of the veins and the competence of the valves assure the successful return of blood from the extremity. With incompetent valves—either as the result of congenital absence or of infection—retrograde flow through the veins occurs and other pathologic changes are prone to follow.

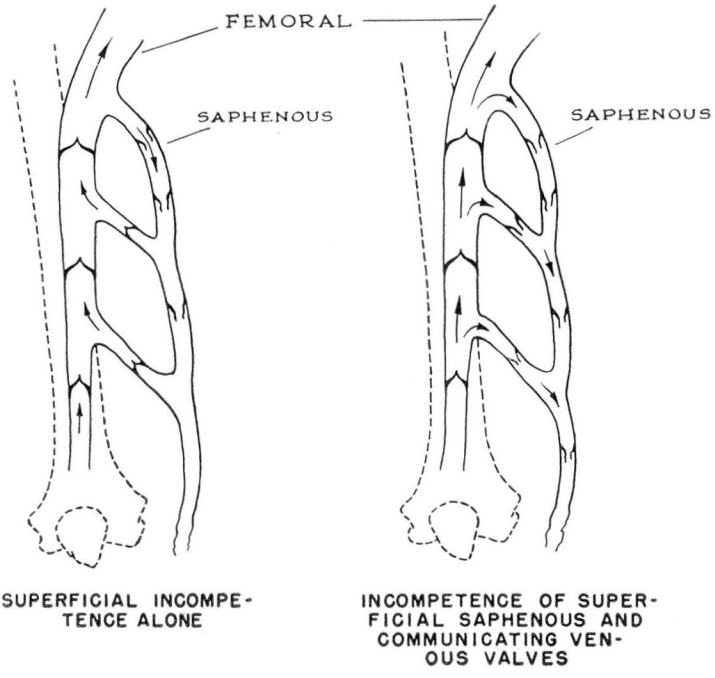

Fig. 1. Types of superficial venous and perforator venous valvular incompetence.

The superficial venous system is composed of the greater saphenous system anteromedially, and the lesser saphenous system posteromedially. The greater saphenous system empties into the deep system at the common femoral vein; the lesser saphenous system is tributary to the deep system at the popliteal vein. The deep system begins in the lower leg as the anterior and the posterior tibial veins which join to form the popliteal vein. In the adductor canal the popliteal vein is known as the superficial femoral vein which unites with the deep femoral vein of the thigh to form the common femoral vein continued in the pelvis to the vena cava as the external iliac vein. Encased in musculo-fascial envelopes, the deep veins dilate only slightly with venous reflux. Hence, when deep-vein valves are incompetent, the reflux of blood flow is forced through the communicating veins into the superficial veins, producing dilatation and subsequently valvular incompetence.

The three systems are intimately associated with the lymphatics that drain the tissue fluids of the extremity. Thus, damage to the veins by infection often is accompanied by lymphatic damage and impairment of drainage of the tissue fluid, resulting in stasis changes.

Stasis changes are those local tissue abnormalities that result from chronic venous reflux and inadequate lymphatic drainage of the area. Centrally in the involved tissue there may be ulceration, surrounding which is a zone of tender, brawny induration known as the zone of stasis cellulitis. Throughout the area are subcutaneous fibrosis and hemosiderin pigmentation that produce a characteristic leathery consistency and tan-to-purple appearance. Often an extensive dermatitis is present, designated as stasis dermatitis (Fig. 2).

Beecher,[3] DeCamp and his associates,[4] Stürup and Højensgard,[5] and others have studied the venous pressures (erect) in the normal leg; in the leg with superficial varices; and in the leg with deep-vein incompetence—that is the "postphlebitic leg." In the resting leg of each (erect) the venous pressure at the foot is equal to the weight of a column of blood from the foot to the level of the right atrium. During walking, in the leg with venous incompetence, whether from superficial varices or deep-vein incompetence, there is constant elevation above normal of the venous pressure (Table). The elevated venous capillary

TABLE

VENOUS PRESSURE (Cm. of H_2O)*

Pressure	Light Exercise, Walking	
	Normal Subjects	Subjects with Varicose Veins
Maximum	75	96
Minimum	28**	96
Resting	63	90
Pulsation	49	0

* *Taken from Beecher.*[3]

** *Persists throughout 2/3 of the step.*

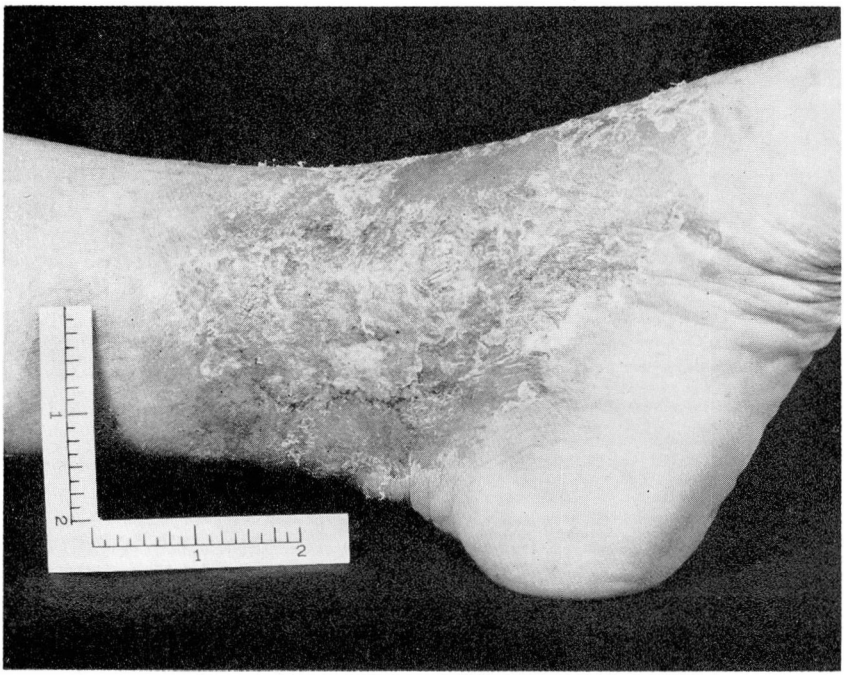

Fig. 2. Stasis dermatitis and ulceration.

hydrostatic pressure then may exceed the colloidal force that normally tends to draw tissue fluid into the capillaries. When the hydrostatic pressure exceeds 60 cm. of water, serum protein is forced into the tissue, thus creating an excess of protein-rich intracellular fluid. Fortunately, the normal lymphatics are capable of rapidly draining away the damaging excess of protein-rich tissue fluid. Hence, if the lymphatics are undamaged, there may be extensive varices with no evidence of stasis change.

The lymphatics, however, often are involved in phlebitis—superficial or deep—and become incapable of removing the excess of protein-rich tissue fluid that collects. Edema then occurs, and the protein-rich tissue fluid not only stimulates fibrosis but acts as an ideal culture medium for infection that furthers fibrosis. As the fibrosis progresses, local blood supply and tissue nutrition diminish, and the slightest trauma or infection is capable of producing ulceration. In the presence of edema there is excessive moisture of the leg and foot, which encourages the growth of fungal and bacterial infections. These infections, once present, produce recurrent lymphangitis with further lymphatic obstruction and also, directly or by antigenic activity, produce ulceration in the tissues damaged by stasis change. Thompson[6] demonstrated that autogenous tricophyton antigens obtained from an infected foot or toenail (athlete's foot) when applied to a distant part of the body could produce a flare of the stasis dermatitis of the leg.

When increased capillary pressure and infection have crucially damaged the

vessel walls, erythrocytes may collect in the tissues and deposit hemosiderin. Unusual pigmentation probably is due to such deposits of hemosiderin.

The pathogenesis of ulcers of the leg that are due to venous incompetence may be summarized as follows: Venous incompetence permits venous reflux with resultant increase in capillary hydrostatic pressure. Proteins diffuse into tissues and, because of inadequate lymphatic drainage, produce edema, stimulate fibrosis, and serve as culture media for infection. Fibrosis and infection result in ulcerations.

Treatment. The objectives of treatment of chronic ulcers of the leg are based on elimination of the five pathologic conditions that have been described, and on prevention of recurrence. Thus, the steps in treatment are elimination of: 1) infection; 2) dermatitis; 3) edema; 4) venous reflux; 5) stasis fibrosis; and 6) prevention of recurrence of ulceration.

Initially, nonoperative treatment is indicated in all patients. Depending upon the severity of stasis changes, ambulation may be permitted, or bed rest may be required.

In the ambulatory patient, the zone of ulceration, the entire leg, and the foot are gently cleansed with phisoderm. The ulcer is then coated with a drying bacteriostatic dye such as a 3 per cent aqueous gentian violet solution, and is covered with fine-mesh gauze and several bulky gauze compresses. To control the edema and provide a bland dressing for the leg, a gelatin boot is applied from the base of the toes to just below the knee, and over this a layer of gauze bandage is fixed with spirals of adhesive tape. The patient is instructed to cleanse between the toes, and around each toenail daily, and to apply an antifungicidal ointment or powder each morning and night. The boot is changed at weekly intervals, with repetition of cleansing and application of gentian violet. The ulcer usually will heal within a few weeks' time. The treatment, however, is continued for several weeks after the ulcer has healed, and thereafter the patient continues to wear an elastic support.

If the ulcer is extensive or so complicated by dermatitis, infection, or edema that ambulatory treatment is not feasible, then bed rest is necessary. The foot of the bed is elevated 4 to 6 inches. The zones of dermatitis and ulceration are gently cleansed with phisoderm, and throughout the day moist saline pressure dressings are applied. At night, a dry, fine-mesh gauze is applied over the area with several layers of overlying compresses, and an elastic bandage is snugly—but not tightly—wrapped from toe to knee. This therapy is continued until healing is sufficiently advanced to permit ambulatory treatment.

Wet saline dressings may aggravate severe dermatitis, and occasionally one must resort to a bland ointment such as vioform ointment or one-half strength coal tar. The use of chlorophyll, antibiotic, or enzyme ointments has not increased the rate of healing significantly; moreover, allergic or chemical reaction may occur and aggravate the dermatitis.

Thus, in the nonoperative treatment, infection is combated by cleanliness and fungicides, and occasional use of systemic antibiotics. The edema is controlled by bed rest and supportive dressings which also act to prevent venous

reflux and congestion. Continuation of support to the leg after the ulcer has healed acts to prevent recurrence of ulceration and progress of the stasis fibrosis.

The operative treatment follows or is combined with the nonoperative treatment. The type and degree of venous incompetence and the patency of the deep veins are determined. A history of postpartum, postoperative, or posttraumatic deep-vein phlebitis is indicative of deep-venous incompetence, as is the presence of incompetent perforating veins. Inspection and palpation of the veins demonstrate superficial varices. The Trendelenburg test and the multiple tourniquet test demonstrate the level of superficial and perforator incompetence. Deep-vein patency is demonstrated by the Perthes' test. Rarely, a venogram may be necessary to establish deep-vein patency. One may assume that one year after an episode of deep-vein phlebitis, recanalization of the vein will have been completed and the deep vein again is patent though it remains incompetent. Ligation of superficial varices is postponed until the deep veins are demonstrated to be patent.

Preoperatively, the greater saphenous system or both saphenous systems and the sites of incompetent perforators and superficial tributaries are marked with the patient erect, using a dye such as 3 per cent aqueous brilliant green. The operation is performed with ligations at the sapheno-femoral junction of the greater saphenous veins and of all tributaries in the area. The main channel of the saphenous vein is stripped using an intraluminal stripper, and then all veins that were marked but not stripped are locally ligated and transected, with particular attention paid to the perforator veins. The lesser saphenous system is treated in similar fashion. The operation may involve 15 to 20 local ligations, over a period of two or more hours, so that general anesthesia is used, or the operation is completed in stages using local anesthesia. Postoperatively, the leg is wrapped from toes to thigh with a snug pressure dressing, and the patient is encouraged to be ambulatory. Sutures are removed seven to ten days postoperatively. The nonoperative treatment of the ulcer is continued until one month after the lesion has been healed.

Successful treatment of deep-vein incompetence by ligation of the popliteal and the superficial femoral veins has been reported[7,8]; however, our own results with these procedures have not been satisfactory.

The large chronic ulcer with stasis fibrosis and marked skin change ultimately will heal with bed rest and ligation of superficial varices; however, it tends to recur repeatedly. In patients having that condition, a wide excision of the zone of ulceration and the zone of stasis fibrosis is performed extending down to the healthy musculo-fascial compartments. The defect is covered with a split-thickness skin graft from the thigh, which provides a healthy skin. These patients must continue to wear elastic supports. If the support proves to be inadequate, by permitting edema to occur with recurrence of ulceration even in the skin graft, then an Aeropulse legging is used.

Aeropulse legging, as described by Merle Scott of Rochester, New York, eliminates the edema and permits rehabilitation and return to work of patients who for several years may have been classified as 100 per cent disabled.

Finally, in the treatment of chronic ulcers of the leg that are due to venous insufficiency, the patient must be impressed with the unpleasant fact that he does not have and never will have a normal leg. He must at all times take such precautions as are necessary to prevent edema and infection, since the presence of either of these conditions will lead to recurrent ulceration. Good elastic support, whether it be a gelatin boot, elastic bandage, elastic stockings, or an Aeropulse legging must be worn during the waking hours. The extremity should be elevated as frequently as possible above hip level. Foot hygiene with control of fungal infection is imperative.

SUMMARY

Ulcers of the lower extremities are classified into six categories according to origin: arterial, venous, specific, posttraumatic, blood dyscrasia, malignant. The pathogenesis of chronic ulcers of the leg of venous origin begins with venous incompetence that is congenital, or arises from trauma or infection. There follow pathologic changes in this order: venous reflux, increased capillary hydrostatic pressure, excess protein diffusion into the tissues, inadequate drainage by the lymphatics, and subsequently edema, fibrosis, infection, and ulceration. Treatment of chronic venous ulcers of the leg initially is nonoperative in all patients. Cleanliness and supportive dressings are indicated, rarely antibiotics. Bed rest also may be necessary. Operative treatment may follow or it may be combined with nonoperative treatment. The patient must be carefully instructed in the care of the extremity: prevention of edema, maintenance of cleanliness, and avoidance of trauma and infection. Where necessary, special elastic or pneumatic supports must be employed.

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