NEUROPATHIC ULCERS OF THE FOOT

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We report a prospective study of the causes and treatment of 26 long-standing neuropathic ulcers of the foot in 21 patients. The most important causal factor, well illustrated by pressure studies, was the presence of a dynamic or static deformity leading to local areas of peak pressure on insensitive skin. All but one of the 26 ulcers had healed after an average of 10 weeks of treatment in a light, skin-tight plaster cast, with the prohibition of weight-bearing.

Recurrent ulceration was prevented in all but one foot by early operation to correct the causative deformity; this was performed after the ulcer had healed and before allowing weight-bearing on the limb. Pressure studies after operation confirmed that pressure points had been relieved.

Ulcers have been a scourge to insensitive feet since Biblical times. They are usually described as trophic ulcers; this implies some abnormality of nutrition of the skin and underlying tissues. Neuropathic ulcer is a more apt term for those ulcers associated with spina bifida, spinal cord lesions, peripheral nerve injuries and leprosy. Diabetic ulceration has elements of both neuropathic and trophic causes, the latter associated with vascular changes.

Loss of sensation, even over the whole of the sole of the foot, is quite compatible with full weight-bearing and the maintenance of skin integrity. It is the abnormal distribution of load, leading to local areas of peak pressure that is the cause of many neuropathic ulcers. Such ulcers are easily and rapidly produced, but are usually slow to heal, causing concern to patient and physician. When treatment is incorrect or inappropriate, the ulcers seem intractable. If low-grade infection spreads to underlying bone, amputation may be the outcome.

This paper reports an investigation of the processes which cause ulceration in feet with sensory loss and proposes a method of treatment based on these causes.

MATERIAL
From 1981 to 1983, 21 patients with 26 long-standing neuropathic foot ulcers entered a prospective trial. The 10 men and boys and 11 girls aged from 3 to 24 years (mean 15 years) were followed-up after their ulcers had healed for a period ranging from 12 to 30 months.

Twenty patients had a lumbosacral myelomeningocele; three of these also had an associated lipoma of the spinal cord and one had an associated haemangioma. The other patient had incomplete paraplegia from a low spinal cord injury. All could walk independently, although some needed calipers or surgical footwear. All had diminished sensation over most of the sole of the foot and complete loss of sensation in the area of the ulcer and for 5 cm around its margin.

Most of the ulcers had been present for many months though two had appeared only four months previously. The maximum duration was 96 months and the mean 31 months. The largest ulcer had a diameter of 3.8 cm and the smallest 1 cm (mean 2 cm). Five ulcers were classified as deep (Figs 1 to 4), reaching almost to bone, 13 were superficial (Figs 5 and 6) involving only dermis and 8 were intermediate with their bases in the subdermal tissues. Of the 26 ulcers, 15 were on the heel (58%) and the remainder in five other regions of the foot (Fig. 7).

All the ulcers had failed to heal with medical management, including topical applications, dressings, and bed rest. Some had been curedt and a few had had plastic operations.

METHOD
Assessment. The patients were examined and note made especially of their weight, mobility, and gait. Any lower limb deformity was noted as were the peripheral circulation and neurological state. Detailed tests were made of sensation to pin-prick and light touch and of proprioception. The power of all muscles acting on the foot and ankle was assessed and any imbalance noted. Orthoses, and surgical or other footwear, were examined. Clinical photographs recorded stance, ankle and foot deformity and the size of each ulcer against a scale.
The progress towards healing of a deep ulcer at the heel shown at three-week intervals during treatment in a light, skin-tight plaster cast.

The progress towards healing of a superficial ulcer during treatment.

The sites, and number at each site, of 26 foot ulcers:
- Heel: 15
- Base of fifth metatarsal: 4
- Tip of great toe: 3
- Head of first metatarsal: 2
- Head of fifth metatarsal: 1
- Medial side of foot: 1

Static foot-pressure recordings were made of 16 of the ulcerated feet before treatment, and these were repeated after corrective operation in seven cases (Figs 8 and 9). Accurate, reproducible recordings of footprints in a calibrated colour code were made using the apparatus developed by Betts and Duckworth (1978).

Before the application of the plaster cast, a swab was taken for microscopic examination, and for the culture of organisms and determination of their sensitivities to a standard range of antibiotics. Inflammation extending more than a few centimetres from the edge of the ulcer was treated by elevation of the limb for a few days and a course of appropriate antibiotics before the cast was applied.

**Plaster-cast treatment.** The ulcer was cleaned with an antiseptic solution and a tulle gras dressing laid over it. A light, skin-tight plaster cast was applied. Wool padding was used only over the malleoli and near the upper end of the fibula; no padding was used on or around the ulcer. For patients with hairy legs, a close-fitting thin cotton stocking was allowed between skin and plaster. The edges of the plaster cast were carefully finished so as not to rub the skin, especially at the toes. Only three plaster bandages of 10 cm width were needed for the average below-knee cast.
Arrangements were made so that the patient, either in or out of hospital, need take no weight on the limb. This depended on home circumstances and the patient's reliability; some patients could not be trusted to keep weight off the plaster, and if there was any doubt, they were kept under observation in hospital.

At three-week intervals the patient was seen for a change of plaster; the ulcer was inspected and photographed (Figs 1 to 6). If seepage and soiling made the cast and the patient socially unacceptable then earlier replacement was allowed. Treatment was maintained at least until the ulcer had healed and often for two or three weeks more to allow the depths of the scar to consolidate and also to allow arrangements to be made for operative correction of deformity when this was indicated.

The clinical examination, and study of footwear and foot-pressure recordings, allowed a decision to be made on the likely cause of each ulcer. Weight-bearing on a foot with a healed ulcer was absolutely forbidden until the cause had been treated either by new footwear or, more often, by operation for deformity. After the correction of deformity, footwear was made to fit the new shape of the foot.

Serial casts were used to treat 22 ulcers. Three were not treated in this way because the ulcers were on the toes, and one ulcer was eventually treated by amputation, carried out for persistent active sepsis in deep soft tissues with osteomyelitis of the calcaneum, indications that conservative treatment was unlikely to succeed. This patient had previously had a below-knee amputation of the opposite limb for a similar problem. Skin flaps incorporating innervated skin were used, a stump with some sensibility was obtained and the result was successful.

**Operations.** Corrective surgery for deformity was performed on 21 feet after the ulcers had healed (Table 1). Mobile deformity in two patients was corrected by rebalancing muscle power without bony correction (Figs 10 to 12). Bone operations to correct rigid deformity were needed for the other 19 feet (Figs 13 to 15). Five feet did not need operations: in two the ulcers were bedsores which developed after major spinal surgery (Figs 5 and 6), another two were due to ill-fitting footwear and one was due to an injury from a carpet nail.

**Table 1.** The cause, site and number at each site of the 26 ulcers in insensitive skin, together with the operations used to correct deformity

<table>
<thead>
<tr>
<th>Cause of ulcer</th>
<th>Ulcers</th>
<th>Treatment after the ulcer had healed</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcaneus deformity</td>
<td>Heel</td>
<td>Supramalleolar osteotomy</td>
<td>4</td>
</tr>
<tr>
<td>Calcaneocavus deformity</td>
<td>Heel</td>
<td>Elmslie triple arthrodesis</td>
<td>2</td>
</tr>
<tr>
<td>Calcaneovalgus deformity</td>
<td>Heel</td>
<td>Ankle arthrodesis</td>
<td>1</td>
</tr>
<tr>
<td>Valgus deformity of heel</td>
<td>Heel</td>
<td>Triple arthrodesis</td>
<td>3</td>
</tr>
<tr>
<td>Bedsore after spinal operation</td>
<td>Heel</td>
<td>Nil</td>
<td></td>
</tr>
<tr>
<td>Faulty footwear</td>
<td>Heel</td>
<td>Nil—new footwear</td>
<td></td>
</tr>
<tr>
<td>Varus deformity of heel and forefoot</td>
<td>Base of fifth metatarsal</td>
<td>Tibialis posterior transfer to cuboid</td>
<td>1</td>
</tr>
<tr>
<td>Drop foot with varus deformity of forefoot</td>
<td>Dorolateral aspect of base of fifth metatarsal</td>
<td>Supramalleolar osteotomy</td>
<td>2</td>
</tr>
<tr>
<td>Equinovarus deformity</td>
<td>Head of fifth metatarsal</td>
<td>Lambrinudi triple arthrodesis</td>
<td>1</td>
</tr>
<tr>
<td>Flexion deformity of interphalangeal joint of great toe</td>
<td>Tip of great toe</td>
<td>Tibialis posterior transfer to cuboid</td>
<td>1</td>
</tr>
<tr>
<td>Equinovalgus deformity of heel and valgus deformity of forefoot</td>
<td>Head of first metatarsal</td>
<td>Interphalangeal arthrodesis of great toe</td>
<td>3</td>
</tr>
<tr>
<td>Drop foot with valgus heel and forefoot deformity</td>
<td>Head of first metatarsal</td>
<td>Lambrinudi triple arthrodesis</td>
<td>1</td>
</tr>
<tr>
<td>Injury from a carpet nail in a diabetic patient with spina bifida</td>
<td>Medial side of instep</td>
<td>Nil</td>
<td></td>
</tr>
</tbody>
</table>
RESULTS

Healing. All the ulcers treated by serial casts healed to complete skin cover in from 3 to 18 weeks (average 10 weeks). The rate of healing measured by the diameter of the ulcer was approximately linear (Fig. 16). The deeper ulcers took longer to heal; after nine weeks, 70% of superficial ulcers had healed but none of the deep ones.

Most patients had their cast treatment at home, using either crutches or a wheel-chair with an extension board for the limb. Five patients needed hospital admission during this part of their treatment. Two were unable to avoid weight-bearing because of social circumstances, two were unreliable children and one was admitted for a concomitant medical problem. The average period of admission was 4.4 weeks, ranging from 2 to 8 weeks.

Pressure studies. For 20 of the ulcers, clinical appearances indicated that a deformity was giving rise to an area of high pressure and thus an ulcer (Figs 10 to 15). Static foot-pressure studies in 16 of these confirmed that in 11 the ulcerated area was at a point of abnormally high pressure (Fig. 8). Clinical assessment of the gait, the deformity and the ulcer site suggested that dynamic pressure studies (Betts et al. 1980) would have shown abnormally high peak pressures in the other five cases. Dynamic studies were, unfortunately, not available for this present study. In one case a posteriorly placed ulcer of the heel was out of contact with the floor when the patient was standing, but was the main point of contact at heel strike during walking.

Pressure studies in seven cases after corrective operations showed that the abnormal distribution of pressure had been corrected in six (Fig. 9).

Maintenance of healing. Twenty feet (95%) remained healed after a follow-up ranging from 12 to 30 months (mean 20 months). One ulcer recurred. This ulcer of the heel was due to a slight calcaneus deformity, aggravated by heterotopic bone around a partially removed calcaneal spur. A supramalleolar osteotomy, combined with removal of the bony spur, has since been performed.

DISCUSSION

The use of serial light skin-tight plaster casts in the treatment of neuropathic ulcers of the feet is safe, effective, quick and inexpensive. It is unfortunate that plaster casts, especially if they are unpadded and close-fitting, are associated by many orthopaedic surgeons with the production rather than the cure of sores. Provided that any existing cellulitis has been treated by elevation and appropriate antibiotics before application of a cast, there is no danger of complications from its tightness. In many cases, the reduction of swelling after application of the cast was a reason for an earlier change of plaster, so as to avoid possible shearing movement and consequent friction, which is one factor in the persistence of ulceration (Bauman, Girling and Brand 1963).
Close-fitting casts have been used successfully for many years in the treatment of lepromatous ulcers (Khan 1939; Andersen 1961; Ross 1962; Brand 1979). These casts were designed to allow weight-bearing, since patients often had to walk some distance to attend a clinic. More recently, weight-bearing and non-weight-bearing casts made of Scotchcast, with a window round the area of the ulcer, have been used to good effect for foot ulcers in diabetic patients (Burden et al. 1983). Our experience suggests that, for neuropathic ulcers when there is foot deformity, complete relief from weight-bearing is essential to achieve rapid healing.

Brown-Séquard, in 1853, wrote that the absence of protective sensation was a major cause of foot ulceration and that complete rest would produce healing. This has been confirmed by many authors, who have described other contributory factors (Khan 1939; Andersen 1961; Ross 1962; Sabato et al. 1982). Brand (1979) listed five physical causes of neuropathic ulcer (Table II). All except the thermal causes concern abnormal pressures and stresses. It is probable that a skin-tight cast, by distributing pressure evenly, allows a neuropathic ulcer to heal. Patients with spina bifida are liable to become overweight in adolescence though their feet remain relatively small. These factors, combined with deformity and an abnormal gait, lead to local pressure and the late development of ulceration which did not occur earlier in childhood.

Failure to correct dynamic or fixed foot deformity, and thus the abnormal pressures associated with it, will inevitably lead to recurrence of an ulcer, however well it has healed in plaster. It is best to correct deformity before an ulcer has developed, and we normally try to do this. If muscle balance has been achieved and fixed deformity has been corrected, the feet of children with spina bifida who can walk have not given problems, although even a minor deformity may sometimes lead to ulceration after the weight increases of adolescence. Pressure studies on the feet have been useful to augment clinical assessment and to predict those which will be at risk; they also indicate the success of corrective operations in relieving local high pressure.

CONCLUSIONS

The prevention of deformity in insensitive feet is the best means of avoiding neuropathic ulcers. Clinical assessment of feet at risk can be helped by static and dynamic pressure studies.

The treatment of choice for an established ulcer, however large and unpleasant, is the application of serial, light skin-tight plaster casts with relief from weight-bearing. Healing of the ulcer is only the preliminary treatment; the cause of the ulcer must then be defined and corrected if recurrence is to be prevented. Management by these methods assures a high rate of success in a condition which is resistant to most other forms of treatment.

Table II. Physical factors leading to ulceration in insensitive feet (after Brand 1979)

1. Continuous high pressure causing necrosis from lack of blood supply
2. Concentrated high pressure causing cutting or crushing by mechanical violence
3. Repetitive moderate mechanical stress causing inflammation and autolysis
4. Pressure on infected tissues
5. Heat or cold causing burning or frostbite

REFERENCES