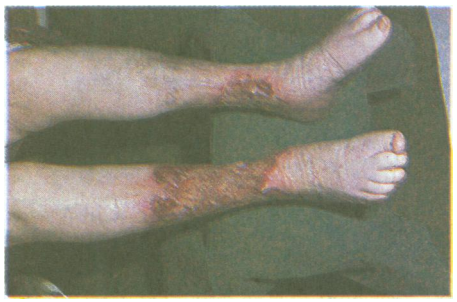
ABC of Dermatology

P K BUXTON

LEG ULCERS

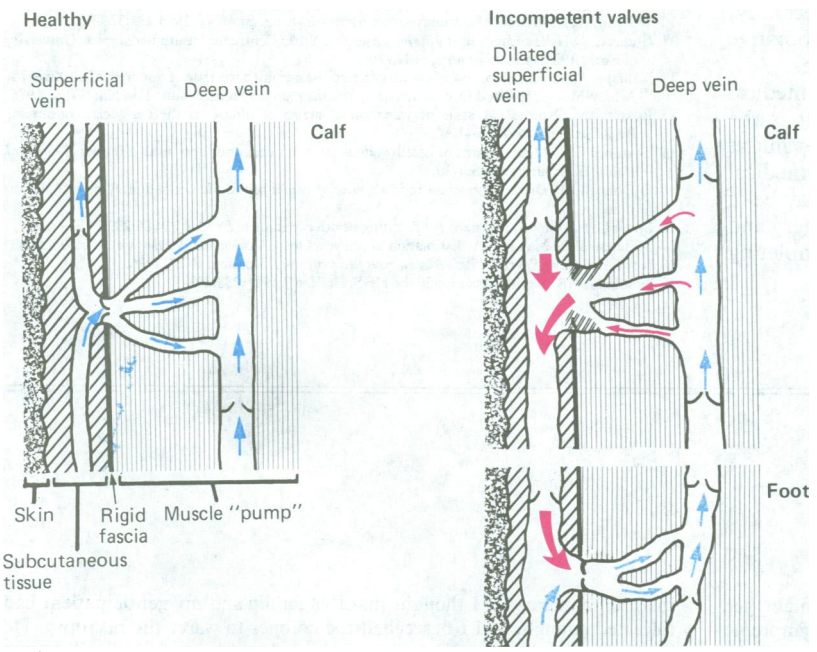


Although the patient will not probably die of this disease, yet, without great care, it may render her miserable. The disease may be very much relieved by art, and it is one of very common occurrence.

SIR BENJAMIN BRODIE (1846)

Despite the great increase in our understanding of the pathology of leg ulcers, their management is still largely "art." Consequently there are numerous treatments, each with their enthusiastic advocates. There are, however, basic concepts which are helpful in management. Since about 95% of leg ulcers are of the "venous" or gravitational variety these will be considered first.

Pathology of venous ulcers



The skin—Ulcers arise because the skin dies from inadequate provision of nutrients and oxygen. This occurs as a consequence of (*a*) oedema in the subcutaneous tissues with poor lymphatic and capillary drainage; and (*b*) the extravascular accumulation of fibrinous material that has leaked from the blood vessels. The result is a rigid cuff around the capillaries, preventing diffusion through the wall, and fibrosis of the surrounding tissues.

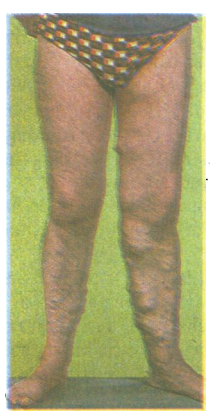
The blood vessels—Arterial perfusion of the leg is usually normal or increased, but stasis occurs in the venules. The lack of venous drainage is a consequence of incompetent valves between the superficial veins and the deeper large veins on which the calf muscle "pump" acts. In the normal leg there is a *superficial* low pressure venous system and *deep* high pressure veins. If the blood flow from superficial to deep veins is reversed then the pressure in the superficial veins may increase to a level that prevents venous drainage with "back pressure" causing stasis and oedema.

Incompetent valves leading to gravitational ulcers may be preceded by:

(*a*) deep vein thrombosis associated with pregnancy or, less commonly, leg injury, immobilisation, or infarction in the past;
(*b*) primary long saphenous vein insufficiency (rare);
(*c*) familial venous valve incompetence that presents at an earlier stage. There is a familial predisposition in half of all patients with leg ulcers; or
(*d*) deep venous obstruction.

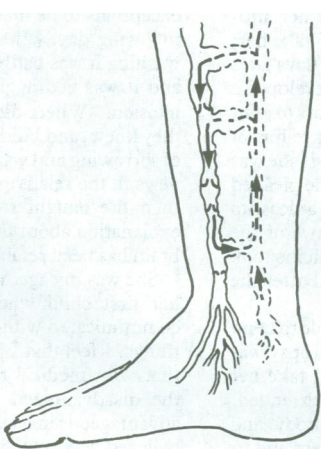
Who gets ulcers?

Mainly women get ulcers—2% of those over 80 having venous ulcers as a long term consequence of the factors listed above. Leg ulcers are more likely to occur and are more severe in obese people.

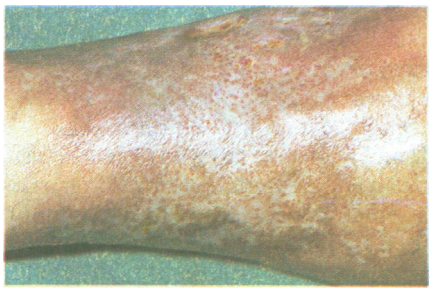


Varicose veins.

Ulcers and fibrosis.



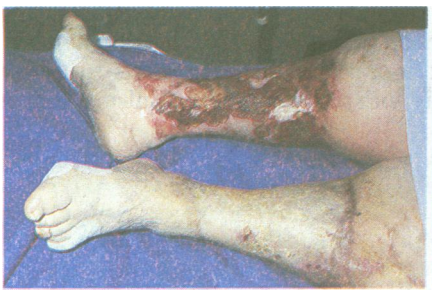
Clinical changes

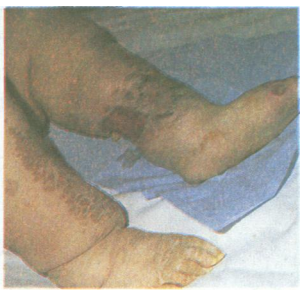


Atrophie blanche.

Oedema and fibrinous exudate often lead to fibrosis of the subcutaneous tissues, which may be associated with localised loss of pigment and dilated capillary loops, an appearance known as "atrophie blanche." This occurs around the ankle with oedema and dilated tortuous superficial veins proximally. This can lead to "champagne bottle legs," the bottle, of course, being inverted. Ulceration often occurs for the first time after a trivial injury.

Lymphoedema results from obliteration of the superficial lymphatics, with associated fibrosis. There is often hypertrophy of the overlying epidermis with a "polypoid" appearance, also known as liposclerosis.



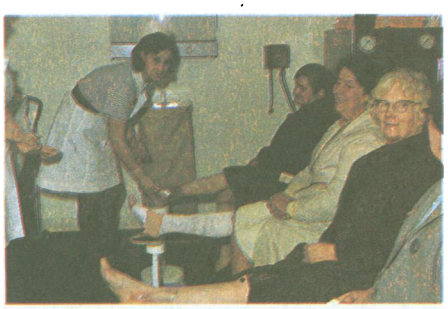


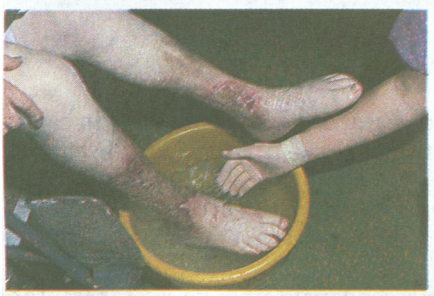
Champagne bottle legs.

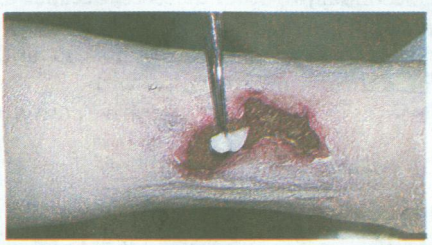
Venous ulcers occur around the ankles, commonly over the medial malleolus. The margin is usually well defined with a shelving edge, and a slough may be present. There may also be surrounding eczematous changes. Venous ulcers are not usually painful but arterial ulcers are.

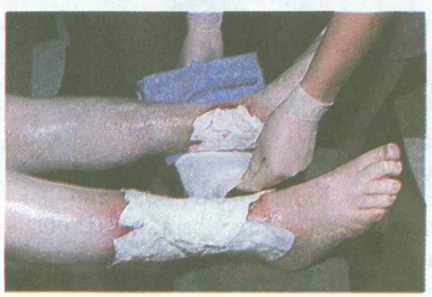
It is important to check the pulses in the leg and foot as compression bandaging of a leg with impaired blood flow can cause ischaemia and necrosis.

Treatment









When new epidermis can grow across an ulcer it will and the aim is to produce an environment in which this can take place. To this end several measures can be taken.

(1) Oedema may be reduced by means of (*a*) diuretics, (*b*) keeping the legs elevated when sitting, (*c*) avoiding standing as far as possible. Raising the heels slightly from time to time helps venous return by the "calf muscle pump," (*d*) applying compression bandages, which may do more harm than good *unless* they are applied *before* the patient gets out of bed in the morning, when there is minimal oedema, and applied with more pressure on the foot than the calf, so as to create a pressure gradient towards the thigh.

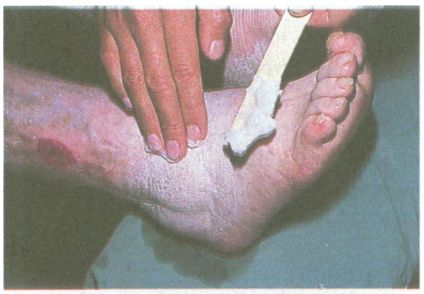
(2) Exudate and slough should be removed. Lotions can be used to clean the ulcer and as compresses—0·9% saline solution, sodium hypochlorite solution, Eusol, or 5% hydrogen peroxide.

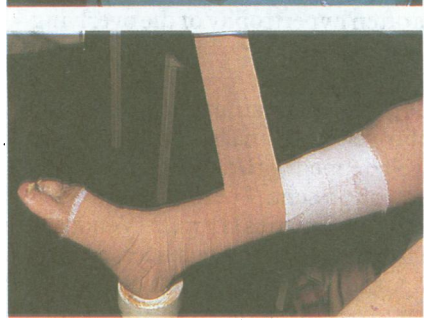
There is some evidence that antiseptic solutions and chlorinated solutions (such as sodium hypochlorite and Eusol) delay collagen production and cause inflammation. Enzyme preparations may help by "digesting" the slough. To prevent the formation of granulation tissue use silver nitrate 0·25% compresses, a silver nitrate "stick" for more exuberant tissue, and curettage, if necessary.

(3) The dressings applied to the ulcer can consist of (*a*) simple non-stick, paraffin gauze dressings. An allergy may develop to those with an antibiotic; (*b*) wet compresses with saline or silver nitrate solutions for exudative lesions; (*c*) silver nitrate cream (Flamazine) or hydrogen peroxide creams (Hioxyl); and (*d*) absorbent dressings, consisting of hydrocolloid patches or powder, which are helpful for smaller ulcers.

(4) Paste bandages, impregnated with zinc oxide and antiseptics or ichthammol, help to keep dressings in place and provide protection. They may, however, traumatise the skin, and allergic reactions to their constituents are not uncommon.

(5) Treatment of infection is less often necessary than is commonly supposed. All ulcers are colonised by bacteria to some extent, usually coincidental staphylococci. A purulent exudate is an indication for a broad spectrum antibiotic and a swab for bacteriology. Erythema, oedema, and tenderness around the ulcer suggest a β haemolytic streptococcal infection, which will require long term antibiotic treatment. Dyes can be painted on





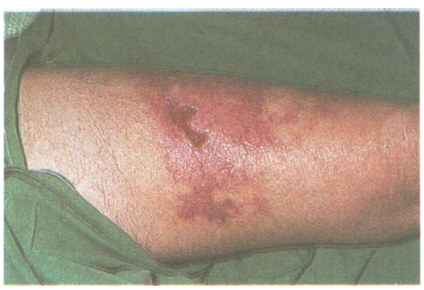
the edge of the ulcer, where they fix to the bacterial wall as well as the patient's skin. In Scotland bright red eosin is traditionally used, while in the south a blue dye, gentian violet, is favoured.

(6) Surrounding eczematous changes should be treated. Use topical steroids, not more than medium strength, avoiding the ulcer itself. Ichthammol 11% in 15% zinc oxide and white soft paraffin or Ichthopaste bandages can be used as a protective layer, and topical antibiotics can be used if necessary. It is important to remember that any of the commonly used topical preparations can cause an allergic reaction: neomycin, lanolin, formaldehyde, tars, Clinaform (the "C" of many proprietary steroids).

(7) Skin grafting can be very effective. There must be a healthy viable base for the graft, with an adequate blood supply; natural re-epithelialisation from the edges of the ulcer is a good indication that a graft will be supported. Pinch grafts or partial thickness grafts can be used. Any clinical infection, particularly with pseudomonal organisms, should be cleaned.

(8) Maintaining general health, with adequate nutrition and weight reduction, is important.

Arterial ulcers

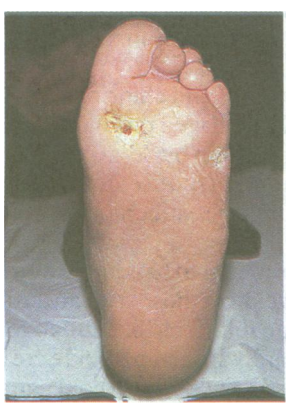


Ulcers on the leg also occur as a result of (*a*) atherosclerosis with poor peripheral circulation, particularly in older patients; (*b*) vasculitis affecting the larger subcutaneous arteries; and (*c*) arterial obstruction in macroglobulinaemia, cryoglobulinaemia, and polycythaemia "collagen" disease—particularly rheumatoid arthritis.

Arterial ulcers are sharply defined and accompanied by pain, which may be very severe, especially at night. The leg, especially the pretibial area, is affected rather than the ankle. In patients with hypertension a very tender ulcer can develop posteriorly (Martorelli's ulcer).

As mentioned above, compression bandaging will make arterial ulcers worse and may lead to ischaemia of the leg.

Diagnosis



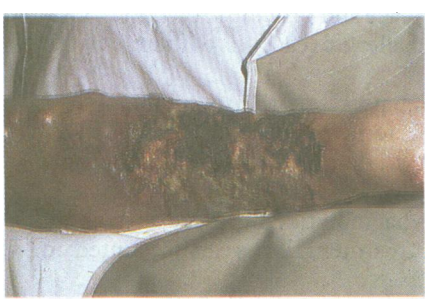
Ulcer in diabetic foot.

The differing presentation of arterial and venous ulcers helps in distinguishing between them, but some degree of arterial insufficiency often complicates venous ulcers.

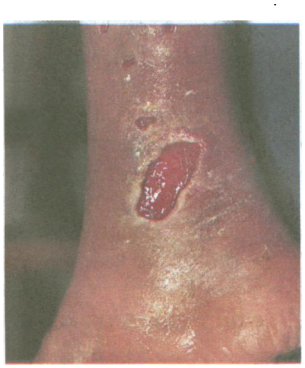
Phlebography and Doppler ultrasound may help in detecting venous incompetence and arterial obstruction, which can sometimes be treated surgically.

Ulcers on the leg may also occur secondary to other diseases, because of infection, in malignant disease, and after trauma.

Secondary ulcers—Ulcers occur in diabetes, in periarteritis nodosa, and in vasculitis. Pyoderma gangrenosum, a chronic necrotic ulcer with surrounding induration, may occur in association with ulcerative colitis or rheumatoid vasculitis.



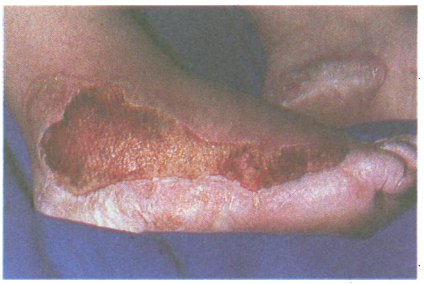
Squamous cell carcinoma in venous ulcer.



Tuberculous ulceration.

Infections that cause ulcers include staphylococcal or streptococcal infections, tuberculosis (which is rare in the United Kingdom but may be seen in recent immigrants), and anthrax.

Malignant diseases—Squamous cell carcinoma may present as an ulcer or, rarely, develop in a pre-existing ulcer. Basal cell carcinoma and melanoma may develop into ulcers, and Kaposi's sarcoma may present as an ulcer.



Trauma—Patients with diabetic or other types of neuropathy are at risk of developing trophic ulcers. Rarely they may be self induced—"dermatitis artefacta."

Dr P K Buxton, FRCPED, FRCPC, is consultant dermatologist, Royal Infirmary, Edinburgh, and Fife Health Board.

In treating venous leg ulcers
(1) Take measures to eliminate oedema and reduce weight—make sure the patient understands these.
(2) Never apply steroid preparations to the ulcer itself or it will not heal. Make sure that both nurses and patients are aware of this.
(3) Beware of allergy developing to topical agents—especially to antibiotics.
(4) There is no need to submit the patient to a variety of antibiotics according to the differing bacteria isolated from leg ulcers, unless there is definite evidence of infection clinically.
(5) A vascular "flare" round the ankle and heel with varicose veins, sclerosis, or oedema indicates a high risk of ulceration developing.
(6) Make sure arterial pulses are present.

Epidemiology

Report from the PHLS Communicable Disease Surveillance Centre

A case of botulism was reported in August and another of infant botulism two months earlier; an outbreak of haemorrhagic colitis took place in the West Midlands in July and August; and two zoonoses received publicity during September, particularly in the veterinary press—rabies in bats and liver fluke disease

Botulism

A case of botulism, widely reported in the press, occurred in a man aged 49 who developed vomiting about 10 hours after eating a prepacked shelfstable kosher meal on a flight from Nice to Heathrow on 30 August. Later he developed diplopia, dysphagia, dysarthria, and increasing generalised paralysis and required artificial ventilation. The clinical diagnosis was made promptly and treatment with antitoxin given. A high titre of type A *Clostridium botulinum* toxin was detected in his serum and in gastric aspirate, which also contained the organism.

The implicated meal included a rice and vegetable dish which was said to have smelt offensive; the victim consumed less than a teaspoonful before discarding it. Other members of his family immediately rejected the dish after opening the sealed containers. All the meal was discarded and none was available for examination. These meals were withdrawn by all airlines and no further cases were reported. Unfortunately it was not possible to identify any rice and vegetable dishes from the same production batch for examination, but three of these dishes which were obtained were not sterile, being contaminated with *Bacillus* spp. Neither *C botulinum* nor botulinum toxin were detected.

Botulism is rare in the United Kingdom. Since the famous Loch Maree outbreak in Scotland in 1922, when eight people died after eating sandwiches containing duck paste, only 25 cases with 14 deaths have been reported in eight incidents. The most recent outbreak took place in Birmingham in 1978. Four people were affected, two of whom died, after eating canned salmon from a tin which was later shown to be defective. Most of these incidents were due to *C botulinum* toxin type A, but some were also due to types B and E.

Botulism has been reported more often in North America, where it is often associated with defective home preserved vegetables, in continental Europe, where it is usually associated with cured meats or vegetables, and in Japan, where it is usually associated with raw fermented and smoked fish.

Botulism should be suspected when neurological symptoms, characteristically visual disturbances, difficulty in swallowing and speaking, and flaccid paralysis occur in a mentally alert patient in association with gastrointestinal symptoms or with epidemiological evidence suggesting a contaminated food. The incubation period is usually less than 36 hours, though it depends on the dose of toxin consumed and it may be as long as eight days. Laboratory confirmation is by detecting toxin in the patient's serum (20-50 ml blood should be obtained before the administration of antitoxin), faeces, or vomitus and by the subsequent isolation of the organism from the suspected food or its container.

Advice and reference laboratory facilities are available at the PHLS food hygiene laboratory, Colindale (01 200 4400), and the public health laboratory, Luton (0582 571898). Supplies of trivalent equine botulinum antitoxins (types A, B, and E) for therapeutic purposes are available through designated centres listed in the *Health Service Purchasing Guide* (section D), the *Chemist and Druggist Annual Directory*, the *PHLS Directory*, and the *Pharmaceutical Supplies Bulletin* 1985;8:1-10. (See Communicable Disease Report of 14 February 1986 (86/07).) Epidemiological advice is available from the PHLS Communicable Disease Surveillance Centre (CDSC) (01 200 6868), from local public health laboratories