Case study

Mr P is a 72-year old male whose type 2 diabetes was diagnosed in 1999. He has a history of venous leg ulceration with recurrent bilateral ulcers over a period of 12 years. These were treated with four-layer compression therapy.

In 2000 he developed ulceration of both feet, with four toes on the right foot and the hallux on the left foot affected (Figures 1 and 2). Treatment for the foot ulcerations proved difficult with the application of a variety of different dressings (including iodine-based dressings, hydrocolloids, hydrogels, honey-based dressings and foam dressings) failing to achieve wound closure. The patient also had recurrent soft tissue infections with Pseudomonas aeruginosa proving to be a particular problem. This resulted in repeated oral antibiotic courses and a six-week intravenous antibiotic course at home.

Whenever the compression therapy for venous ulceration was suspended (usually for social reasons), it was noted that the foot ulceration improved, while the venous ulceration deteriorated.

Clinical dilemma

These findings left the multidisciplinary team with a clinical dilemma. The compression therapy that was essential venous ulcer treatment aggravated his recurrent diabetic foot ulcers. Following a study day involving lymphoedema nurses at the local hospital it was suggested that lymphoedema bandaging up to and including the toes should be tried. It was felt that the venous compression was waterlogging the tissues around the toes preventing healing and providing an environment where infection could flourish.

A literature review revealed that there was limited work done in this area but Todd et al (2003) reported an observational study of 89 patients who had lymphoedema bandaging treatment for either unilateral or bilateral leg swelling, including 21 who had venous ulceration. None of the patients developed toe ulceration, despite digital ulceration/gangrene being an anticipated outcome.

Assessment and treatment

Neurological assessment with a 10g monofilament and 128mgHz tuning fork revealed no loss of protective sensation. Ankle brachial pressure indices, toe brachial pressure indices and toe waveforms were all recorded. This provided objective evidence for both the gross circulation to the foot and also the extreme peripheral circulation. The reason for exhaustive vascular assessment was:

1. End artery compression may lead to tissue necrosis
2. Vascular calcification is more common in diabetes (Goss et al, 1991).

Fortunately the results of the non-invasive vascular studies were within normal limits.

As the initial neurological and vascular assessment indicated the patient was suitable for lymphoedema bandaging, he was booked into the weekly multidisciplinary foot ulcer clinic that was attended by staff from the various disciplines involved in diabetic foot ulcer management. This includes podiatrists, diabetes specialist nurses, diabetologists and microbiologists. Input is also provided by other disciplines such as district nursing and tissue viability nursing when required.

On this occasion, the lymphoedema nurse came to provide an expert opinion. A consensus decision was reached that lymphoedema bandaging should be tried, after all the potential contraindications were taken into account (infection, deep vein thrombosis,
cardiac therapy, untreated hypertension and proven arterial insufficiency (Williams and Keller, 2005).

**Results**
As can be seen in Figures 3 and 4, the lymphoedema bandaging resulted in healing of all the long-standing digital ulcers within 12 weeks.

**Discussion**
This experience suggests that in situations where four-layer compression bandaging is needed to control venous ulceration but exacerbates diabetic foot ulceration, lymphoedema bandaging may be a safe and effective approach. However, all types of compression bandaging are contraindicated by significant peripheral vascular disease.


