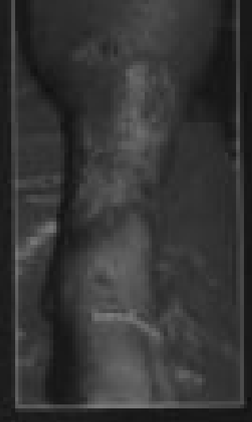
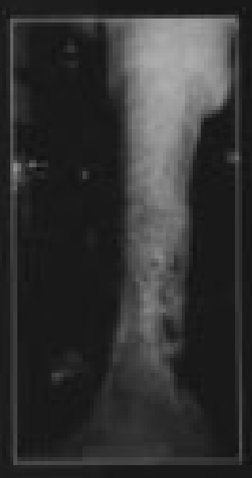




Guidelines for the Management of Leg Ulcers in Ireland

This book is published by

Smith+Nephew
First Choice in Wound Management

GUIDELINES FOR THE MANAGEMENT OF LEG ULCERS IN IRELAND

Edited by

Pierce Grace,
Professor of Surgical Science,
University of Limerick,
Ireland.

Published by Smith & Nephew Ltd.

ISBN 0 - 9542474 - 0 - X

List of Contents & Contributors

Foreword

Venous ulceration has long been regarded as a Cinderella condition. This attitude has changed in recent times with the demonstration that control of venous hypertension using sophisticated compression systems, brings about healing in the majority of patients without requirement for expensive in-hospital facilities. The challenges now are to achieve healing in resistant groups and to accelerate healing in others. Exciting new information is beginning to emerge from studies of local wound and systemic host molecular changes. It is clear that analysis of the sequence of these molecular events is critical to the development of cytokine strategies capable of altering wound healing. It is probable that many ulcers in the future will be treated by a combination of local mechanical approaches and local systemic molecular strategies.

However, it is important to build on the mutli-discipline approach to assessment and the combined hospital-community management, which has acheived so much. These guidelines provide a clear framework for the assessment and management of patients with leg ulcers and will be very useful to health workers whose job is to treat people with chronic leg ulcers. It is particularly gratifying that Irish professionals who are familiar with the Irish health system have developed these guidelines. I believe that they will be an invaluable resource in the management of leg ulcers in Ireland and a useful reference for anyone with an interest in this topic.

David Bouchier-Hayes, MCh, FRCS, FRCSI, FACS, FRCP&S Glas.
Professor of Surgery and Chairman
Department of Surgery
Royal College of Surgeons in Ireland

Preface

Leg ulcers are a common and, frequently, chronic problem seen in the health care system in Ireland. They occur in approximately 0.12% of the population at any given time and cost about €6.5m per annum to treat. Various professionals encounter patients with leg ulcers but responsibility for the day-to-day management of these patients falls largely on nurses, including public health nurses (probably the majority), practice nurses or nurses working in a hospital setting. This booklet aims to provide practical guidelines on the management of leg ulcers in an Irish setting and is evidence based to reflect best international practice.

The booklet was written by professionals working in Ireland who are familiar with the Irish health care system and the issues that arise in relation to the management of leg ulcers in this country. In the first section, I have attempted to provide a synopsis of the pathophysiology of leg ulceration. Fionnuala O'Brien and Paul Burke provide an overview of the size of the problem of leg ulcers in one health board area based on epidemiological research that they carried out in the Mid-Western Health Board area in conjunction with Prof Ivan Perry of The Department of Epidemiology in UCC. Mary Paula Colgan, from her extensive experience at the Veins Unit of St James's Hospital Dublin, provides clear guidelines on the clinical assessment of patients with leg ulcers. In particular she describes the methodology for performing and interpreting Doppler assessment of leg ulcers. Pauline Diamond provides a detailed description of the technique of dressing and bandaging leg ulcers and provides an overview of compression therapy and its indications and contraindications. Dermot Hehir discusses the management of infected leg ulcers and the thorny issue of when to prescribe antibiotics for patients with leg ulceration. Dorothy O'Sullivan and Frank Powell provide excellent advice on the dermatological problems that frequently complicate leg ulceration and may make their management difficult. Liam Joyce discusses the management of the difficult to heal chronic venous leg ulcer.

Smith and Nephew, who have been providing wound care products in Ireland for many years, are the sponsors of this booklet. Over the years they have been generous in their financial support for research into leg ulcers in Ireland as well as providing educational and training fora for professionals managing this difficult problem. The idea for this booklet came from Sandra O'Shaughnessy, Clinical Support and Education Manager with Smith&Nephew. She responded to a need, expressed by carers, for clinical guidelines to inform practice in the management of leg ulcers in the Irish setting. I congratulate the authors on their excellent contributions and I hope that the professionals who look after patients with leg ulceration will find this booklet a useful evidence-based resource for informing their practice.

Pierce Grace
Professor of Surgical Science
University of Limerick
Jan 2002

SECTION 1

Pathophysiology of Leg Ulcers

Pierce A. Grace

Leg ulcers are common and are due to a wide variety of pathologies. How a limb becomes ulcerated depends on the underlying pathophysiology and this chapter attempts to explain this for the common leg ulcers seen in Ireland.

Definitions: An ulcer is defined as an area of discontinuity of the surface epithelium. A leg ulcer is a discontinuity of the squamous epithelium of the skin, usually around the ankle or on the foot. A chronic leg ulcer is more difficult to define but many people consider ulceration of more than 4 to 6 six weeks duration as being chronic (1).

There are numerous causes of leg ulceration ranging from the common e.g. venous ulcers, to the exotic e.g. Buruli ulcers seen in Uganda and Zaire. The common ulcers encountered in Ireland are listed in Table 1.

Table 1. Common causes of leg ulcers in Ireland

TYPE	CAUSE
Venous	Post DVT
	Varicose veins
Arterial	Peripheral vascular disease
Diabetes Mellitus	Ischaemic
	Neuropathic
Malignancy	Squamous carcinoma
	Basal cell carcinoma
	Malignant melanoma
Miscellaneous	Vasculitis
	Infections
	Pyoderma gangrenosum

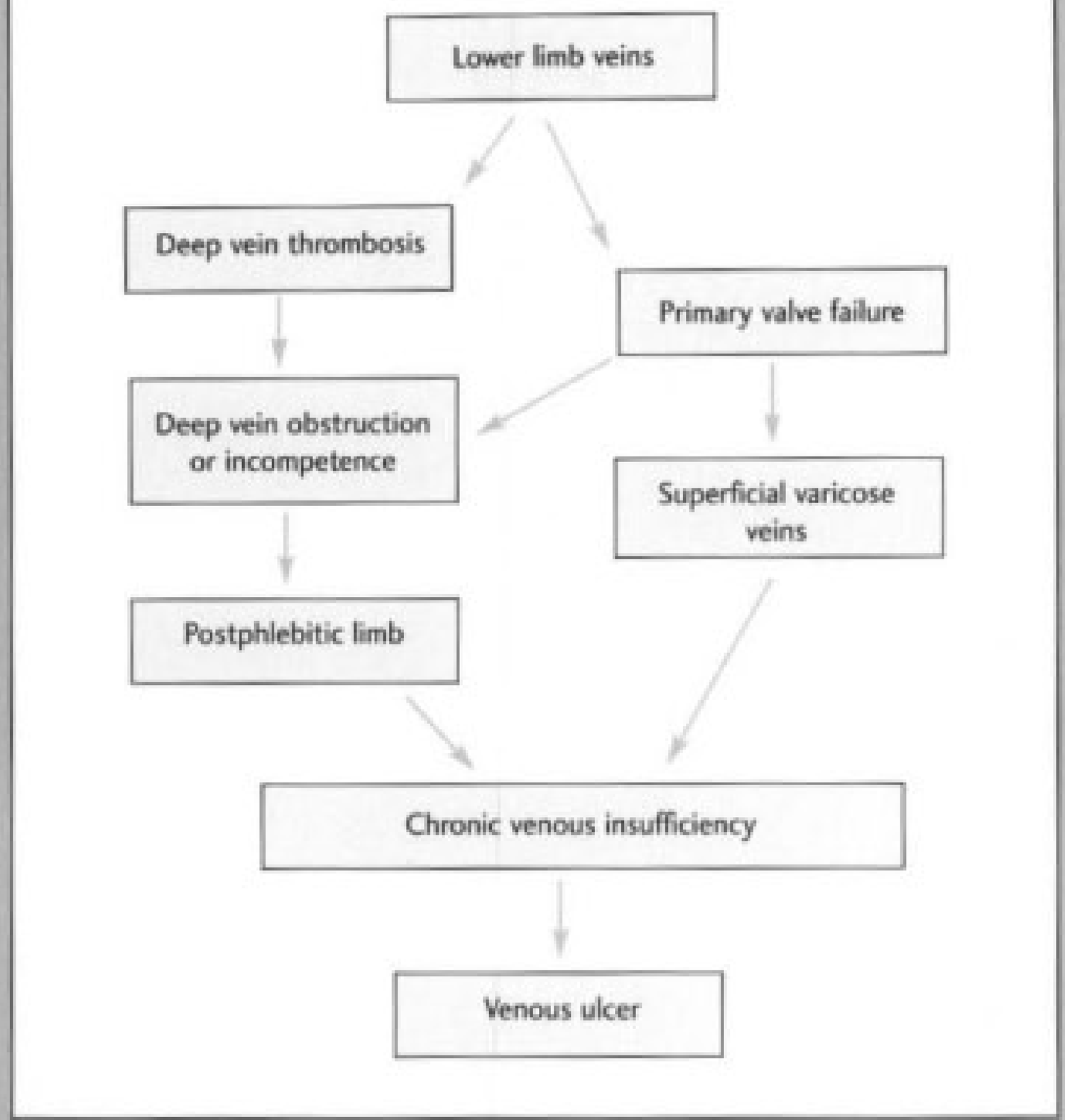
DVT = deep venous thrombosis

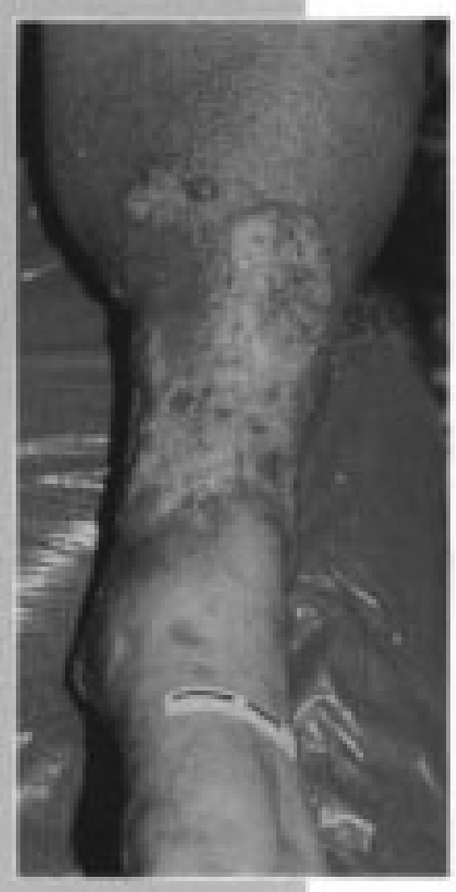
Venous ulcers

Venous ulcers arise because of venous hypertension. Normally blood is drained from the lower limb by deep and superficial veins. The blood flows from the foot to the groin and from superficial to deep veins in a unidirectional manner. Reverse flow is prevented by the action of valves in the veins. The venous pressure at the ankle while standing is 125cm H_2O but on walking the action of the calf muscles surrounding the veins pushes blood out of the leg and reduces the pressure to about 40 cm H_20. Incompetence of the valves in either the superficial or deep venous system results in an increase in pressure in the veins at the level of the ankle which in turn leads to swelling of the tissue, sequestration of red blood cells, iron deposition, lipodermatosclerosis and ulceration.

Superficial valve incompetence is seen in patients with varicose veins. A thrombus developing in the deep veins initially causes obstruction to flow. As the thrombus is lysed and removed by the healing process, the valves in the deep veins may be destroyed resulting over time in chronic venous insufficiency, poor venous return, venous hypertension and ulceration. This sequel to venous thrombosis is called the postphlebitic or post thrombotic limb (Fig 1).

Figure 1 Algorithm for the development of a venous ulcer.





There are two theories as to why the tissues ulcerate in the presence of venous hypertension. The fibrin cuff theory postulates that the rise in venous pressure causes a loss in plasma proteins through the capillary walls in the tissues. These proteins, especially fibrinogen, form a cuff around the capillaries, which can be seen using histochemical methods. These cuffs may then interfere with tissue oxygenation although this has never been proven (2). A second theory suggests that trapping of white cells in the tissues with subsequent release of cytokines and proteases may be the underlying cause of ulceration (3). Whatever the exact mechanism the result is tissue damage and ulceration.

Arterial Ulcers

Arterial ulcers indicate the presence of severe peripheral occlusive arterial disease (POAD). The function of the arterial circulation is the delivery of oxygen and nutrients to the tissues in the body. Inadequate blood supply to tissue is called ischaemia and, if this is severe, the tissue may actually die (necrosis) and develop gangrene (i.e. digestion of the tissues by saprophytic organisms). In patients with POAD ischaemia of the foot leads to death of skin and ulceration over the pressure areas (heel, heads of first and fifth metatarsals) and the toes. POAD is caused mostly by atherosclerosis (thrombosis or [micro]-embolism) of the peripheral arteries (don't smoke!) but it is also seen in patients with vascular trauma, diabetes mellitus, vasospasm (Raynaud's phenomenon) or inflammation (Buerger's disease) of the arteries (4).

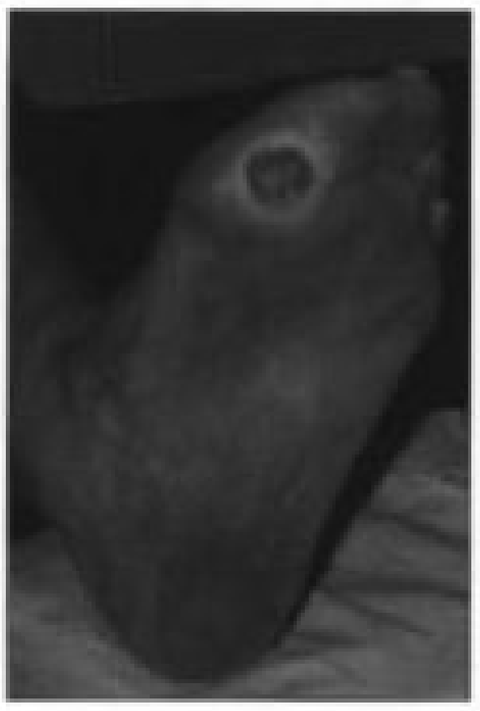
Diabetic Ulcers

Patients with diabetes have either Type1 Diabetes (reduced insulin secretion because of reduced numbers of ß-cells in the Islets of Langerhans in the pancreas) or Type 2 Diabetes (normal insulin output but few insulin receptors on the cells in the body tissues that utilize insulin). The result in either type is an accumulation of glucose in the tissues (hyperglycaemia).

A number of biochemical mechanisms appear to be involved in developing complications in diabetes, including leg ulceration.

- Firstly, in the presence of hyperglycaemia, glucose in the tissues binds to proteins, thus affecting the shape and function of those proteins. Abnormal protein function leads to cell damage. The amount of protein binding present can be determined by measuring the amount of haemoglobin that is bound to glucose, gylocosylated haemoglobin.
- Secondly, in some tissues (e.g. aorta, peripheral nerves) high glucose levels result in the accumulation of sorbitol (an alcohol) and fructose (a sugar) in the cells which in turn causes water to be brought into the cells by osmosis; the cells swell and are damaged.
- Lastly, because of the high levels of sorbitol in the cells a substance called myoinositol cannot enter the cell and the lack of myoinositol may also have a role in developing complications in diabetes.

Diabetic ulcers are of two types: Ischaemic and Neuropathic. Ischaemic ulcers are due to atherosclerosis just as in the non-diabetic. But atherosclerosis is much more pronounced in diabetes because of hypertension (50% of diabetics have high blood pressure) and high cholesterol and triglyceride levels in the blood. Diabetics also develop microvascular disease affecting the arterioles and capillaries thus reducing blood flow to the tissues and making them prone to ulceration. Established ulcers or wounds are also very difficult to heal. Neuropathic ulcers develop because the patient has a peripheral nerve dysfunction and is unaware of the trauma or irritation to the foot that initiates ulceration. Bacterial infection of the surrounding tissues via the ulcer produces cellulitis and extensive tissue destruction. Diabetics have impaired mechanisms for dealing with infection and the glucose in the tissues is an excellent medium for bacteria (5).



Malignant Ulcers

The malignant ulcers that one might encounter on a leg are squamous cell carcinoma, basal cell carcinoma and malignant melanoma. All three are predisposed to by exposure to sunlight, especially ultraviolet light.

- Squamous cell carcinoma arises from keratinocytes in the epidermis. It appears as an ulcer with heaped up everted edges; it grows rapidly, is invasive and metastasizes. Solar keratoses and Bowen's disease are precursors of squamous carcinomas of the skin. Chronic irritation of a long standing venous ulcer can induce malignant change, Marjolin's ulcer.

- Basal cell carcinoma (Rodent Ulcer) is rarely seen on the leg. The tumour arises from basal cells in the epidermis. They present as small nodules that ulcerate producing a pearly coloured rounded edge. They are locally destructive (hence the name "rodent ulcer") but do not metastasize.

- Malignant melanoma sometimes presents as an ulcer on the leg. It is a tumour that arises from the melanocytes in the skin. They grow radially and vertically and spread in the lymphatic system to the regional lymph nodes and in the blood to the brain and the liver. Malignant melanomas usually present as pigmented lesions in the skin. Ulceration indicates a poorer prognosis (6).

Miscellaneous

Leg ulcers rarely may arise from a number of unusual conditions. Table 2

Table 2 Unusual causes of leg ulceration.

Cause	*Explanation*
Trauma	Minor trauma may result in a leg ulcer in patients with poor circulation, malnutrition or on steroids
Vasculitis	Leg ulcers are often seen (and difficult to treat) in patients with Rheumatoid arthritis, scleroderma and Buerger's disease
Infections	Tuberculosis, syphilis, tropical diseases
Pyoderma gangrenosum	A destructive necrotizing non-infective ulcer that starts usually as a nodule on the skin. Seen with ulcerative colitis and Crohn's disease

References

1. Callam MJ. Leg ulcer and chronic venous insufficiency in the community. In: Ruckley CV, Fowkes FGR, Bradbury (eds), Venous Disease, Epidemiology, Management and delivery of care. Springer- Verlag, London. 1999; pp15-26.

2. Michael CC. Oxygen diffusion in oedematous tissue and through pericapillary cuffs. Phlebology 1990;5:223-230.

3. Scott HJ, McMullin GM, Coleridge Smith PD, Scurr JH. Venous ulceration and the role of the white blood cell. J Med Sci Tech 1990;14:184-7.

4. Cushieri A, Hennessy TPJ, Greenhalgh RM, Rowley DI, Grace PA. Clinical Surgery, Blackwell Science, Oxford 1996, pp277-341.

5. Craighead JE. Diabetes. In Rubin E, Farber JL (eds) Pathology. JB Lippincott Company, Philadelphia 1988, pp1166-1177.

6. Cushieri A, Hennessy TPJ, Greenhalgh RM, Rowley DI, Grace PA. Clinical Surgery, Blackwell Science, Oxford 1996, pp266-273

SECTION 2

Prevalence and aetiology of *leg ulcers in Ireland*

Fionnuala O' Brien
Paul Burke

Introduction

People are living longer and chronic diseases such as leg ulceration are an increasing source of morbidity and cost in the community (1,2,3). In Ireland, we are liable to see a rise in the numbers presenting with leg ulcers as the population over 65 years of age grows by a fifth over the next ten years (4). Adequate service planning will be essential to pre-empt the increased health care demands of those affected with leg ulcers.

Effective planning requires information on the number of people in the population receiving treatment for leg ulcers, as well as information on the underlying aetiology of ulceration. The most appropriate method of gathering this information is by way of a prevalence survey (1,5,6).

The importance of prevalence surveys

Well conducted prevalence surveys assess the impact of specific diseases on the population and are always important (7). Medical care needs are identified, providing information to inform resource allocation and care.

Prevalence

- quantifies the number of people with a specific disease or lifestyle in a defined population during a given time
- assesses the health status and healthcare needs of a population;
- highlights gaps and inequalities in care;
- informs resource allocation;
- informs change;
- forms the basis of clinical audit.

The prevalence of leg ulcers in Ireland

We undertook a prevalence survey of leg ulcers in the Mid-Western Health Board (MWHB) region of Ireland over a two-month period in 1998 to address the lack of information available on the size of the problem of leg ulcers in Ireland (8). A leg ulcer was defined as an open sore below the knee and included foot ulcers. We also took the opportunity at that time to look at the main causes of ulceration. The MWHB has a large urban/rural population of around 300,000 with a similar age-sex distribution to the national population. We could therefore make general inferences from our findings for the Irish population.

Three hundred and eighty nine patients were identified with leg ulcers resulting in a prevalence rate of 12/10,000. In other words, we found that 12 people out of every 10,000 suffer from active leg ulcers at any given time. We also found that leg ulcers were most evident in the elderly; the average age being 72 years. When we looked at the older age groups in particular, we noted that the prevalence of leg ulcers rose dramatically to 103 in every 10,000 aged 70 years and over.

The ratio of men to women in our survey was 1:2 respectively. While the age specific prevalence rates for men and women under 60 years of age were relatively similar, leg ulcers were a much greater problem for women in the older age groups (figure 1).

Prevalence of leg ulcers in Ireland

- 12 people out of every 10,000 suffer from leg ulcers at any given point in time.
- Prevalence increases eight fold in the population over 70 years.
- The average age of patients with leg ulcers is 72 years.
- Women are twice as likely to be affected as men.

Importance of assessment

An understanding of the origins of the patient's ulcer is a pre-requisite to the appropriate management of that patient. Multifactorial causes complicate the classification of some leg ulcers. However, a working diagnosis is necessary to prioritise the mode of treatment. For the purpose of this survey, we considered the main cause of ulceration adequately investigated if the patient's assessment had included the measurement of an anklebrachial pressure index (A.B.P.I.). (9,10)

Level of assessment undertaken in the MWHB

Only half of the patients with leg ulcers in the MWHB had been properly assessed at the time of the survey. The majority of these assessments had been carried out at one of the two specialist leg ulcer clinics in the region. There was minimal use of doppler ultrasound by community healthcare workers. Just seven percent of patients were assessed locally in the community. Overall, the lack of assessment was most evident in areas furthest from specialist leg ulcer clinics.

Long Term Ulceration

We also found a large number of patients with open leg ulcers longer than expected, or who had recurring problems with ulceration. The average duration of patients' ulcers was eight months. Up to a quarter had persistent ulcers for at least two years and a notable number of these patients did not have the cause of their ulcers determined.

Leg ulcer assessment

- is a prerequisite to effective leg ulcer management;
- minimises the improper use of treatments;
- reduces the risk of long term ulceration;
- facilitates the early detection of potential limb threatening aetiologies.

Aetiology of leg ulcers

The reported ulcer type of patients who had undergone an assessment prior to the survey were pooled with the findings of our follow up examinations of patients with leg ulcers of unknown or uncertain cause. In all, the main cause of ulceration was documented for 90.5% of all patients identified with leg ulcers in the MWHB. The majority of patients' limbs were found to have venous leg ulcers while a degree of arterial disease was present in 17% of limbs.

Diabetes, a significant risk factor for leg ulcers, was more prevalent in patients with an arterial component to their ulcer (27%) than in patients with venous ulcers (5%). However, diabetic neuropathy was seldom the single causative factor of ulceration. Likewise, while 18% of patients were reported to have rheumatoid arthritis, few were determined to have ulcers solely caused by vasculitis.

Priority was given to the presence of arterial disease in our classification of leg ulcers, but the contribution of diabetes and rheumatoid arthritis to the multifactorial aetiology of leg ulcers should not be underestimated.

Aetiology of leg ulcers in Ireland

Venous	81%
Arterial	10%
Mixed Arterial / Venous	7%
Diabetic neuropathy	1%
Malignancy	1%
Rheumatoid	0.5%

Conclusion of study's findings

Leg ulcers are common in the elderly in Ireland and are usually venous in origin. Long-term ulceration remains a problem for many despite improvements in our understanding of the causative factors and the treatment of leg ulcers.

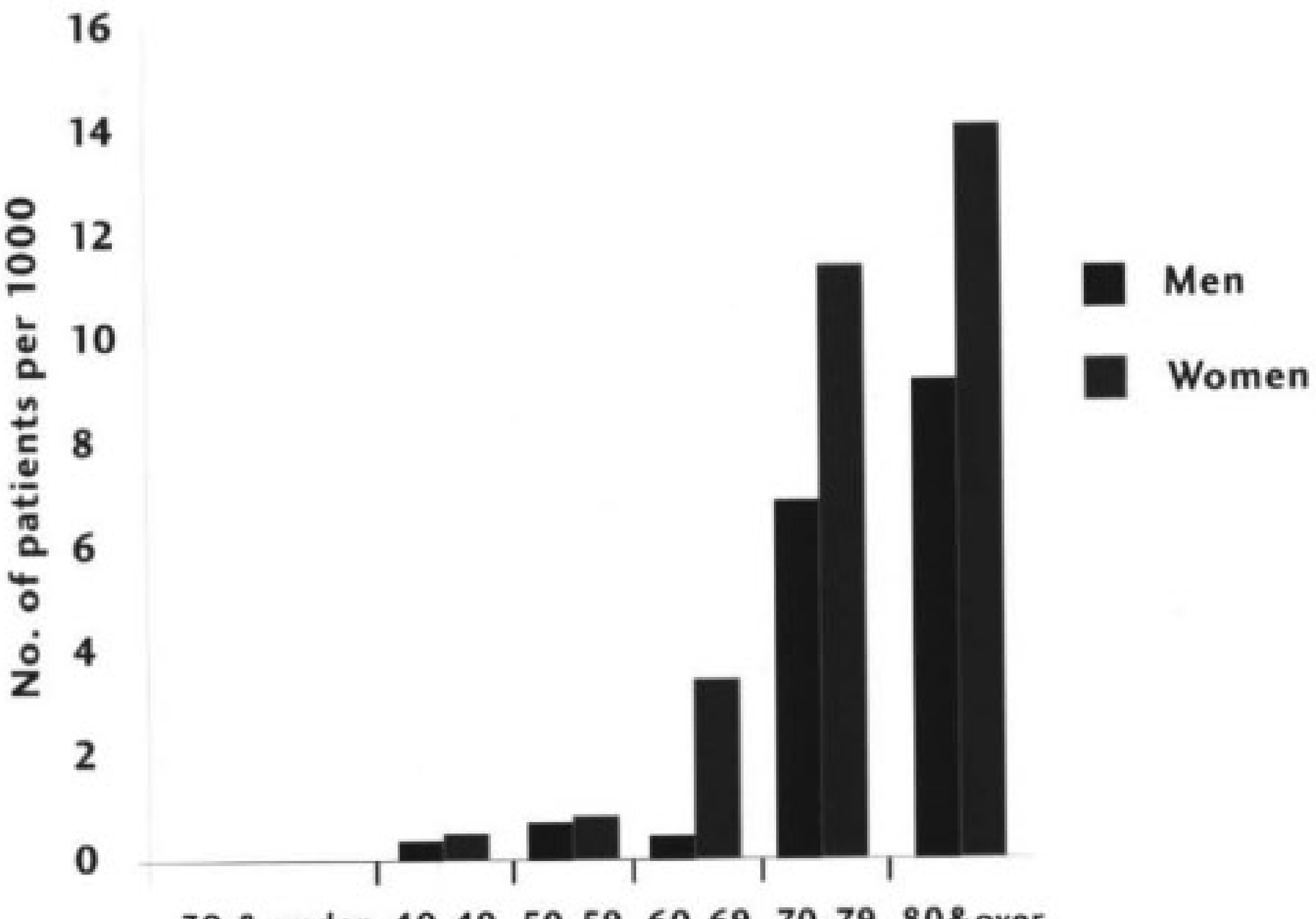
Recommendations for practice

1. A standardised and integrated hospital and community based leg ulcer service to facilitate early assessment and use of treatments with proven effectiveness.
2. Staff training to improve leg ulcer management skills in the community.
3. The use of compression bandaging for venous leg ulcers.
4. Corrective surgery for venous and arterial disease of the lower limbs.
5. Renewed emphasis on the hard to heal leg ulcer.
6. Clinical audit to monitor practice and prevalence rates.
7. Randomised controlled trials to evaluate primary and secondary preventive measures for venous leg ulcers.

Recommendations

- Standardised integrated policy for leg ulcer care.
- Increased expertise in community assessment and treatment of leg ulcers.
- The use of proven clinically effective treatments.
- Surgical option to correct underlying venous or arterial disease.
- Ongoing audit to monitor practice.
- Valid studies to evaluate primary and secondary preventive interventions.

Figure 1. Age specific prevalence of leg ulcers in men and women in the MWHB



Acknowledgement.
We would like to thank Prof Ivan Perry of the Department of Epidemiology at UCC for his invaluable help with this project.

References

1. Callam MJ, Ruckley CV, Harper DR, Dale JJ. Chronic ulceration of the leg: extent of the problem and provision of care. British Medical Journal 1985;290:1855-6.
2. Freak L, Simon D, Kinsella A, Mc Collum C, Walsh J et al. Leg ulcer care: an audit of cost effectiveness. Health Trends 1995;27:133-6.
3. Faresjö T, Frödin T, Vahlquist C, Klevbrand M, Elfström et al. Cost of the treatment of leg ulcers: initiating a quality assurance process. International Journal of health Care Quality Assurance 1997;10:125-30.
4. Central Statistics Office. Population and labour force projections. Dublin: Central Statistics Office, 1999.
5. Cornwall JV, Dore CJ, Lewis JD. Leg ulcers: epidemiology and aetiology. British Journal of Surgery 1986;73:693-6.
6. Nelzen O, Bergqvist D, Lundhagen A, Hallbook T. Chronic Leg ulcers: an underestimated problem in primary health care among elderly patients. Journal of Epidemiology and Community Health 1991;45:184-7.
7. Hennekens CH, Buring JE. Measures of disease frequency and association. In: Mayrent SL, editors. Epidemiology in medicine. Boston: Little, Brown and Company.1987;54-98.
8. OBrien JF, Grace PA, Perry IJ, Burke PE. Prevalence and aetiology of leg ulcers in Ireland. Irish Journal of Medical Science 2000;169:110-2.
9. Moffatt C, Harper P. Leg ulcers. UK: Churchill Livingstone, 1997.
10. Callam MJ. Leg ulcers and chronic venous insufficiency in the community. In: Ruckley CV, Fowkes FGR, Bradbury AW, editors. Management and delivery of care. London: Springer, 1999:15-25.

SECTION 3

Assessment of Patients with leg Ulcers.

Mary-Paula Colgan

In an ideal world with no limit on health resources all patients presenting with leg ulceration would be fully assessed clinically, non-invasively and perhaps invasively. However resources are scarce and choices must be made. The prerequisite to assessing patients with ulceration is a firm knowledge of the aetiology and pathophysiology of lower extremity ulceration (see section 1). A comprehensive assessment allows one to determine:

- The aetiology of the ulcer
- Local and general factors that may cause a delay in healing
- Social circumstances and optimum setting for care.

There are several reports highlighting the importance of appropriate clinical assessment to ensure effective treatment (1,2). The assessment of patients with ulceration falls into four categories:

- Medical history
- Physical examination
- Non-invasive investigations (including ABPI)
- Invasive investigations

This chapter will deal with history and physical examination in addition to ABPI measurements. A discussion of available non-invasive and invasive investigations is beyond its scope.

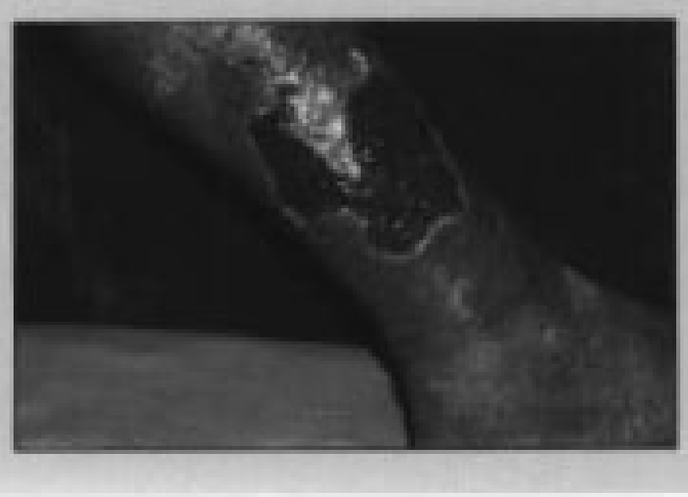
Medical History

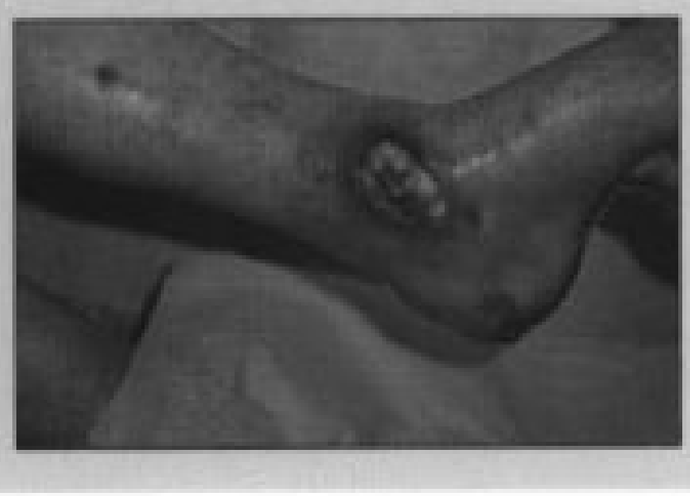
A full medical history is essential and specific questioning to uncover venous and/or arterial disease is required (Table I). Though a positive venous or arterial history signifies the presence of disease it does not mean that the ulcer aetiology is one and the same. The presence of associated diseases such as rheumatoid arthritis, SLE and diabetes should be noted. Patients should also be questioned regarding medications, as some, e.g. steroids or cytotoxics will slow wound healing.

Allergies must be accurately documented. Many patients will report allergies to dressings however on questioning it becomes clear that it was pain/discomfort that was experienced rather than an allergic reaction. This information allows one to reconsider important dressings in the present clinical setting.

Table I. Venous and arterial history

Venous	Arterial
Varicose veins History of phlebitis History of deep vein thrombosis History of trauma/fracture	Intermittent claudication Rest pain Diabetes Smoking Cardiac disease/hypertension





Cardiac disease/hypertension

It is usually possible with careful history taking to distinguish arterial pain from other causes of leg pain such as arthritis or infection. Claudication comes from the Latin claudicare, which means, "to limp". Patients classically describe a feeling of tightness affecting their calf or buttocks that is brought on by exercise and relieved by a few moments rest. The pain will occur more rapidly if the patient is going uphill or walking at a more rapid rate. Rest pain is usually described as a "burning"pain affecting the toes and feet, which in its early stages occurs when the patient lies down. Patients will usually volunteer that they get relief by dangling their legs over the edge of the bed. As it progresses it is present constantly and patients will often sit out in a chair at night to get some relief.

Intermittent claudication signifies mild/moderate arterial disease while rest pain signifies critical ischaemia. One must remember that patients with lower extremity ulceration are often elderly and immobile and they may not exercise enough to experience claudication pain in spite of the presence of arterial disease.

Peripheral neuropathy is the commonest cause of ulceration in the diabetic patient however; this may be complicated by the additional presence of arterial disease. Patients should be questioned in particular about foot deformity and altered sensation.

Physical examination

It is important that patients are examined both lying and standing and particular attention should be paid to their gait. Reduced joint movement particularly of the ankle is associated with poorer healing rates. General signs of venous and arterial disease are noted in Table 2.

Table 2 Signs of venous and arterial disease

Venous	Arterial
Varicose veins	Pallor
Ankle flare	Hair loss
Pigmentation and eczema	Absent pulses
Atrophe blanche	Dependent rubror
Leg swelling	Arterial bruits

Ulcer Examination

The site, size and appearance of the ulcer are crucially important. Venous ulcers are usually shallow and located on the gaiter area of the leg. Arterial ulcers may have a "punched-out" appearance and occur often on the toes, feet or heel. Neuropathic ulcers are classically over bony prominences on the foot such as the metatarsal heads. Often there is a build up of surrounding callus. Vasculitic ulcers are less easy to classify but one distinguishing feature is severe pain. Malignancy is a rare cause of lower extremity ulceration. An unusual site or appearance such as rolled edges may suggest the possibility of malignancy and then punch biopsy must be performed.

The condition of the ulcer and surrounding skin should also be recorded as this will be important in deciding on best treatment, e.g if there is surrounding eczema then a topical steroid may be required or macerated skin may require additional protection (see section 6).

A variety of methods exist to measure wounds with most focusing on area rather than depth (3,4). As a general rule sophisticated measuring devices are not required in everyday practice. Serial tracings provide a reliable and cheap guide to ulcer progress. Ulcer size should always be recorded at the initial visit. Monthly recording is probably sufficient thereafter, unless there is a deterioration during treatment. It is important to remember that size alone does not give the entire picture and comment should also be made about the ulcer depth, edges and base and if possible whether there is significant exudate or not. These parameters will all aid in choosing the best dressing. In summary the ulcer parameters required are:

- Ulcer site
- Ulcer size
- Ulcer depth
- Ulcer edge
- Ulcer base
- Exudate
- Surrounding skin

General Assessment

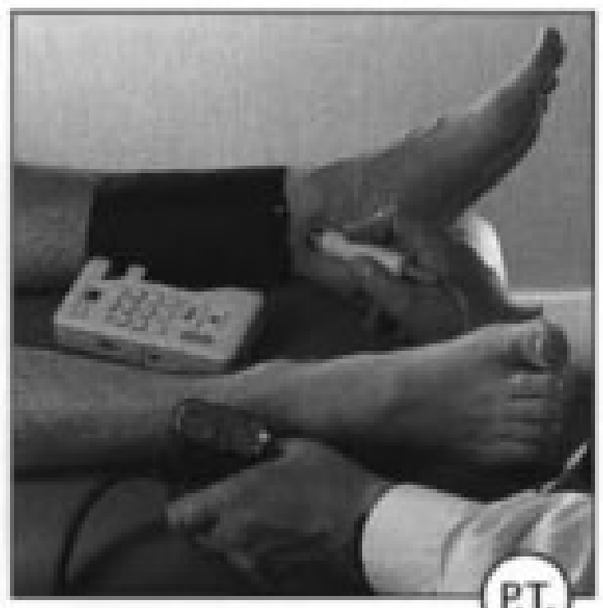
Blood pressure, weight and urinalysis should be recorded on all patients at first presentation. A general nutritional assessment is also useful. Several guides are available and discussion with the local support dietician is invaluable and allows appropriate referral for nutrition advice. Baseline haematology and biochemistry are also useful.

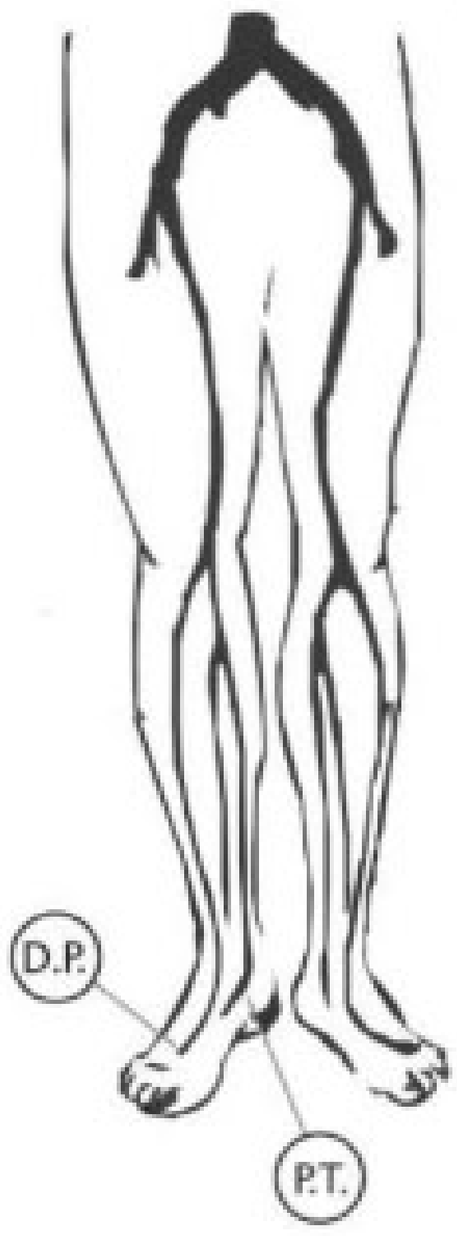
Ankle Brachial Pressure Index Measurements (ABPI).

Arterial pressure measurements are an essential component of ulcer assessment. They identify and quantify the presence and severity of arterial disease. They should be performed by personnel trained and experienced in the area (5,6,7). Several simple steps should be followed to obtain accurate recordings:

- Patients should be supine and resting for 10-15 mins. in a warm room.
- The procedure should be explained to the patient.
- A cuff is placed around the upper arm and the brachial or radial artery insonated using a Doppler probe.
- The cuff is inflated until the arterial signal is lost and then slowly deflated. The pressure at which the arterial signal returns is the systolic pressure.
- The procedure is repeated on the other arm.
- A cuff is placed on the lower leg above the malleoli protecting the ulcer site.
  Do not put the cuff on the calf above the ulcer as this will give inaccurate results.
- The dorsalis pedis and posterior tibial arteries are insonated in turn with the Doppler and the systolic pressure recorded as for the arm.
- The ABPI is calculated by dividing the highest ankle pressure by the higher of the two arm pressures

$$\text{ABPI} = \frac{\text{A = highest ankle systolic pressure}}{\text{B = higher Brachial systolic pressure.}}$$





Indices are normally >1.0 in the absence of arterial disease. Several guidelines exist as to the significance of ABPI measurements (Table 3). One of the most important points to remember is that although 0.80 is taken as the cut-off for using high compression dressings any value <0.92 represents the presence of arterial disease. Therefore if patients find high compression intolerable then one should consider that perhaps their arterial disease is more significant than the ABPI would lead you to believe. Conversely, if a patient has an easily palpable pulse in the foot it is unlikely that they have any significant arterial disease.

(D.P.) Dorsalis Pedis
(P.T.) Posterior Tibial

Table 3 Significance of ABPI measurements

>0.92	Normal
0.5 - 0.92	Mild/moderate claudication
0.35 - 0.50	Severe claudication
<0.35	Critical ischaemia

One must always question the accuracy of ABPI measurements if it is difficult to occlude the arteries as deceptively high pressures will be recorded in the presence of calcification, e.g. in diabetics (8). If any doubt exists then patients should be referred to specialist units where more detailed non-invasive tests can be performed.

Sample of Leg Ulcer Patient Assessment Form is given in appendix.

References

1. Elliot E, Russell B, Jaffrey G. Setting a standard for leg ulcer assessment. J Wound Care 1996;5:173-175.
2. Stevens J, Franks PJ, Harrington M. A community/hospital leg ulcer service. J Wound Care 1997;6:62-68.
3. Etris MB, Pribble J, LaBrecque J. Evaluation of two wound measurements methods in a multi-center, controlled study. Ostomy Wound Management 1994;40:44-48.
4. Buntinx F, Beckers H, Briers A, et al. Inter-observer variation in the assessment of skin ulceration. J Wound Care 1996;5: 166-170.
5. Ray SA, Srodon PD, Taylor RS, et al. Reliability of ankle/brachial pressure index measurement by junior doctors. Br J Surg 1994;81:188-190.
6. Callam MJ, Harper DR, Dale JJ. Et al. Arterial disease in chronic leg ulceration: an underestimated hazard? Lothian and Forth Valley leg ulcer study. BMJ 1987;294:929-931.
7. Fisher CM, Burnett A, Makeham V, et al. Variation in measurement of ABPI pressure index in routine clinical practice. J Vasc Surg 1996;24:871-875.
8. Carter SA. The relationship of distal systolic pressures to healing of skin lesions in limbs with arterial occlusive disease, with special reference to diabetes mellitus. Scand J Clin Lab Invest 1973;239(Suppl):31.

SECTION 4

Management of Venous Leg Ulcers

Pauline Diamond

Compression therapy

The mainstay of conservative management of venous leg ulcers is focussed on the reversal of venous hypertension and the most effective way to manage uncomplicated venous leg ulcers is the correct application of graduated compression to the leg (Table 1).
This should only take place following clinical and Doppler assessment of each patient with leg ulcers. (see section 3).

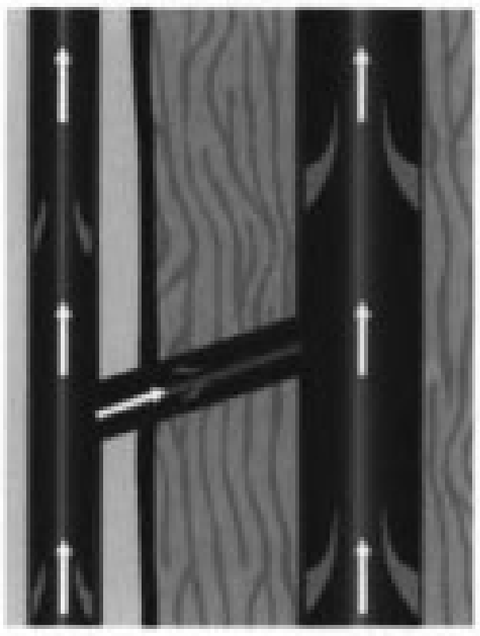
Table 1. Rationale for compression therapy of leg ulcers

To reduce high pressure in the superficial veins.

To aid venous return of blood to the heart, by increasing the velocity of flow in the deep veins.

To reduce oedema by reducing the pressure difference between the capillaries and the tissues.

To transport metabolic products away from tissues and allow the ulcer to heal.



Compression bandaging is usually used to achieve healing of venous ulcers while compression stockings are fitted for prevention of recurrence of ulceration, and for controlling oedema. Surgical referral may be appropriate via the general practitioner, for superficial vein surgery.

The first line management of venous leg ulcer patients following assessment with A.B.P.I. 0.8-1.2 is graduated high compression bandaging, including short stretch bandage regimes. Patients with an ABPI of < 0.8 should be referred for vascular assessment.

Before applying compression check the ABPI, and the ankle circumference.

Evidence regarding compression therapy

Compression vs. No Compression

Several studies have shown that compression improves healing rates compared to treatments using no compression or standard treatment.
In the Sligo/Leitrim study on venous leg ulcer patients where no compression was used in the period 1988/89 reported healing rates were 35% at six months (1). Following the introduction of Profore* four layer bandaging and staff training in theory and practice, healing rates improved to 79% at 3 months (2). Compression therapy is more cost-effective than non compression therapy because the faster healing rates saves nursing time. Studies have shown that compression improves healing rates compared to treatments using no compression (3,4).

The different grades of compression bandages are described in Table 2

* Trade Mark of Smith & Nephew

ABPI = Ankle Brachial Pressure Index

Table 2 (5)

Types of compression

Class 3 a	light compression	14-17mmHg at ankle
Class 3 b	moderate compression	18-24mmHg at ankle
Class 3 c	High compression	25-35 mmHg at ankle

High compression vs. low compression

The advantage of high compression was confirmed in a randomised controlled trial (RCT) in which patients with either four layer or short stretch bandaging healed faster than those receiving a paste bandage with outer support (6). More patients heal in 12 to 15 weeks with high compression (7). There is reliable evidence from two RCTs that high compression achieves better healing rates than low compression.

Multi-layer vs. single-layer

The superiority of multi-layer high compression systems over single layer systems has been shown by one large and two small trials which found more ulcers had healed at 24 weeks using 4-layer bandaging than were healed using a single layer adhesive compression bandage (8).

Multi-layer vs other types of compression bandaging

In a recent study Moffatt found higher healing rates when Multilayer bandaging was compared to two layer high compression (9)

The recent Cochrane review summarizes the systematic review of 22 trials on compression for venous leg ulcers, the reviewers' conclusions are outlined below.

Cochrane Library Review 2001

Reviewers' conclusions

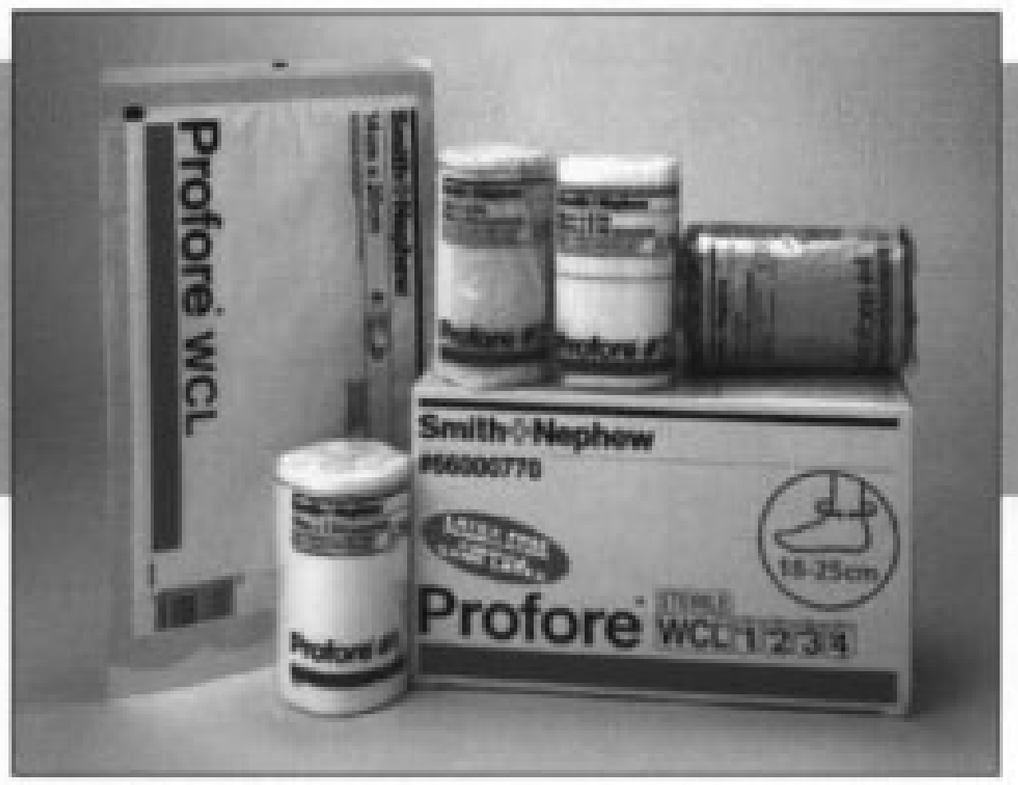
- Compression increases ulcer healing rates compared to no compression
- Mulitlayered systems are more effective than single-layered systems
- High compression is more effective than low compression but there is no clear difference in the effectiveness of different types of high compression

*Cullum N, Nelson EA, Fletcher AW, Sheldon TA. Compression for venous leg ulcers(Cochrane Review). In : The Cochrane Library, issue 1, 2001. Oxford: Update Software.

Multi-layer compression bandaging system (Profore)*

The multi-layer system was developed at Charing Cross Hospital in London by a clinical group (10). The concept underpinning the bandage development was the requirement for sustained compression, outlined by Stemmer in 1980 (11). This theoretical framework suggested an external pressure of at least 40mmHg at the ankle was required to achieve ulcer healing. With adequate padding this system is capable of sustained compression for at least a week. Recent studies by Hafner et al 2000 have confirmed this (12).

The ankle circumference measurement determines which Profore kit is to be used to achieve the optimal compression for each size of ankle. Kits available according to ankle measurement are: **<18cm, 18-25cm, 25-30cm, >30cm.**



All bandages are applied from the base of the toes to the tibial tuberosity with a 50% overlap. When using compression, accuracy of application is crucial to good management, and staff education is required.

The multilayers are:

- a padding layer (wool)
- a crepe bandage
- a light compression bandage
- a flexible cohesive moderate compression bandage
- a high compression bandage (in kits with ankle circumference over 25cm).

Short stretch bandages:

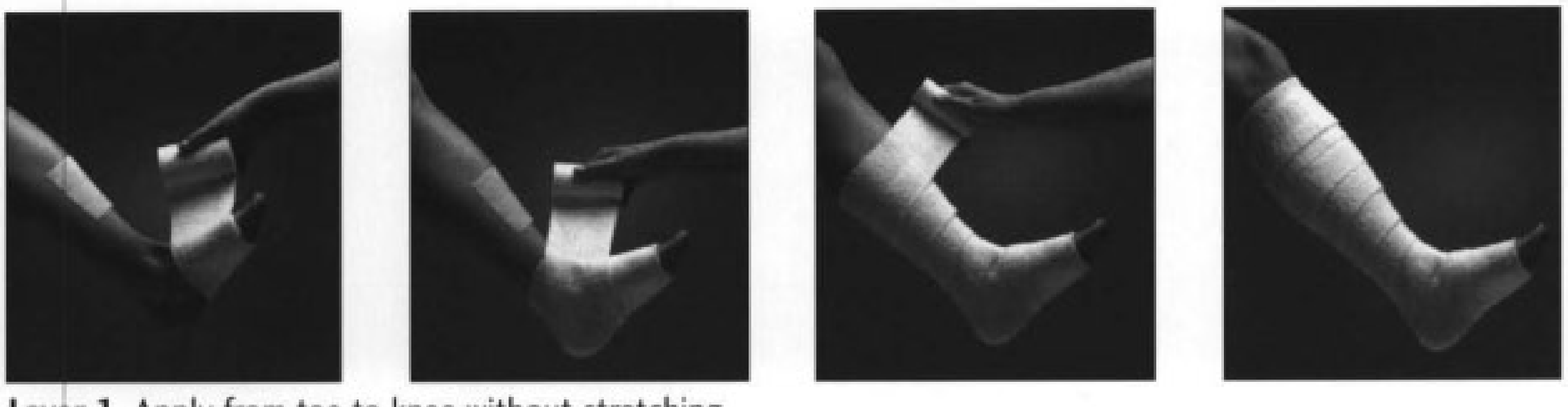
Short stretch bandages are inelastic. Effective pressure is achieved through low resting pressure and high working pressure. Short stretch bandaging is a suitable treatment for ambulant patients who find multi-layer bandage system a problem at night.

Patients suitable for reduced compression

Following consultation with the vascular team, reduced compression can be considered for selected patients. Patients with ABPI of 0.6-0.7 can have reduced compression using a padding layer, a crepe layer and a light compression bandage applied in a figure of eight fashion. Patients with ABPI of 0.7-0.8 can be considered for padding layer, crepe bandage and a moderate compression bandage e.g. cohesive bandage. (Profore Lite)*

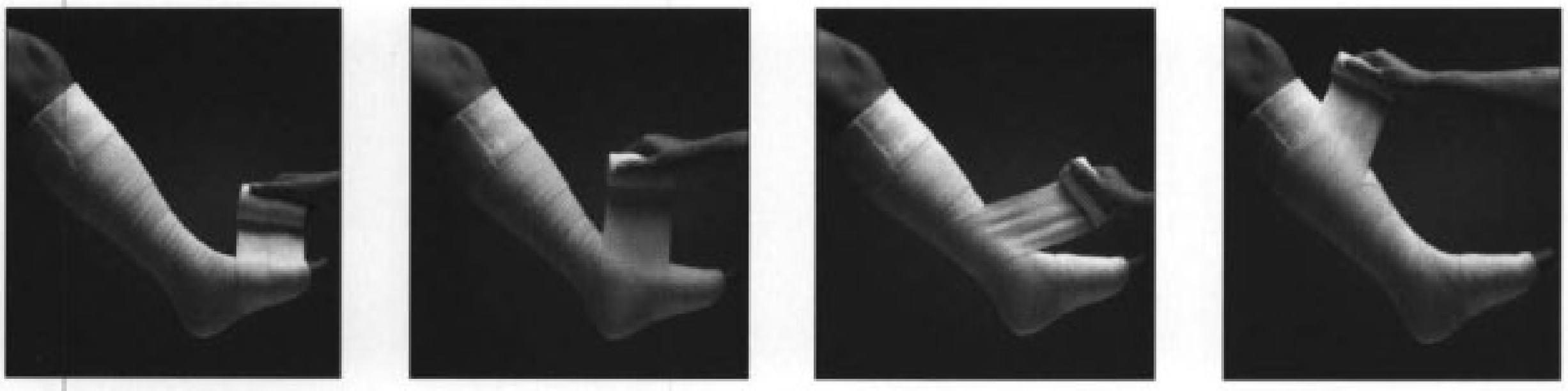
* Trade Mark of Smith & Nephew

Profore Bandage Application



Layer 1. Apply from toe to knee without stretching

Layer 2. Apply from toe to knee at 50% overlap in a spiral fashion



Layer 3. Apply from toe to knee at 50% stretch in a figure of eight fashion

Layer 4. Apply from toe to knee at 50% stretch and 50% overlap in a simple spiral

Profore +* Apply from toe to knee at 50% stretch and 50% overlap in a simple spiral

For a Bandage workshop in your area contact:
Clinical Nurse Advisor - Wound Management Smith & Nephew

Profore +* bandage available in kits where ankle measurements are 25 - 30cm or > 30cm

Patients not suitable for compression

Patients with ABPI of 0.5 or less indicating severe arterial disease should not receive any compression bandaging and should be referred to the vascular surgeon.
Patients with diabetes may have false high APBI readings, and compression should be used with caution. These patients should be referred to the vascular surgeon for assessment. Small vessel disease may be a problem (see section 1).

Patients with arterial disease are not suitable for high compression therapy. It can exacerbate ischaemia by decreasing limb perfusion. Patients with venous ulcers usually have an ABPI equal to or greater than 0.8. All leg ulcer patients need to be assessed to provide appropriate treatment (see section 3).

Management of leg ulcers

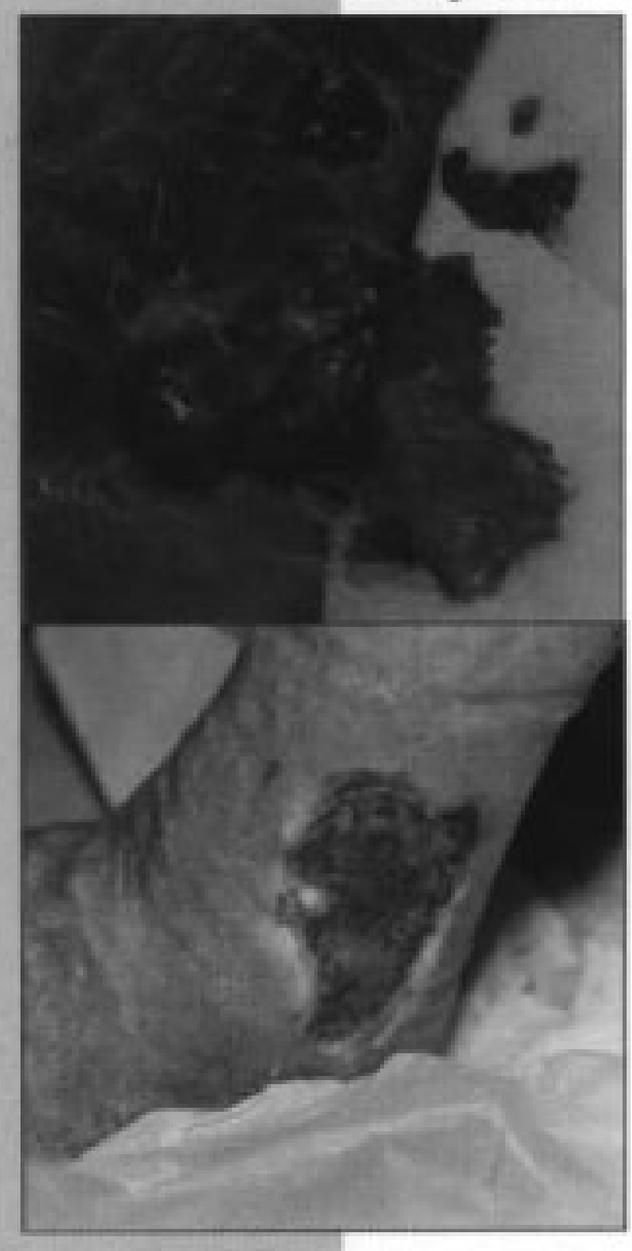
Environment for healing

- Involve patient with their care and management plan.
- Where adequate diet may be lacking, ensure a high protein diet, with zinc and vitamin C to promote healing. Where patients' clothing has become loose fitting this can be a good indicator of recent weight loss and referral may be required after nutritional assessment.
- Obese patients may require referral to nutritionist.
- In order for healing to take place give encouragement to motivate the patient.
- Patients living alone or with elderly should be referred to day care services, where social isolation would also be addressed.
- Diabetic patients may need referral to the community nutritionist.
- Patients with ill fitting dentures should be referred, for dental care.

Cleaning leg ulcers

Most leg ulcers are colonised with non-pathogenic organisms. Attempts to "cleanse" or dry the wound are unnecessary other than to remove excess slough, and foreign bodies. Cleaning a wound that is granulating may interfere with the healing process.
Cleansing should take the form of irrigation rather than swabbing. It is gentler on healing tissue, causes less pain and will not interfere with the healing process. A clean technique is acceptable (13).

Dirty Ulcer



Clean Ulcer

Good practice for cleansing leg ulcers

- A plastic apron should be worn and changed after each patient.
- Hands should be washed and gloves worn for removal of dirty dressings and for cleansing activity.
- Place the leg in a plastic lined container of warm drinkable tap water (14).
- It will render the leg 'socially clean' and is comforting to the patient.
- It facilitates the removal of wound debris and de-scaling of dry skin. (A plastic liner for each patient)
- Apply emollients to skin e.g. 50% white paraffin in 50% liquid paraffin.

Pain management

Venous leg ulcers are frequently painful. Sustained compression may relieve pain.
Elevation and rest will also reduce oedema. Monitor pain level with each assessment.
New pain may indicate infection. Refer patient to G.P. for appropriate pain management.

Factors that may delay healing

- Immobility
- Malnutrition.
- Careless bandage application and wound dressing technique.
- Too frequent dressing change.
- Application of inappropriate or harmful wound care products.
- Negative attitudes to healing by patient and staff.
- Failure to document changes and to evaluate the effectiveness of treatment regimes.

Dressing Leg ulcers

The application of sustained graduated sustained compression is of far greater significance than the dressing selection. Venous leg ulcers are usually moist wounds and a simple low-adherent low cost permeable dressing can be used with a hydrogel. Leg ulcers should not be allowed to dry out as research has demonstrated that moist wounds heal faster. Dressings remain in-situ for one week. In heavily exuding wounds more frequent dressing change is required.

To provide the optimum wound healing environment the choice of dressing may need to change as the wound progresses through the stages of healing.
The main aim is to select a dressing material that most suits the individual patient.

Treatment of chronic wounds

To prepare the wound bed to heal, slough and bacteria have to be removed and exudate managed.

Removal of slough (debridement)

- Excision of necrotic tissue is rarely necessary with leg ulcers.
- Debridement may be enhanced by the use of an enzymatic wound debridement agent (e.g. *Iruxol mono), hydrocolloid dressings or hydrogels.

Reduction of bacteria

Cadexomer iodine, (Iodoflex†) may be useful in removing slough and reducing the bacterial load and thus accelerate healing. Danielson found slow healing wounds with pseudomonas aeruginosa had a negative culture following one week of treatment with cadexomer iodine (15).

* Trade Mark of Knoll AG. Licenced to Smith & Nephew
† Trade Mark of Smith & Nephew

Colonisation by micro-organisms with no host reaction in itself is not associated with delayed wound healing (16). Infection is described as deposition of bacteria in tissue with associated host reaction (17). Wound swabs are only necessary if clinical signs of infection are present when systemic antibiotics may be prescribed. Topical antibiotics should never be prescribed.

Reduction of exudate

The presence of serous fluid in an open wound plays a vital role in the healing process. Excessive exudate can produce maceration and excoriation of surrounding skin, which may lead to infection (18). Increase in exudate may indicate infection. With venous leg ulcers high compression bandaging will help to reduce exudate. Re-measurement of the ankle may be required, to indicate that correct compression is being applied. The choices of dressing to absorb exudate include: Alginates, Hydrocellular dressings, Hydrogels.

Malodorous wounds

Malodorous wounds can be very distressing for the patients and their families, leading to loss of appetite, social isolation and depression. If clinical signs of infection are present the infection should be treated with systemic antibiotics. A variety of dressings are useful in the management of the malodorous wound:

- Charcoal impregnated with silver is bacteriostatic.
- Alginate and charcoal dressing.
- Cadexomer iodine, (Iodoflex)* where tolerated can eliminate odour and absorb exudate.

Eczema/Dermatitis (see Section 6)

- Patients present with an itchy erythema and a weeping scaling leg.
- Immerse the leg in warm drinkable tap water to assist de-scaling
- Apply an emollient.
- Avoid the use of tapes and adhesives. A light cotton tubigauze will support the dressings when surrounding skin is fragile.
- Graduated compression is the main treatment for venous eczema.
- Refer to G.P. who may prescribe a mild topical steroid to treat the inflammatory response.
- Referral to the dermatologist may be required via the G.P.

Patient education

Education is the key to the prevention of leg ulcer recurrence.
In order to prevent ulcer recurrence, the patient's family and carers should be educated.
Education leaflets should also be used.

Patient lifestyle - education

- Walk for 30 minutes at least twice a day, where possible.
- Practice leg and foot exercises when sitting.
- Elevate legs above waist level when sitting, never allow them to be dependent, where possible.
- Standing for long periods should be avoided.
  Minor lifetime changes can improve situations (Use of a high stool when preparing food)

* Trade Mark of Smith & Nephew

The recurrence rate of leg ulceration, measured in 1998, was found to be18.5% (19). All patients with venous ulceration should be fitted with compression stockings (see Table 3).

Guidelines to prevent ulcer recurrence:
Usually below knee compession stockings are used.
Replace two pairs of compression stockings at least every six months at re-assessment.
Ankle circumference is measured 2 cm above the malleolus.
Stockings are put on before getting up in the morning.
Skin care should continue nightly and good skin hygiene maintained.
Patients should be encouraged to remain active. Elevation and rest should be emphasised.
Re-assessment should be planned for all patients.

Monitoring and evaluation

Holistic accurate assessment and weekly re-assessment should be documented, using a leg ulcer assessment and progress chart. Wound tracing will give indication of the ulcer progress and re-assure patients and staff.
The patient and carers should be kept informed and involved in the decision-making process. Lifestyle changes eg. diet, exercise, elevation , skin care, social contact should be part of the care plan. Outcomes should be evaluated.

Table 3 Hosiery compression classes

Pressure (mmHg)	British Class	European Class
14-17	I	
18-21		I
18-24	II	
25-32		II
25-35	III	
36-46		III

References

1. Diamond P. Management of leg ulcers in a rural community. A report of a study which examined the use of research-based information by nurses treating leg ulcers in the community. J Wound Care 1994; 3:269-71.

2. Diamond P. Cash savings from treatment of ulcers. Irish Medical Times 1996: September 27th (News).

3. Moffatt C.J,Franks PJ, Oldroyd M et al. Community clinics for leg ulcers and impact on healing. BMJ 1992;305:1389-1392.

4. Callam MJ, Harper DR, Dale JJ et al. Arterial disease in chronic leg ulceration: an underestimated hazard? Lothian and Forth Valley leg ulcer study. BMJ 1987b; 294: 299-31.

5. Thomas S. Bandages and bandaging, the science behind the art. Care Science and Practice 1990;8:56-60.

6. Duby T,Hoffman D, Cameron J et al. A randomised trial in the treatment of venous ulcers comparing short stretch bandages, four layer bandage system and long stretch-paste bandage system. Wounds compendium of Clinical Research and Practice 1993; 5: 276-279.

7. Callam MJ. Prevalence of chronic leg ulceration and severe chronic disease in western countries. Phlebology 1992; 1: 6-12 (supp).

8. Nelson A, Harper DE, Ruckley CV et al. A randomised trial of a single layer and multi-layer bandages in the treatment of chronic venous ulceration. Phlebology 1995b; 915-916 (supp 1).

9. Moffatt CJ Symposium on compression therapy, a randomised control trial comparing 4 layer with 2 layer high compression bandaging in the management of chronic leg ulceration. Back to the future. 11th Conference of European Wound Management Association. May 2001 Dublin, Ireland.

10. Blair SD, Wright DDI, Backhouse CM, Riddle E, McCollum CN. Sustained compression and healing of chronic venous ulcers. BMJ. 1998;297:1159-61.

11. Stemmer R, Marescaux J, Furderer C. Compression treatment of the lower extremities particularly with compression stockings. Dermatologist 1980; 31: 355-65.

12. Hafner J, Botanakis I, Burg G. A comparison of multi-layer bandage system during rest, exercise and over 2 days wear time. Arch. Dermatol 2000;136: 857-63.

13. Lovell,1980. Cited by Ewan C and White R. Teaching Nursing: A self instructional Handbook.Croom Helm, London, 1986.

14. Angeras NH, Brandberg A, Falka A, Seeman T. Comparison between sterile saline and tap water for the cleansing of acute traumatic soft tissue wounds. Eur. J. Surg. 1992; 158: 347-350.

15. Danielson L, Cherry GW, Harding K, Rollman O. Cadexomer iodine in ulcers colonised by pseudomonas aeruginosa. J Wound Care 1997; 6: 169-172.

16. Trengrove et al. Qualitative bacteriology and leg ulcer healing, J Wound Care 1996; 5: 277-280.

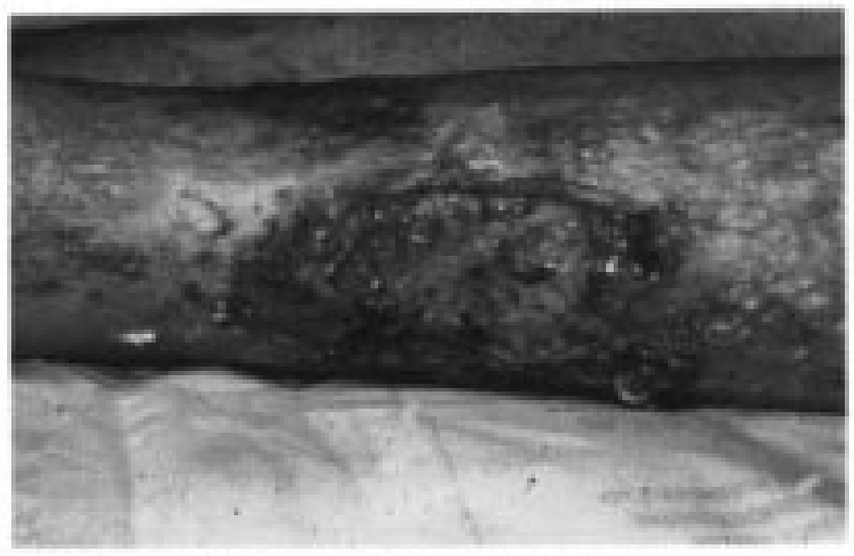
17. Ayton M. Wounds that won't heal. Nursing Times 1985; 46; 16-19.

18. Thomas S. Assessment and management of wound exudate. J Wound Care 1997; 6: 327-330.

SECTION 5

The Management of Infected Leg Ulcers and Guidelines for referral to hospital.

Dermot Hehir



The incidence of leg ulcers in the community varies from 0.06 - 0.20% of the total population - the majority of patients are elderly females and are managed in the community by Public Health Nurses (see section 2). Most ulcers are associated with venous hypertension and are managed successfully by a combination of compression therapy, elevation, nutritional support and exercises. When healed, recurrence should be minimised by appropriate compression hosiery / exercise regime. Unfortunately, some patients' ulcers fail to respond to conventional methodology and require further evaluation.

A chronic ulcer occurs when the preferred ordinary sequence of repair is disturbed at one or more stages during the healing process (Table 1).

Table 1 Usual sequence of regeneration / repair.

Inflammation
Proliferation
Re-epithelialisation
Remodelling

When the skin is breached, the major host defence mechanism is injured. In addition, serous exudate at body temperature is a prime culture medium and the gaiter area - site of most lower limb ulceration - is a site of sub-optimal wound healing.
The presence of wound associated bacteria does not necessarily indicate infection - some bacteria may even facilitate healing and wound inflammation may not necessarily be caused by infection (1). Additionally, wound bacteria may be transient and not detected on random culture swabs.
Distinguishing the difference between bacterial presence and pathological contamination is important; the presence of the cardinal signs of sepsis - fever, erythema, swelling, pain and leucocytosis are usually obvious and a diagnosis of infected ulcer is readily made. More difficult however, is the assessment of the
combination of host pathology and bacterial factors and to what extent it may contribute to impair ulcer healing. The risk of significant wound infection is broadly determined by host resistance versus bacterial virulence; bacterial load (a healthy,
fit active individual is less likely to develop wound infection than an elderly
debilitated patient).
The most common host factors associated with delayed wound healing of leg ulcers are outlined in Table 2.

Table 2 Factors associated with difficulty in healing leg ulcers.

Ischaemia / arterial disease
Infection
Vasculitis
Diabetes mellitus
Rheumatoid arthritis / collagen disorders
Malignancy
Drugs
Immunosuppression
Lymphoedema
Malnutrition

Since the risk of significant infection is increased in ulcers of long duration, factors associated with delayed healing are also potentially associated with wound infection. A large spectrum of organisms may be isolated from leg ulcers - the most frequently isolated bacteria in leg ulcers are outlined in Table 3 (2).

Table 3 Distribution of bacteria in leg ulcers (2).

Organism type		% distribution
Staphlococcus	aureus	56%
Streptococcus	pyogenes	29%
Streptococcus	fecalis	33%
E. Coli		56%
Proteus		52%
Pseudomonas		33%
Klebsiella		33%
Clostridium perifringens		26%
Bacteroides		7%
Acinobacter		4%

Assessment

When the ulcer proves resistant to conventional therapy, further assessment is desirable. Many factors associated with poor wound healing are also associated with infection and it is necessary therefore to identify all potential relevant factors. Table 4 includes relevant information obtainable from the patient, the GP, PHN or other reliable collateral source. It is important to elicit a history of systemic conditions that might cause pruritus - e.g. dermatological complaints, allergy, skin infestation. A history of poor patient compliance or tendency to interfere with bandages is valuable. Patients with painful ulcers may not be compliant, especially if the dressing results in further pain. Previous lower limb trauma with silent DVT may have resulted in post phlebitic limb, information regarding occupation (possibly involving long periods of standing), drug therapy and systemic diseases should be sought.

Table 4 Information obtainable from the patient or collateral source.

History of varicose veins
Deep venous thrombosis
Lower limb fracture / injury
Family history of varicose veins, eczema
Phlebitis
Aching
Nocturnal cramps
Pregnancy
Skin changes - ulcer in gaiter area, lipodermatosclerosis, oedema, flaring, eczema

A thorough physical examination is necessary specifically seeking evidence of the disorders as outlined in Table 2. Assessment of the ankle brachial index is required if any doubt exists about peripheral pulses; in the presence of infection, oedema may present difficulty in palpating peripheral pulses.

Wound assessment is vital. On removal of the dressing, malodour may indicate infection - the characteristic sweet odour of pseudomonas is an example. The presence of profuse exudate may indicate infection, as may purulent eschar. Surrounding erythema / cellulitis may indicate inflammation or infection. Oedema may result from infection but may also indicate lymphoedema, venous hypertension associated with varicose veins or the presence of co-morbid conditions such as cardiac failure or intra-abdominal malignancy.

Routine blood investigation should include full blood count, ESR, urea and electrolytes, C reactive protein, liver function tests, lipid profile and antibody screen. Duplex scan is required to outrule silent deep venous incompetence in particular but may also identify unsuspected long or short saphenous incompetence.
Plain X-rays are frequently unhelpful in suspected osteomyelitis and it necessary to obtain an isotope bone scan to confirm suspected underlying bone involvement.

Bacteriological assessment. If possible, the laboratory technician should extract and process samples as quickly as possible in order to maximise efficiency and minimise erroneous bacteriology. The most effective sampling of bacterial flora is by obtaining a sample of exudate from the wound (3). If the patient is hospitalised, screening for MRSA is prudent. In overt sepsis, culture swabs should be obtained prior to administering systemic antibiotics. Delayed healing may be due to sub-clinical bacterial infection and when suspected, quantitative cultures may be useful especially when considering transient use of topical antimicrobial agents.

Treatment / dressings

General symptomatic treatment is directed at removal of debris (escharectomy), controlling exudate (thus reducing local skin damage), maintaining a moist environment (to promote epithelialisation), pain relief to optimise mobility and compression therapy (removal of venous hypertension).
The most common sources of infection include self contamination, contact (clothing, dressings, hands) and airborne (dust, droplets)- this knowledge must be utilised to disrupt bacterial contamination.

Wound cleansing:

Reduction of exudate and eschar is achieved initially with normal saline or water. This can be performed with a moist gauze swab, gently pouring the solution over the ulcer, irrigation with a syringe or proprietary spray cleanser. Chemical or surgical debridement may be required in addition.

Antiseptic agents have antibacterial properties but also have cytotoxic properties which may inhibit healing by interfering with healthy granulation tissues (4). The agents of choice (Iodine or Chlorhexidine) are used when blood supply is impaired and in patients with impaired immunity. Their use is primarily to reduce bacterial load.

Antibiotics:

The use of antimicrobial agents in the management of lower limb ulceration is controversial: unfortunately there is a tendency to over-treat, thus resulting in the unfortunate side effect of encouraging the growth of and subsequent colonisation by antibiotic resistant organisms.

Topical antimicrobials.

The aim of topical agents is to reduce bioburden and the choice is determined by the offending organisms. While clinical judgement may detect organisms (i.e. pseudomonas - green colour, characteristic odour) laboratory culture reports are usually used to select a topical agent. If available, quantitative culture may be utilised to indicate decrease in the bioburden (no. of bacteria) as a response to treatment. Skin sensitivity may occur - neomycin, perfumes and lanolin may be present in topical antimicrobial preparations. These should be avoided where possible as they are common allergens (5). Topical agents can only treat the wound surface and will not effectively treat underlying deep infection. Their use should be re-assessed after 2 weeks or if deeper infection is suspected.

Systemic antibiotics:

Systemic antibiotics should be used:

(a) when there are signs of systemic sepsis
(b) when infection extends beyond the ulcer margin or
(c) if the ulcer base extends deeply to underlying bone.
(d) if the patient is obviously septic, standard hospital protocol will probably dictate the administration of broad spectrum antibiotics.

In an ulcer of short duration, gram positive cover is usually adequate pending the availability of culture / sensitivity reports. In patients with immunosuppression, broad spectrum agents including gram negative and anaerobes should be included.

When to refer to hospital

The majority of leg ulcers are managed very well in the community by nurses. However, some leg ulcers should be referred to hospital clinics for further assessment or a review of treatment. Arterial or malignant ulcers may need surgery as will those venous ulcers that arise secondary to varicose veins. Diabetic ulcers need careful management, while those with severe skin problems will require dermatological advice. The criteria for referral are listed in Table 5.

Table 5. Criteria for referral of the patient to hospital.

Suspected malignancy
Arterial / mixed ulcers
Diabetes mellitus and ulcer
Sepsis - cellulitis, fever, leucocytosis
Absent pulses with impaired ABI
Failure to heal with standard regime
Underlying co-morbidity
Deterioration
Recurrent ulceration
Need for surgery - varicose veins surgery, skin graft, biopsy, revascularisation
Pain management

References.

1. De Haan B, Ellis H, Wilkes M. The role of infection in wound healing. Surg Gynecol Obstet 1974; 105, 283.

2. Lawrence JC. Reducing the spread of bacteria. J Wound Care 1993; 2, pp.48-52.

3. Lawrence JC. Wound infection. J Wound care 1993; 2, pp.277-280.

4. Rodeheaver GT. Wound cleansing, wound irrigation, wound disinfection. Krasner D, Kane D. eds. Chronic wound care: A Clinical Source Book for Healthcare Professionals 2nd ed. 1997: pp. 97-108.

5. Cameron J. The importance of contact dermatitis in the management of leg ulcers. J Tissue Viability 1997; 5 pp.182-185.

SECTION 6

Dermatology and Skin Care of the Lower Limb

Dorothy O'Sullivan
Frank C. Powell

The skin of the lower legs is susceptible to all the dermatoses that affect the gereral cutaneous surface, but is particularly prone to some specific inflammatory skin reactions. These may occur de novo, or appear secondarily to skin injury or disease. Chronic venous stasis, oedema with distension of the skin and ulceration are particular problems of the lower limb, which lead to distinctive cutaneous changes.

Eczema is a common feature in the clinical presentation of chronic leg ulcers. The term eczema encompasses a broad range of skin conditions and is used interchangeably with dermatitis.

Eczema/Dermatitis presents as:

- Itching (pruritis),
- Inflammation of the skin (redness, pain, swelling)
- Weeping (exudation) and scaling (exfoliation).

Eczema/Dermatitis may be:

- Endogenous and related to constitutional factors such as atopy or venous insufficiency
- Exogenous and precipitated by irritating substances in contact with the skin (irritating contact dermatitus), or development of allergy (allergic contact dermatitis).

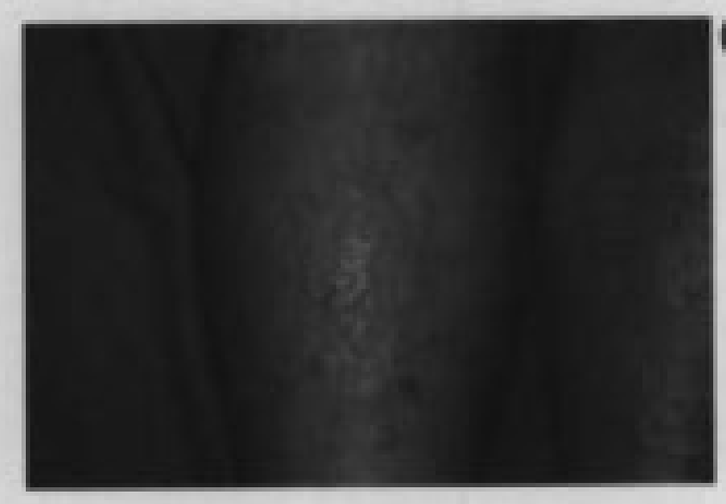
Sometimes elements of both endogenous and exogenous factors combine to produce eczematous change (1.6).

The common causes of eczema of the lower limbs include.

- Asteatotic Dermatitis
- Stasis Dermatitis (varicose eczema)
- Atopic Dermatitis
- Contact Dermatitis

1. Asteatotic Dermatitis:

This is a severe form of dry skin of the lower legs where fissuring or cracking of the surface takes place due to the loss of oily secretions. In this condition the skin is so dry and cracked, it resembles a dried out river bed in a desert climate. The low humidity of central- heating during winter, swimming, over-washing and the use of diuretics are contributing factors. This poorly recognised condition is particularly common in elderly patients. It is pruritic and presents as very dry skin with a network of erythematous superficial fissures which resembles 'crazy paving'(2)



(Fig1)

Management of Asteatotic Dermatitis:

The management of Asteatotic Dermatitis requires

- the use of a topical steroid ointment to reduce inflammation and itch
- a liberal application of bland emollients to counteract dryness
- avoidance of vigorous washing.

As with all applications of ointments and creams to the skin of the lower legs, it is important to apply these in a downward direction, consistent with the angle of growth of the hair follicles. Failure to do this can result in an occlusive folliculitis with the development of multiple minute itchy pustules on the leg. The topical steroid should be applied twice daily to the inflamed skin. This is then covered with an emollient such as 50% white soft paraffin and 50% liquid paraffin. When the eczema has cleared the continued use of emollients and soap substitutes prevents recurrence of the dryness. It should be emphasised to the patient that this maintenance treatment needs to be continued indefinitely (3).

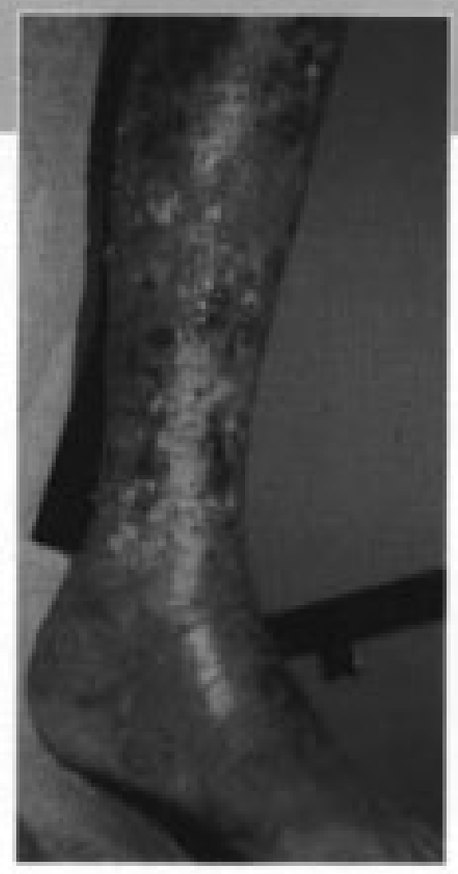
2. Stasis Dermatitis:

Venous insufficiency compromises the cutaneous surface of the lower limbs by reducing the efficiency of the microcirculation in the skin, and by distention due to oedema of the lower limbs.

Stasis dermatitis or varicose eczema:

- tends to affect older patients
- usually involves the gaiter area, particularly the medial aspect of the lower leg, around the medial malleolar region
- if secondarily infected, can become more generalised.
- stasis dermatitis may be acute or chronic

The *acute phase* is characterised by pruritus, erythema, and scaling. Small blisters or vesicles can appear, which can rupture and weep and subsequently form crusts. Excoriation causes further skin damage, and the introduction of pathogenic bacteria, which leads to secondary infection, ie impetiginised eczema.
In the *chronic phase* of stasis dermatitis the skin develops a scaly brown pigmentation due to the deposition of haemosiderin from extravasated red blood cells and the surrounding cutaneous surface becomes bound down (indurated) (see section 1). Eczematous skin with its compromised barrier function is vulnerable to irritation by topical medicaments, which may provoke dissemination of the eruption to other skin areas (4).



(Fig 2 Acute Stasis Dermatitis)

Management of Stasis Eczema:

Health care professionals looking after patients with leg ulcers know the importance of maintaining healthy skin around the ulcer. Failure to do so can lead to increased morbidity and a delay in healing (5).

It is important that the underlying venous hypertension is controlled by adequate support stockings or compression bandages. The acute stage of stasis eczema is managed by using a dilute solution of potassium permanganate which has an antibacterial effect as well as assisting in drying exudation. The affected leg should be immersed in the solution for no longer than 10-15 minutes at each dressing change. This should then be followed by the application of a moderate strength corticosteroid ointment such as betamethasone 0.1% RD ointment daily for one week approximately. This should be gradually replaced by a simple emollient, which does not contain known sensitisers. Creams instead of ointments are often more acceptable to the patient as they are less greasy. However, it is important to inform patients that creams contain preservatives and emulsifiers, and these allergens may cause sensitivity problems in patients with a leg ulcer. A simple emollient such as a mixture of 50% white-soft paraffin and 50% liquid paraffin should be used regularly to keep the skin lubricated. If treatment with the topical corticosteroid is stopped suddenly, there may be a rebound effect and the eczema may recur, so gradual reduction is important (1,4).

In the chronic phase when the skin is dry and scaly the affected leg should be lubricated with an emollient such as 50% White Soft Paraffin and 50% Liquid Paraffin to keep the skin hydrated. Systemic antibiotics should be given over a period of a week to 10 days only if significant infection is present. Topical antibacterial corticosteroid preparations are indicated if infected eczema is present.
The skin surrounding the ulcer should be protected from wound exudate by using an absorbent dressing and the application of a topical barrier preparation such as zinc paste.

Management of Stasis eczema

- Treat venous hypertension (compression therapy)

Acute

- Soak leg in potassium permanganate solution 10-15 min
- Apply moderate strength corticosteroid ointment daily for one week
- Reduce steroid and replace with simple emollient (50% white-soft paraffin and 50% liquid paraffin) to lubricate skin

Chronic

- 50% White Soft Paraffin and 50% Liquid Paraffin to keep the skin hydrated
- Protect surrounding skin with topical barrier e.g. zinc and absorbent dressing
- Systemic antibiotics only for established infection

3. Atopic Eczema

Atopy is a term used for a genetic predisposition to form excessive IgE antibodies and to develop one or more of a group of disorders, which include asthma, hay fever, urticaria, some food allergies and a particular type of eczema (8).
Atopic eczema generally starts after the second month of life. It affects 3% of infants but its appearance may be delayed until adult life. It usually improves as the child grows older, but in approximately 10% of patients it persists through adult life. It is a chronic dry itchy and irritating skin condition. The cause of atopic eczema is unknown. It affects the flexural surfaces with involvement of the popliteal fossae and the anticubital fossae but it can become generalised. Young children and some adults are affected particularly on the ankle region. The persistent and severe pruritus (itch) leads to constant scratching (excoriation), with damage to the cutaneous barrier function and susceptibility to secondary bacterial infection (impetiginisation).

Management of Atopic Eczema:

General management measures are directed towards reducing pruritus and correcting dry skin. Avoidance of a warm atmosphere (which increases skin heat and dryness), woollen clothing (which irritates the sensitive skin), strenuous exercise, or prolonged sunlight exposure (which increases body heat and perspiration) are important.
Atopic patients are prone to viral infections especially widespread herpes simplex, warts and molluscum contagiosum, and the clinician must be clinically vigilant to recognise these developments. Treatment is aimed at regular lubrication of the skin, the intermittent use of mild topical steroids and antihistamines. Systemic antibiotics are often required for flares of eczema as the inflamed skin becomes infected. It is important to recognise infection in the eczematous skin. If the increased inflammation is mistakenly thought to represent more active eczema, a stronger topical steroid might be prescribed resulting in deterioration of the patient's condition.

Management of Atopic Eczema

- Avoid warm atmosphere ⟶ increases skin heat and dryness
- Avoid woollen clothing ⟶ irritates sensitive skin
- Avoid strenuous exercise or prolonged sunlight ⟶ increases body heat and perspiration
- Watch for viral infections ⟶ especially herpes simplex and molluscum contagiosum
- Regular lubrication, mild topical steroids, antihistamines, systemic antibiotics for established infection.

4. Contact Dermatitis

Contact Dermatitis is an exogenous eczema caused by external factors that either directly irritate the skin (Irritant Contact Dermatitis) or cause an allergic reaction (Allergic Contact Dermatitis). Contact Dermatitis is normally limited to areas of direct contact with the skin, but if severe or secondarily infected Contact Dermatitis may become generalized.

(a) Irritant Contact Dermatitis

Acute irritant dermatitis usually occurs after a short single exposure to a potent irritant for example, strong acid or alkali. Wound exudate has a very irritant effect on skin surrounding an ulcer and can create an irritant contact dermatitis. Preparations used in the treatment of varicose eczema such as, antiseptics, adhesives, bandages applied directly to the skin may be contributing factors in the production of this type of skin reaction (4).

(b) Allergic Contact Dermatitis

This occurs following exposure to and sensitisation by an agent capable of initiating an allergic reaction (allergen). Further exposure to the agent in a sensitised person results in an eczematous reaction. This process occurs as a result of cell-mediated immunity and is a type 1V hypersensitivity reaction. The occlusive nature of many leg ulcer applications on broken or eczematous skin can create the perfect environment for sensitisation to develop. Patients may become allergic to any component part of their topical therapy including the dressings, emollients, creams, bandages or bath additives. Sensitisation requires exposure to the allergen and about 10 -14 days later the reaction becomes visible in the skin. The individual may not realise sensitisation has occurred until re exposure to the agent results in a severe reaction. Continued exposure to an allergen results in a progressively deteriorating clinical picture. To investigate patients suspected of having allergic contact dermatitis, referral to a dermatologist for patch testing is important. Identification of the causative allergen is essential for successful management, which involves its strict avoidance. Patients should be made aware that more than one product may have the allergen as a constituent (6, 7, 8).

Allergic Contact Dermatitis

General approach to skin care.

It is important to avoid the use of known irritants and allergens in the management of venous leg ulcers. When the barrier function of the skin is disturbed the patient is at risk of developing contact dermatitis. Therefore the following risk factors should be considered.

- The use of ointments instead of creams is advisable thus reducing the risk of sensitisation. Avoid using antiseptics and topical antibiotics
- Protect skin from elasticated bandages
- Do not use woollen bandages on dry skin
- Use appropriate dressings to absorb wound exudates
- Protect surrounding skin with the application of an appropriate barrier
- Products containing lanolin or fragrances should be avoided
- Ideally vinyl gloves should be used instead of latex especially where rubber allergy is suspected (4,5,6,7).

Recognition of infection.

The early recognition of the signs and symptoms of infection is important in the management of varicose eczema such as;

- ***Pain***
- ***Erythema***
- ***Increase in the level of exudate***
- ***Malodour***

Reserve a swab for culture and sensitivity and treat with an appropriate oral antibiotic.

Patient Education:

- Identification of allergens (sensitisers) by patch testing and the avoidance of allergens and irritants play an important part in the patient's management.
- The patient should be provided with a list of known sensitisers and advised not to use perfumated or "over the counter products" which may lead to sensitisation.
- Emollients are the first line of treatment and should be used regularly to hydrate the skin. A simple emollient which is unlikely to sensitise the patient such as 50% white soft paraffin /50% liquid paraffin should be used.
- Apply emollients in a downward motion in the direction of the hair growth and continue on a long term basis.

Table 1 (6) Common Allergens found in Venous Ulcer Therapies.

Allergen	Where found
Lanolin (Wool Alcohols)	Creams, barrier preparations, ointments, bath additives, baby products
Topical Neomycin/ Framycetin	Medicaments, medicated tulles, medicated powders
Parabens	Preservatives in medicaments and some paste bandages
Cetylstearyl alcohol.	Most creams, emulsifying ointments & paste bandages containing emulsifying wax
Colophony / ester of rosin	Adhesive used in tapes, bandages and dressings
Rubber	Elastic, tubular and cohesive bandages
Fragrances	Many over the counter preparations including creams and baby products

Legend for Figures

Fig 1
Asteatotic Dermatitis of the lower legs showing erythematous fissured skin on anterior aspects. Treatment with topical steroid application and emollients are essential here.

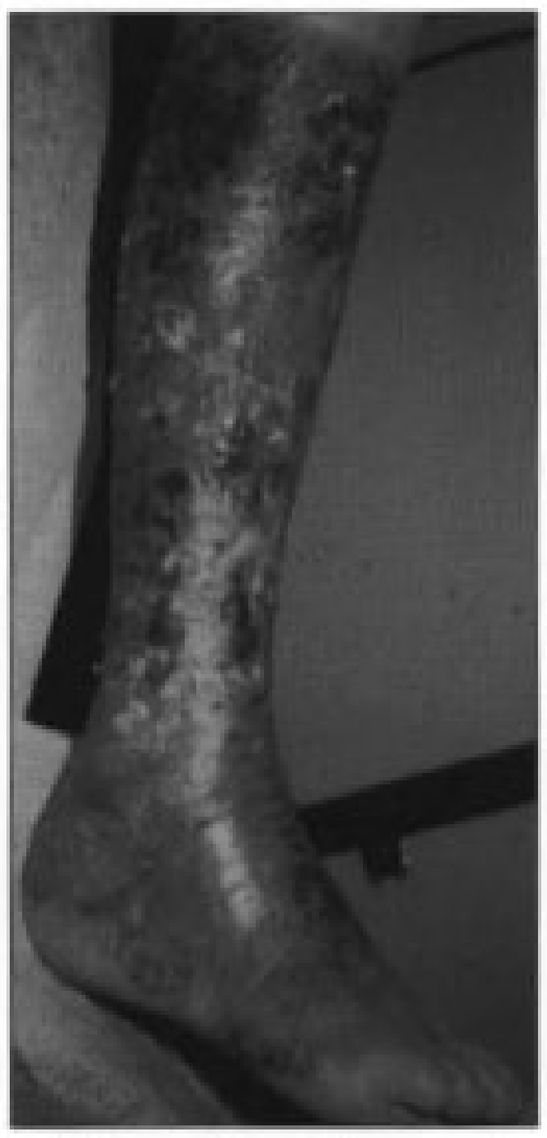


Fig 2
Acute Stasis Dermatitis with secondarily infected erythematous inflamed skin. Note crusting and exudation. Treatment with systemic antibiotics and topical antibacterial/ corticosteroid preparations led to complete clearing of the skin.

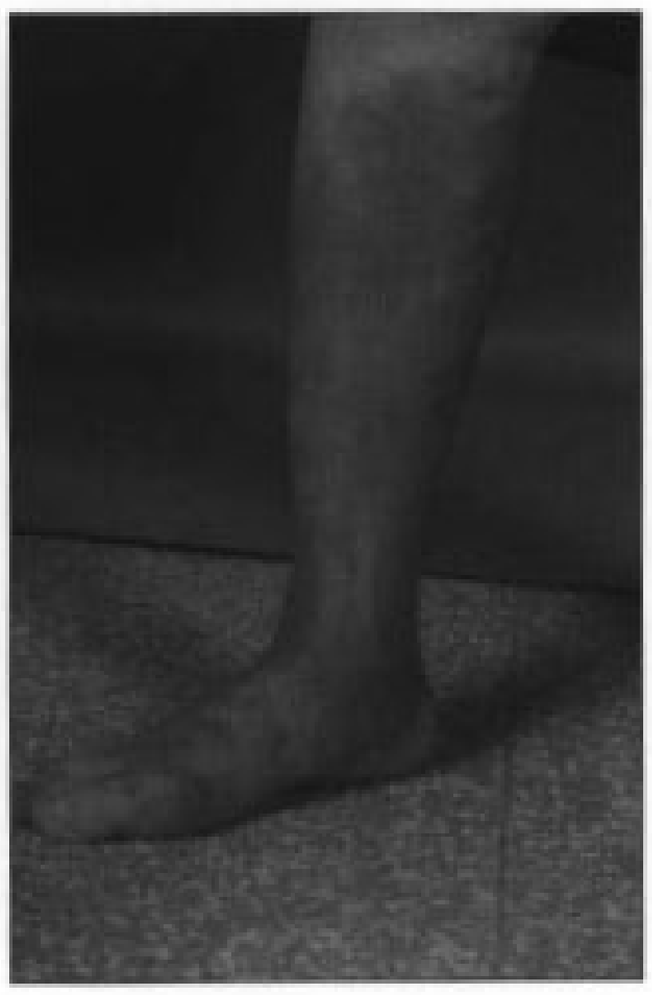


Fig 3
Allergic Contact Dermatitis to rubber, a constituent of tubular bandages. Note the clear outline of the rash corresponds to the area of contact between the skin and the allergen.

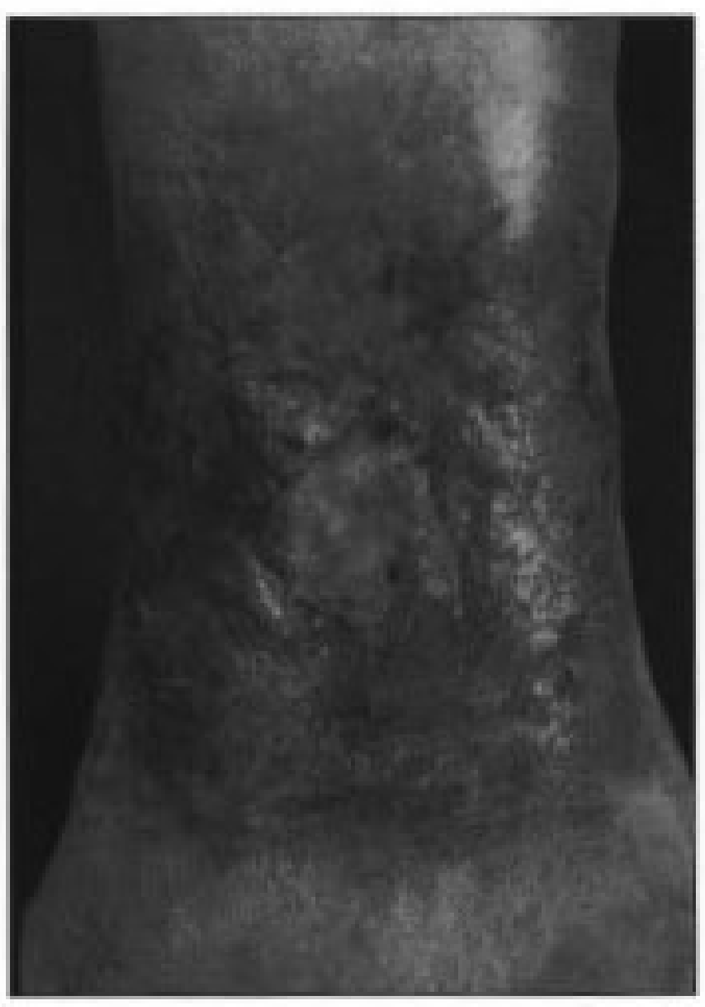


Fig 4.
Chronic changes around longstanding venous ulcer showing erythema and pigmentation secondary to haemosiderin deposition in the skin from extravasated red blood cells.

References

1. Cameron J. Skin Allergy Problems in Patients with Chronic Leg Ulcers. British Journal of Community Nursing 1999; 4; 6-12.

2. Hunter J. A.A., Savin J. A. & Dahl M. V. Clinical Dermatology. Blackwell Scientific Publications 1989: Chapter 9; 82.

3. Watts J. Out on a Limb. Nursing Times 1998; 94: 63.

4. Cameron J. Skin care for patients with chronic leg ulcers Journal of wound care 1998; 9; 461-2.

5. Cameron J. Powell S. Contact Kept to a Minimum. Nursing Times 1996; 92: 84-86.

6. Cameron J. The Importance of Contact Dermatitis in the Management of Leg Ulcers. Journal of Tissue Viability 1995; 5; 182-185.

7. Gooptu C, Powell S. The problem of rubber hypersensitivity (types 1&2) in chronic leg ulcer and stasis eczema patients. Contact Dermatitis 1999; 41: 89-93.

8. Powell, S. Contact dermatitis in patients with chronic leg ulcers. Journal of Tissue Viability 1996; 6: 103-6.

SECTION 7

Management of the Hard to Heal Leg Ulcer.

William P. Joyce

The impact of venous ulceration on the population at large is immense. Approximately 2-5% of the entire population may develop a venous ulcer. Since the establishment of specialized venous ulcer clinics (1) the rate of healing has increased significantly using simple wound care protocols and graduated compression therapy.

The Cavan Venous Ulcer Clinic was established in 1995 and since then we have treated over 1300 new patients with leg ulcers. The overall healing rate achieved is 81% with a recurrence rate of 6.5%. These excellent results can be achieved by dedicated trained staff and simple wound management protocols. However, a significant number of patients will show signs of being slow to heal or become non-healers.

In larger centres like our own "non healers" equate to a large number of patients who require significant extra resources as out-patients and they will also "eat-up" many hospital bed-days. Special strategies to treat this difficult group of patients must be developed to speed their rate of healing when compared to more conventional wound management strategies.

The first difficulty encountered is how to identify the patient that will be difficult or slow to heal at initial consultation. Various ad hoc methods have been used to date with little success. We have modified a scoring system originally described by Margolis et al (2) to predict healing. We have called this "The Rule of Six" and have found it to be a very accurate determinent of healing in a consecutive series of 100 patients with chronic venous ulcers.

The rule states that venous ulcers larger than six centimeters squared present for six months or more when treated with graduated compression are unlikely to heal within six months (table 1). Once these patients are detected alternative modalities of treatment can be used to accelerate healing and prevent many fruitless, costly and time consuming visits to the venous ulcer clinic.

Table 1. Rule Of six

Ulcer > 6cm^2
Ulcer present for > 6 months
Unlikely to heal in compression in six months

Wound Bed Management.

The management of chronic wounds has historically been the same as the management of acute wounds. Nobody will doubt the importance of wound debridement in the management of acute wounds. The situation with chronic wounds such as chronic venous ulcers and in particular the slow to heal ulcer is slightly different.

Chronic venous ulcers are intensely inflammatory producing large amounts of exudate which may interfere with the healing process. So the chronic venous ulcer in addition to producing non-viable tissue and eschars also has a major exudative component.

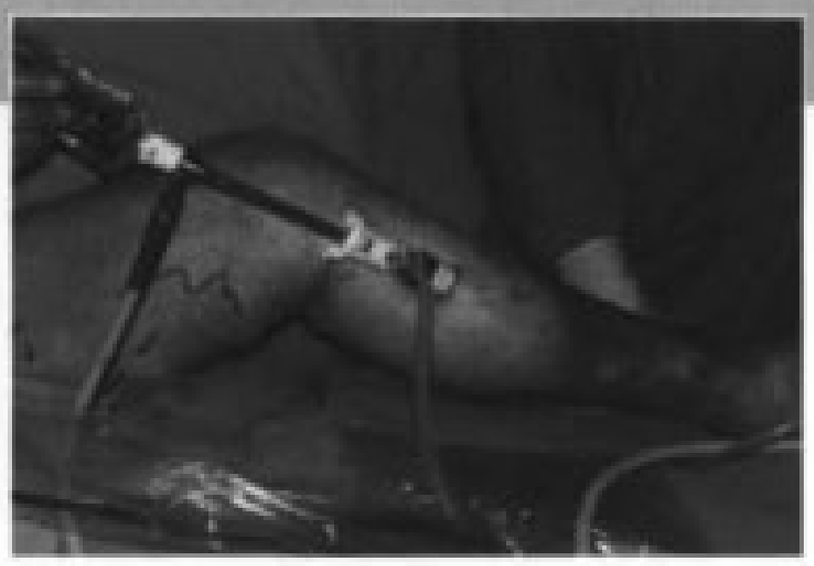
A healthy wound bed is vital for healing. This may be achieved by regular and frequent debridement, the use of agents such as topical collagenase and also by decreasing the local bacterial burden by the judicial use of slow release iodine products. There is no doubt that some non-healing chronic venous ulcers are 'stuck' in one of the phases of normal healing. Proper wound bed management can sometimes move the healing process onwards towards the next phase.

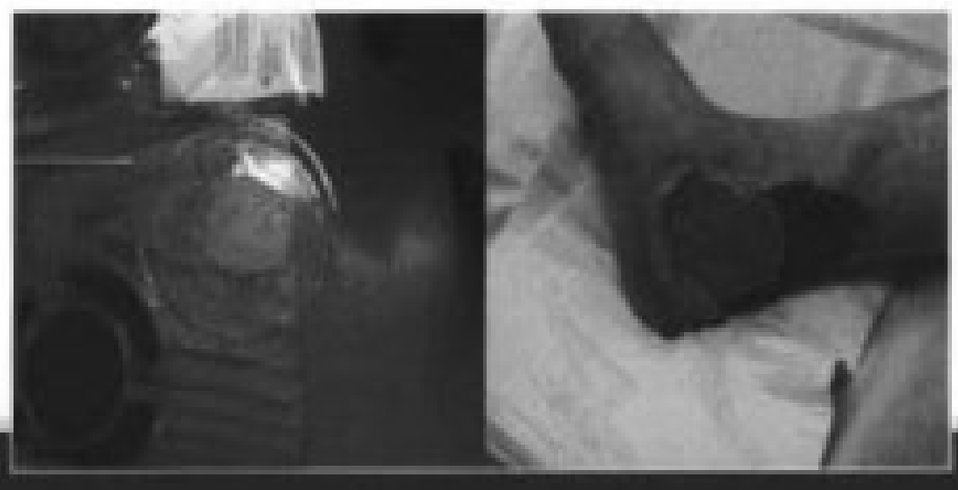
Sub-fascial Endoscopic Perforator Vein Surgery (SEPS).

This procedure should be reserved for patients with difficult to heal leg ulcers or patients with a previously healed leg ulcer i.e CEAP classification V and V1 (2). All patients undergoing this surgical procedure should have pre-operative assessment of the lower limb anatomy by colour coded Doppler ultrasonography. The incompetent perforators can then be marked on the skin of the leg in the operating room.

By insertion of the endoscope in the sub-fascial compartment of the lower leg individual perforators are seen and divided thus not damaging the overlying diseased skin. This sub-fascial space is always accessible even in patients with large ulcers and narrowing of the lower leg. The advent of a space making balloon device (Figure 1) has made the technique even easier to perform.

When clinically indicated conventional superficial venous surgery is always done in association with SEPS. The author has now performed this procedure on over 40 patients who fulfill "the rule of six". All ulcers to date have healed with no recurrences.





Bio-engineered Skin Substitutes.

The ideal treatment for chronic venous ulcers would entail the reversal of the underlying abnormalities of the veins which cause superficial venous hypertension. Compression therapy can achieve this goal for the majority but not for patients with the difficult to heal ulcer. Direct stimulation of wound fibroblasts with bio-engineered substitutes are potential treatments. It has been suggested that as individual characteristics of different chronic wounds become better understood specifically tailored cytokine/growth factor cocktails may be developed (4).

Tissue engineering is an emerging field which aims to maintain or restore tissue function using biomaterials, living cells or living tissue. A chronic venous ulcer is a live living model for wound healing so it would seem an appropriate setting to apply the principles of bio-engineering to the healing of venous ulcers.

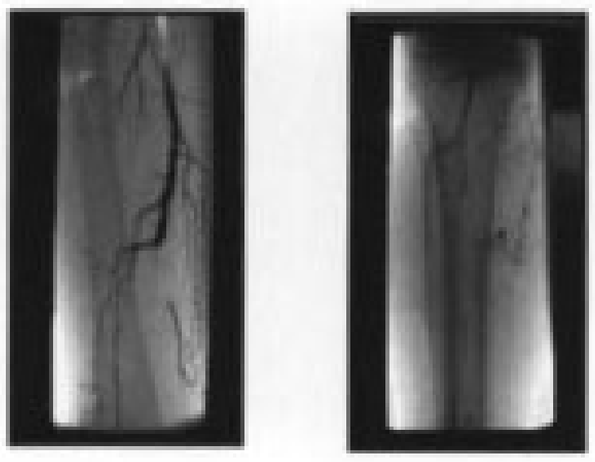
There are bio-engineered skin sustitutes commercially available which may accelerate healing. They consist of a complex combination of living fibroblasts embedded in a collagen matrix covered by keratinocytes (Figure 2). Although theoretically this may seem an attractive treatment option early results only show modest increases in healing rates in conjunction with standard compression therapy (5).

At the Cavan Venous Ulcer Clinic bio-engineered skin substitutes have been used on 24 patients with non-healing venous ulcers who have been in compression therapy for greater than one year. To date one third of patients have healed, one third now show signs of healing but are not healed yet and one third have shown no change.

Mixed Arterial Venous Ulcers.

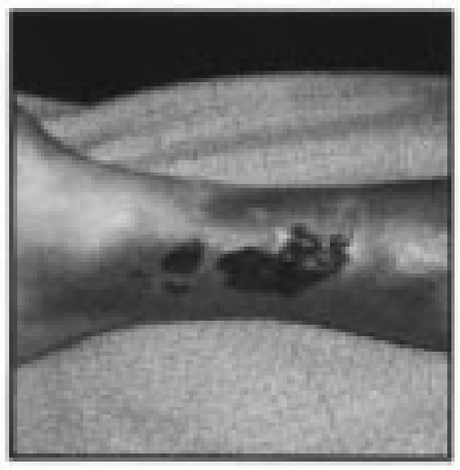
Accurate assessment in a specialized venous ulcer clinic by a vascular team can quickly distinguish a chronic leg ulcer with a significant arterial component. However a small group of patients with a slow to heal chronic venous ulcer who are **not** diabetic and have 'normal' (non-invasive) vascular assessment may still have a significant arterial component. The key to identifying these patients is the presence of persistent pain.

Invasive angiography may be required to reveal significant arterial disease which otherwise may have gone undetected. Endovascular or bypass surgical techniques may be required to achieve healing.



Angiography

Pre-surgery



Post bypass surgery

Summary

Chronic venous ulceration remains the most common but least understood cause of chronic leg ulcers. Current practice entails the institution of blind therapies which will be ineffective for a substantial number of patients with longstanding leg ulceration.

The clinical challenge is to identify the patterns of venous reflux and tissue damage which may respond to various prescribed treatments. The identification of the patient at initial consultation that will be slow to heal is essential. Therefore, alternative therapies may be commenced for those patients in whom conventional therapy would be sub-optimal or ineffective.

References.

1. Murphy MA. Joyce W.P. Chronic venous ulceration: current concepts and management. J Irish Coll of Physicians and Surgeons 2001,30:97-103.

2. Margolis DJ, Berlin JA, Strom BL. Which Venous Leg Ulcers will heal with limb compression bandages? Am J Med 2000;109 :15 -19.

3. The International Consensus Committee on chronic venous disease. J Vasc Surg 1995;21:635-645.

4. Growth Factors a time to take note. Editorial: Lancet 1990;336: 777-778.

5. Katz MH, Harris B, Carson P. Topical use of recombinant epidermal factor (h-ECG) in venous ulcers. J Dermatol Surg Oncol 1992 ,18:604-606.

Appendix

A Multi-Disciplinary Leg Ulcer Patient Assessment

Clinic:

Date_____

G.P's. Name: _____ P.H.N's. Name: _____

Patient's name: _____ Address: _____

G.M.S. No: _____ Telephone No: _____Referred by: _____

D.O.B: _____Sex: M ◯ F ◯ No. Pregnancies: ◯ Leg problems: _____Height: _____

Weight: _____Weight Loss: _____ Occupation: _____ Standing No.Hrs.Day: _____

Tobacco Usage Y/N: _____ How Many: _____ No. Years smoking in the past: _____

Nutrition: Fair/Poor, Balanced Diet, Proteins, Fruit, Veg, Fibre. Dairy Products, Carbohydrates

Takes Food Supplement or Vitamins Y/N: _____ Dentures Fit Y/N: _____ Pain 0-10: _____

Patient Medication for Sleeping: _____ Attending Day Centre Y/N: _____

How do you feel having a Leg Ulcer: _____ Do you think it will heal:? _____

Psychological State: Depressed, Anxious, Senile, Cheerful, Positive Attitude to treatment

Social Isolation: _____ Living with: _____Home Help Y/N: _____

What medical Problem has patient: _____

Is there a family history of Leg Ulcer or Varicose Veins Y/N: _____

HISTORY:

	RIGHT	LEFT
Onset 1st Ulcer (in years).....	_____	_____
Duration present Ulcer (in mths)	_____	_____
No. of episodes.....	_____	_____
D.V.T./White Leg.....	_____	_____
Pulmonary Emboli.....	_____	_____
Phlebitis.....	_____	_____
V. Veins Injections/Surgery.....	_____	_____
Leg Fractures/injury.....	_____	_____

ALLERGIES:

Antibiotics, etc..... _____

Dressings, Tapes..... _____

Paste Bandages..... _____

PREVIOUS TREATMENTS

Dressings:_____

Bandages:_____

Who Prescribed:_____

Who Dresses:_____

How Often:_____

EXAMINATION FINDINGS:

Walks: Alone/with stick/with frame

Bed Bound: Yes/No. Chair Bound: Yes/No

Fully Mobile/Limited Range/Immobile.

Obesity: None/Mild/Severe

	RIGHT	LEFT
Patient's Blood Pressure.....	_____	_____
Stasis Oedema.....	_____	_____
Eczema.....	_____	_____
Brown Pigmentation.....	_____	_____
Atrophe Blanche.....	_____	_____
Visible Varicose Veins.....	_____	_____
Induration/Lipodermatosclerosis...	_____	_____
Ankle Flare.....	_____	_____

ANKLE PULSES:

Paplable	Y/N	Y/N
Reduced	Y/N	Y/N
Absent.....	Y/N	Y/N
Ankle Circumference.....	_____cm	_____cm
Calf Circumference.....	_____cm	_____cm
Ankle Movements:	Full/Ltd./Fixed	Full/Ltd./Fixed

DOPPLER ASSESSMENT:

	mmHg	mmHg
Dorsalis Pedis Pressure.....	_____	_____
or		
Posterior tibial Pressure.....	_____	_____
Brachial Pressure.....	_____	_____
$\frac{\text{Ankle Pressure}}{\text{Arm Pressure}}$ = index	_____	_____

Bandage Combination or compression stocking to be implemented:

OTHER RELEVANT CONDITIONS:

Hypertension..... _____

C. Vascular A. ..... _____

Myocardial Infarction..... _____

Anaemia/Angina..... _____

Claudication/Rest Pain..... _____

Diabetes..... Yes/No

Urine Tested ..... Yes/No

By whom/Initial ..... _____

Congestive Cardiac Failure..... _____

Rheumatoid Arthritis..... _____

Osteo Arthritis..... _____

Transient Ischaemic Attacks..... _____

Poor Tissue Perfusion..... _____

Patient Medications ..... _____

SUMMARY OF CAUSES OF ULCER
(please circle)

Venous	Obesity	Poor Mobility
Hypertension	Aged	Heavy Smoker
Arterial	Arthropathy	Rheumatoid
Trauma	Vasculitic	Frailty	Diabetic

Malignant, Other: _____
Letter of:

Referral: to G.P Date: / /

Referral: to Other / /

DIAGNOSIS:
Venous Ulcer/Gravitational Ulcer,
Arterial Element/Severe Arterial Disease.

	ASSESSMENT						PLAN	IMPLEMENTATION			EVALUATION		
Date	**Ulcer** Type: Location: Size Exudate Colour Type, Amount Mild Moderate	**%Ulcer Bed** Necrotic Sloughy Granulating Epithelializing Wound margin: Colour Oedematous	**Infection?** Suspected Swab sent. Organism isolated. Erthema of skin inc. Exudate. inc.Pain. Heat Maceration Odour None/some Offensive	**Skin** Eczema Wet/dry Erythema Measure distance from wound	**Pain** Severity (0-10) Continuous Intermittent Site: at wound/ elsewhere Frequency Day/night At dressing change	**Trace** Yes/No Record Length Breadth Dept Date/Day	To Debride To reduce To treat Infection To maintain Compression etc.	**Skin** Treated	**Ulcer** Dressings applied	**Bandage** Combination applied	**Outcome** Reduction in Circumference of wound size or ankle size or wound healed	**App.** Date	**Assessor** Initials
	Right leg												
	Left Leg												

		Comprehensive	Reassessment	4wk									
		Comprehensive	Reassessment	8wk									
		Comprehensive	Reassessment	12wk									

***Referral to Surgical Services via GP when no improvement is recorded after 3/12 treatment**

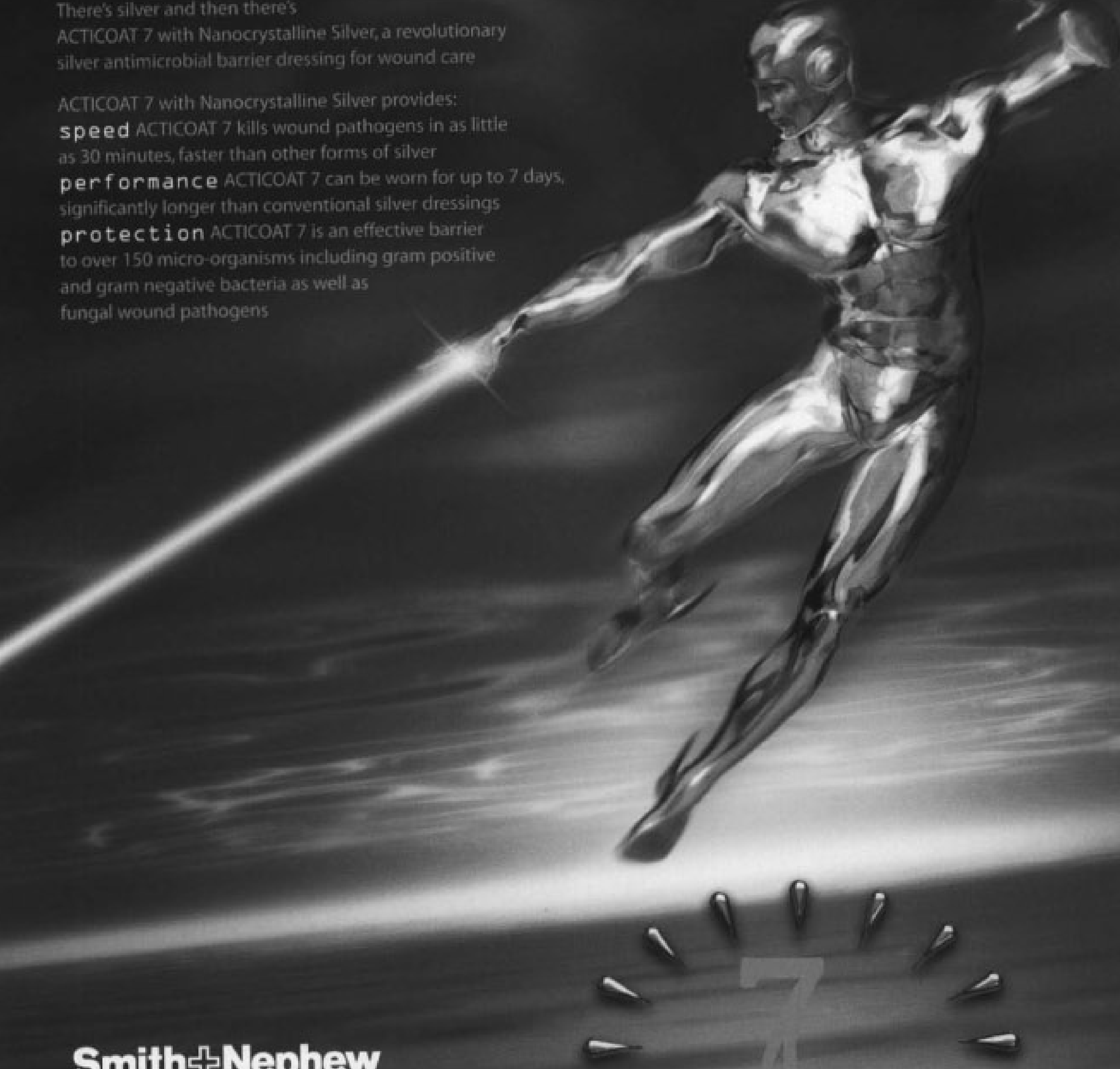
Adapted Dale J. Gibson B. Moffatt C. **Pauline Diamond PHN-General Practice Unit, North Western Health Board June 1996**

Zap bacteria

rapid destruction, sustained protection

BETTER SILVER
There's silver and then there's
ACTICOAT 7 with Nanocrystalline Silver, a revolutionary silver antimicrobial barrier dressing for wound care

ACTICOAT 7 with Nanocrystalline Silver provides:
speed ACTICOAT 7 kills wound pathogens in as little as 30 minutes, faster than other forms of silver
performance ACTICOAT 7 can be worn for up to 7 days, significantly longer than conventional silver dressings
protection ACTICOAT 7 is an effective barrier to over 150 micro-organisms including gram positive and gram negative bacteria as well as fungal wound pathogens



Smith+Nephew
First Choice in Wound Management

Smith & Nephew Limited,
Carraig Court, George's Avenue, Blackrock, Co. Dublin.
Tel: (01) 2170 444 Fax: (01) 2170 455
www.smith-nephew.com

6392/ACTICOAT7/ADVERT/GLOBAL/0801

NEW **Acticoat*** *with Nanocrystalline Silver†*

Because leg ulcers come *in all* shapes *and* sizes...

...*So do* PROFORE kits

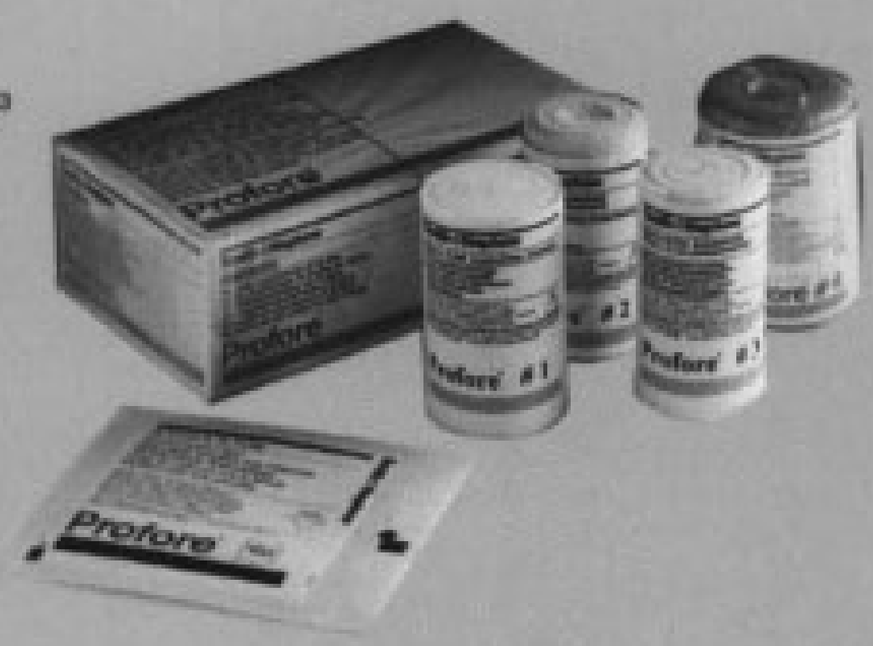
All shapes and sizes of leg ulcers mean that when you are dealing with venous leg ulcers and multi-layer compression bandaging, time taken in selecting the appropriate length of the right bandages can soon add up.

PROFORE takes all the guess work out of this process for you by providing five different kits, made up of the correct components to suit a variety of leg circumferences.

So whether your patients ankle circumferences vary from 18cm to 30cm plus, you know that by prescribing a PROFORE kit, you'll be making a safe choice. As well as the optimum range of kits, PROFORE gives you:

- The most clinical evidence
- A proven healing rate of 79% in 12 weeks[2]
- 65% savings in total costs over existing treatment regimes[2]
- 35% reduction in annual cost of materials[2]
- 59% saving on total nursing time[3]

Profore*



Proven clinical & cost effectiveness

Refs:

1. Moffat CJ, Franks PJ, McCollum CN et al. Randomised trial comparing two four layer bandage systems in the treatment of venous leg ulceration. Poster presented at Symposium on Advanced Wound Care and Medical Research Forum on Wound Repair, New Orleans, April 1997.
2. Simon DA, Freak L, Kinsella A, Walsh J, Lane C, Groarke L, McCollum C. Community Leg Ulcer Clinics: A comparative study in two health authorities. BMJ Vol 312, 1996.
3. McCollum C, Data on file, Smith & Nephew.

Smith+Nephew
First Choice in Wound Management

Smith & Nephew Limited,
Carraig Court, George's Avenue, Blackrock, Co. Dublin.
Tel: (01) 2170 444 Fax: (01) 2170 455

ISBN 0 - 9542474 - 0 - X

Smith+Nephew

First Choice in Wound Management

Smith & Nephew Limited,
Carraig Court, George's Avenue, Blackrock, Co. Dublin.
Tel: (01) 2170 444 Fax: (01) 2170 455