UNIVERSITY COLLEGE SALFORD
BSc (Hons) PODIATRY
PROJECT

Salford College of Technology

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THE ROLE OF THE PODIATRIST IN THE PREVENTION AND TREATMENT OF DIABETIC NEUROPATHIC ULCERATION

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Abstract

This review opens with information on the classification of neuropathy and a brief history of the knowledge of the diabetic foot. This is followed by the aetiology of diabetic neuropathy, Charcot joints and neuropathic ulcers. Practical methods for the measurement of plantar pressures and neuropathy are described.

Thirteen pressure relief methods used in the prevention and treatment of diabetic plantar neuropathic ulcers are reviewed. These are discussed regarding their advantages and disadvantages in terms of expense and maintenance of patient mobility. The need for short and long term treatment plans becomes obvious. Diverse methods of pressure relief were found to be appropriate in differing situations and were not mutually exclusive.

The role of the podiatrist in the care of diabetics was explored, with particular reference to diagnosing neuropathy and excessive plantar pressures and prescribing pressure relief methods. The importance of the podiatrist's involvement is the prevention of neuropathic ulceration or timely intervention to prevent the all too frequent progression to osteomyelitis and amputation.

Research into pressure relief as a method of prevention and treatment of diabetic neuropathic ulcers can also benefit people with other pathologies caused by deformities or biomechanical abnormalities.

1 INTRODUCTION AND LITERATURE REVIEW

Two seminar days were given to introduce the care of the "High Risk Patient". From these a strong interest developed in the available methods of pressure relief and their use in the treatment of diabetic neuropathic ulcers.

1.1. What is neuropathy?

Neuropathy is a term used to describe any disease of the peripheral nerves, usually causing weakness or numbness. In a *mononeuropathy* only one nerve is affected so that the spread of symptoms is directly related to the distribution of that nerve. In a *polyneuropathy* many or all of the nerves are affected and the symptoms are most severe at the extremities of the limbs.

Neuropathy is characterised by nerve fibre loss, segmental demyelination and decreased nerve velocities. Plantar neuropathic ulcers occur on the plantar surface of insensitive feet. According to Delbridge *et al* (1985), the first reference to a neuropathic ulcer is made by Mott in 1818 when he described

> "A round ulcer in the sole of the foot surrounded by a remarkably tough hardening of thick cuticle..... characterised by a great degree of insensitivity".

1.2. Classifications of neuropathy

A full table showing the classifications of neuropathy appears in Appendix 1. It can be seen from the

highlighting that diabetes mellitus is responsible for many different forms of neuropathy.

1.3. Diabetes Mellitus

Diabetes mellitus is a state of persistent hyperglycaemia due to the deficiency or reduced effectiveness of insulin. Primary diabetes occurs in two forms: the first is insulin dependent diabetes (IDDM), predominately of juvenile onset, the predisposition being HLA linked. The second is non insulin dependent diabetes (NIDDM), which is not HLA linked and tends to be of mature onset. Secondary diabetes occurs in a minority of cases as a result of some other disease (e.g. acromegaly, carcinoma pancreas), or as the result of the treatment of another condition (e.g. cortico-steroid therapy).

1.4. Knowledge of the diabetic foot

Although Mott (1818) gave a very good description of a neuropathic ulcer, it was not until the second half of this century that opinion veered away from the long held belief that diabetic gangrene was the only serious foot problem for these patients. During the second world war Robin Lawrence, a diabetic surgeon, could no longer operate since losing the sight of one eye following infection. He asked K C McKeown to perform a revolutionary wedge resection of a gangrenous toe and the operation proved to be a great success. Lawrence also began to understand the etiology of the neuropathic ulcer.

He believed that the thickened callous and insensitive skin of the foot in diabetics combined with the descent of the metatarsal heads in the cavus foot type to produce pressure necrosis of the tissue between. He also noticed that the dry skin of the diabetic foot often led to fissuring with the attendant risk of infection (McKeown, 1992).

1.5. Diabetic Neuropathy

> "Although both major types of diabetes are affected by both microangiopathy and macroangiopathy, the IDDM patient of early onset is particularly susceptible to microvascular problems." (Gill, 1991)

Microangiopathy manifests itself in the form of complications: retinopathy, nephropathy and neuropathy. There is some correlation between retinopathy and neuropathy (the foot/eye syndrome).

Poor metabolic control is one factor which may be implicated in the commencement and progress of diabetic neuropathy. The effects of an increased blood glucose concentration on diabetic nerve have been studied mainly in diabetic rats or with nerve cells in vitro. According to Greene *et al* (1989), four hypotheses have emerged from these studies. They report those by a)Greene *et al*, b) Low *et al* (1985), c) Vlassara *et al* (1982) and d) Sima *et al* (1983):

a) <u>The sorbitol-accumulation/myo-inositol depletion hypothesis</u>: Slowing of nerve conduction in myelinated

nerve fibres in acutely diabetic animals has been attributed to a set of interrelated metabolic abnormalities in diabetic peripheral nerve, resulting from hyperglycaemia and involving sorbitol, myo-inositol and (Na,K)-ATPase regulation.

b) The hypoxia/ischaemia hypothesis: This demonstrated that the exposure of normal rats to chronic hypoxia slows conduction velocity in the specialised caudal nerve. Low also exposed normal and chronically diabetic rats to hyperbaric oxygen and showed that some of the changes (metabolic and functional) to the peripheral nervous system might be interpreted as selective partial reversal of some of the elements produced by the diabetic state.

c) The non-enzymatic glycosylation hypothesis: In response to increased ambient glucose concentration, an additional metabolic abnormality in diabetic nerve is non-enzymatic with possible alteration in protein function. Glycosylation of peripheral nerve tubulin may have consequences on both fast and slow axonal transport; also on the physical characteristics of the axon cylinder.

d) The insulin/neurotrophism/axonal-transport hypothesis: In advanced diabetes in rodents the dominant structural change consists of axonal atrophy and degeneration. The mechanisms underlying the characteristic axonal atrophy in experimental diabetes are not well understood. They most

likely involve several factors, some of which could reflect decreased neuronotrophism.

Metabolic control in the insulin dependent diabetic is achieved through a combination of insulin, diet and monitoring. It is worth mentioning that drawing up insulin for injection and reading BM-stix results or testing urine require reasonable eyesight. The author has nursed an elderly lady who was admitted in hyperglycaemic coma with a blood sugar too high to be normally compatible with life. She eventually recovered and it was discovered that she had been administering insulin and testing urine by guesswork as her sight had deteriorated markedly.

There have been several long term prospective studies into the effects of strict metabolic control on the progress of complications in IDDM patients. Three of these have included the progress of neuropathy. In the Oxford-Aylesbury study (Holman *et al*,1983), 74 patients with background retinopathy were randomly assigned to two groups: the first to continue with normal diabetic care; the second to have a more intensive programme of care, using long acting insulin for basal care and fast acting insulin at mealtimes. After two years there was a significant deterioration in the vibration sensory threshold of the members of the 'normal care' group and a significant improvement in that of the members of the 'intensive therapy' group.

In the Steno 1 study (Lauritzen *et al*,1985), 30 IDDM patients with moderate background retinopathy were split

into two groups, one to be treated with normal insulin therapy and the other to use a continuous infusion pump. After two years there was no significant change in vibration sense in either of the treatment groups.

In the Oslo study (Dahl-Jorgensen *et al*,1986), forty-five IDDM patients were randomly assigned to three groups:

1. Pump insulin therapy
2. Multiple insulin therapy
3. Conventional insulin therapy.

After two years motor nerve conduction velocity tests revealed no change in groups 2 or 3 but a significant improvement in the pump treated group.

> "Poor dietary compliance may be as important a cause of unsatisfactory metabolic control in some IDDM patients" (McCulloch 1983).

Dietary histories are sadly unreliable and the patient's energy requirement is better calculated according to sex, age, height and level of physical activity. It is very important that as circumstances change, energy requirements and the dietary prescription should be reviewed. Needless to say, it is important for the progress of NIDDM patients to be monitored in terms of metabolic control. Deterioration should not go unnoticed and drugs should be adjusted.

Another feature of poor metabolic control is susceptibility to infection and delay in healing. The base of a neuropathic ulcer should normally be pink but it is clearly an ideal medium for the culture and

proliferation of bacteria, usually gram positive in nature. Purulent drainage and possibly an offensive odour are signs. The ulcer will deepen and there is a risk of bony involvement in the form of osteomyelitis. Organisms frequently implicated are: *Staphylococcus aureus*, group B streptococci, *Peptococcus*, *Peptostreptococcus* and *B.fragilis* (Joseph 1990).

The neuropathic foot is generally a good colour with strong pedal pulses. This may be due to arterio-venous shunting and the tissues can therefore be hypoxic. Smoking also decreases oxygenation; these factors delay healing and encourage anaerobic proliferation.

1.6. Natural History of Diabetic Neuropathy

Diabetic neuropathy can go unnoticed for some time. Early loss of vibration sense is not something which is discovered unless regular testing takes place. Different forms of testing will be mentioned in the next chapter.

The first person to notice the function of the small muscles of the foot was Lambrinudi, Senior Orthopaedic Surgeon at Guy's Hospital at the outbreak of the second world war. He was interested in the large numbers of recruits with foot problems and was the first to describe according to foot function rather than anatomical description. Lambrinudi delivered a lecture at the Royal Society of Medicine in which he stressed that in addition to the action of the long flexors and extensors, the interossei helped to maintain the normal alignment of the

metatarso-phalangeal joint, while the lumbricals maintained the normal extension mechanism at the inter-phalangeal joints (McKeown, 1992). An early aspect of diabetic neuropathy can be the denervation and atrophy of these intrinsic muscles. As a result, the metatarsophalangeal joints become hyperextended and the interphalangeal joints are flexed, eventually producing the claw toe deformity. The selection of the intrinsic muscles as the first to suffer motor neuropathy is no accident; it is a simple function of the length of the nerves involved. The cavus foot type with clawed toes is also seen after poliomyelitis and other neurological diseases such as Charcot-Marie-Tooth disease.

A further problem caused by claw toes is that the specialised fat pad which protects the metatarsal heads is pulled forward away from its working position. The function of this fibro-fatty pad is to dissipate the stress away from the metatarsal heads and so absorb shock. When the bones are deprived of this protection they suffer many times the shock with every step. According to Alexander *et al* (1990), Ctercteko *et al* showed that the foot without intrinsic muscle function failed to transmit the normal amount of weight through the toes. This led to abnormally high pressure being transferred through the metatarsal heads. Boulton *et al* (1983) performed a similar study in which they found that the neuropathic patient was seven times more likely to have abnormally high pressure transferred through the metatarsal heads.

Sensory neuropathy occurs in two main forms: chronic painful neuropathy and symmetrical sensory neuropathy. Benbow *et al* (1992) found that 7.5% of 962 patients attending an adult diabetic clinic had symptoms of chronic neuropathic pain. Onset is usually insidious with a variety of symptoms including numbness, burning, tingling, hyperparasthesia and stabbing or shooting pains typically affecting the lower limbs. Sadly these symptoms may be worse at night.

Distal sensory polyneuropathy follows a "glove and stocking" distribution, with the longest fibres being first affected. These two forms of neuropathy seldom co-exist.

Autonomic neuropathy is frequently associated with sensory neuropathy - with quite devastating effects. Laing and Klenerman (1991) state that autonomic neuropathy alters the normal autoregulation of the microcirculation in the following way: loss of sympathetic tone leads to increased peripheral blood flow and loss of postural vasoconstriction. This in turn causes increased pressure in the capillaries and basement membrane thickening. The outcome is arteriovenous shunting.

Another aspect of autonomic neuropathy is that sweating is reduced, leading to dry plantar skin and increased incidence of fissuring. Proprioception is also reduced or absent in the feet so that the patient has no sense of the position of the joints.

1.7. Aetiology of Charcot's Joints

Charcot's joints develop as a result of mixed sensory and autonomic neuropathy. According to Gventner (1987), J K Mitchell first made the connection between a neural lesion of the spinal cord and problems in peripheral joints in 1831. Tabes dorsalis was the most common cause of this type of arthropathy and it was this condition which led J M Charcot to describe the neuropathic joint thirty-five years later. It was actually W R Jordan in the 1930's who first described the same changes in diabetics.

Initially with the lack of proprioception the joints of ankle and foot are subjected to extreme ranges of motion without any pain signals to warn the subject of consequent damage. Joint capsules and ligaments stretch and fail to prevent subluxation. Arteriovenous shunting and the greatly increased blood flow through the area causes demineralisation and rarefaction of the bone. The inability of the patient to perceive pain and trauma causes damage in the form of microfractures at the joint surface (Stess & Hetherington, 1990). Further weightbearing leads to dislocation and osteochondral fragmentation. The normal inflammatory response produces swelling and hyperaemia, which unheeded leads to further instability and reabsorption. Left untreated, Charcot's joints continue with this viscious circle of fragmentation, dislocation and deformity. Frequently the

process begins with an unknown trauma such as ankle fracture. According to Edmonds (1986),

> "In the Joslin clinic series of 101 cases of Charcot joints, 28 had evidence of fracture."

A great deal of damage can occur when weightbearing continues on a traumatised joint. However, it is also possible for the repeated microtrauma caused simply by walking to initialise similar joint damage.

According to Stess & Hetherington (1990), Forjacs in 1982 classified the Charcot changes in three stages:

Stage 1: The initiation of symptoms occur, there is subluxation of the joints, osteoporosis occurs, cortical defects become obvious.

Stage 2: The deformity progresses with osteolysis, fracture and periosteal elevation or new superosteal bone formation.

Stage 3: First healing stage, where reconstruction occurs with the subsiding of swelling; reorganization of cortical defects and ankylosis of the joints may occur.

1.8. Testing for diabetic neuropathy

The podiatrist should be alert to changes in the sensation discrimination of the diabetic patient over a period of time. According to Dent *et al* (1992), Sosenko *et al* states:

> "In assessing patients for risk of foot ulceration, abnormality of large fibre function appears to be more strongly related to risk of

ulceration than abnormality of small fibre function."

Simple vibration testing with an ordinary tuning fork is at best a crude test of large fibre function with no way of detecting deterioration; simply whether the sense is present or absent. The vibration perception threshold can be measured using the "biothesiometer" (Bio Medical Instrument Co. Ohio, USA) and the measurements recorded for future comparison provided the calibration is also noted and adhered to. This is a hand held instrument with a stylus driven at twice the mains frequency (100Hz). The amplitude of vibration is proportional to the square of the voltage applied and can be varied over a range from 0 to 50V. Even this method will not detect early neuropathy and many podiatrists will not be able to afford the machine.

Thermal discrimination tests, while a useful guide to the function of small myelinated and unmyelinated afferent nerves, are difficult and time consuming to perform. There is a "thermoaesthesiometer" (VU Hospital, Amsterdam, Netherlands) which consists of two probes which can be rapidly heated or cooled to different temperatures. The patient is asked to identify the warmer probe and the temperature difference can be set to anything between 0.1 and 20 degrees centigrade. It is possible to use hot or cold eg test tubes but it is difficult to elicit much information this way.

An inexpensive device for the testing of pressure sense is the Semmes-Weinstein Pressure Aesthesiometer

which consists of a series of 17 nylon filaments, each of identical length but with differing diameters. The pressure applied depends on the diameter of the fibre: the pressure required to make it bend. Testing begins with the largest diameter fibre and progresses down to the first fibre which cannot be felt. This method can perform as well as vibration testing and is a more reliable method than temperature discrimination for predicting ulceration risk.

Nerve conduction studies can be performed using needle electrodes to measure the velocity of conduction in myelinated nerves. Conduction will be slowed where demyelination has occurred.

Electromyography is also a test which uses needle electrodes and is the most sensitive test for motor neuropathy.

The manifestations of autonomic neuropathy are not restricted to the lower limb. There are a number of cardiovascular tests for autonomic neuropathy. These include quiet supine rest; single deep breath; Valsalva manoevre (maintaining the pressure of 40mm Hg for 15 seconds by forced expiration into the tubing of a sphygmomanometer); max/min heart rate ratio after standing up; systolic fall after standing. It is necessary to compare the results to age adjusted normal values.

There is some evidence to suggest that diabetic autonomic neuropathy follows the pattern of distal symmetrical sensory neuropathy, the longest fibres being affected first (Ryder *et al*, 1992). As a result sweating

disorders in the feet may significantly precede any cardiovascular symptoms. It is therefore useful to test the feet for sweating disorders as an early test of autonomic neuropathy. The acetylcholine sweatspot test was developed for this reason. It makes use of the fact that acetylcholine injected intradermally causes sweating in nearby sweat glands when the nerve supply to these glands is intact.

1.9. Aetiology of diabetic neuropathic ulcers

As early as the latter part of the 19th century, Frederick Treves, the London Hospital surgeon and anatomist now famous for the rescue of John Merrick (The'Elephant Man'), recognised that ulceration could be caused by deformity of the foot causing changes in the pattern of weightbearing. He noted that this was particularly the case when "sensibility was disturbed". (Duckworth *et al*, 1985)

Since that time it has generally been accepted that neuropathic ulcers form at areas of excessive pressure or shear, or at the site of existing trauma which goes unrecognised because of the absence of protective pain signals. According to a review which combines the results of a Miami study with one from King's College Hospital and another from Manchester:

> "Neuropathy can be implicated as an aetological factor in 90% of over 600 episodes of diabetic ulceration" (Boulton, 1992).

Autonomic neuropathy is known to cause dry skin and is a primary cause of fissuring of the plantar surface, especially the heels. This is another typical starting point, both for ulceration and for infection.

There have been various studies which link high foot pressures with a history of ulceration but there has also been one prospective study in which patients with the combination of neuropathy and high foot pressures were followed up over two and a half years. 28% of this group ulcerated during the follow-up period (Veves *et al*, 1992). Callus which forms as a result of high pressures may further increase the risk of ulceration. Young *et al* (1992) have confirmed that chiropodial removal of callus significantly reduces foot pressure.

A study by J Bevans (1992) shows definite correlation between biomechanical abnormalities and plantar ulcer location. Twenty-eight feet with active or recently healed diabetic forefoot ulcers were assessed. Heel bisection angles were measured, both with the foot in subtalar joint neutral stance and in relaxed calcaneal stance, and the ulcer position was mapped. In every case biomechanical abnormalities were discovered, shown by abnormal positions in subtalar joint neutral and/or relaxed stance and quantified by compensatory pronation. There was a strong correlation ($r=0.8696$, $P<0.0001$) between relaxed calcaneal stance heel angles and metatarsal ulcer sites. Lateral ulcers were associated

with inverted heels and medial ulcers were associated with everted heels.

1.10. Measurement of Peak Pressures

At about the same time as Frederick Treves was working in London, in Paris efforts were being made to make dynamic recordings of plantar foot pressures. According to Alexander *et al* (1990), these first studies were performed by Carlet and Marey. They used shoes which had air chambers in the soles. In Carlet's experiments these were connected by pneumatic tubing to a central recording instrument while the subject walked around a specially constructed circular walkway. Marey took this equipment one stage further to make the recording instrument portable. Different researchers continued to explore and extend this kind of measurement but for many decades all attempts were to make special modifications to shoes. The main disadvantage was usually that the converted shoes were cumbersome and therefore altered gait.

An obvious extension of the modified shoe measuring system is the *in* shoe system. Many variations of this idea have been tried, with differing degrees of success. The basic principle is to instal very thin transducers either in an insole or adherent to the patient's feet. The obvious advantage of this system is that individual data can be amassed as to the pressures affecting the foot within the shoes which are actually worn regularly. In

practice, these systems have incurred development problems and are not yet widely available. Langer biomechanics markets a system which involves the patient wearing a data collection box strapped to the waist. The information is stored there, then loaded into a computer. This kind of system tends to be very expensive.

Other systems are very expensive to install and need building modifications eg. the force plate, which, as it is is very sensitive to vibration, has to be set in a concrete floor. Another difficulty associated with a fixed plate is that it is quite difficult for the patient to walk normally while trying to place one foot squarely on the plate. This makes it impossible to measure anything but bare feet while it is known that most people spend a majority of walking time in shoes. However, at least this is a dynamic measurement which will provide more appropriate information than a static measurement alone. Duckworth *et al* (1985) found that some peak pressure areas fail to show on static measurement because there is a natural tendency to alter stance to off-load painful areas. They also reported that dynamic measurements alone missed some risk areas which showed on static measurement. It can therefore be argued that it is wise to record both static and dynamic measurements in patients at risk of ulceration.

Floor mounted transducer system studies when analysed by computer can provide very interesting visual representations of the peak pressures (Appendix 2).

The simplest and cheapest way of recording excess plantar pressures uses the Harris Mat (a special rubber mat which is inked with a roller). An example of a Harris mat result can also be seen in Appendix 2. Other available systems include the Pedobarograph (Critical Light Reflection Technique), Polyurethane Photoelastic Plastic Sheet, Shear Sensitive Liquid Crystals and Piezoceramic transducers. The last of these when linked to computer analysis can create clear 'relief maps' of the pressure areas on the plantar surface of the foot (Cavanagh *et al*, 1985).

2 PRESSURE RELIEF IN THE TREATMENT AND PREVENTION OF DIABETIC PLANTAR NEUROPATHIC ULCERS

2.1. Footwear for the diabetic

New or ill fitting shoes are a risk to any diabetic but especially to those with peripheral neuropathy. Retracted toes begin to receive dorsal pressure and exert back pressure on the metatarsal heads in most normal shoes because even good shops which measure the length and width of a foot, have no facilities for measuring the depth. Shoes need to be fastened well so that the ankle is held secure: if the foot is allowed to move and slide around in the shoe, friction lesions will develop. Add to these naturally existing problems the need to take up depth with insoles and it becomes obvious that clinics must give good footwear advice. Some centres carry a list of local suppliers who stock unusual fittings: wide, narrow, deep, etc. As mentioned below, training shoes can be very suitable. They are usually deep, well laced, cushioned and fashionable.

2.2. Rest in bed

This is the method of choice where there is the complication of infection. Rest is in this case is combined with elevation and antibiotics, surgical removal of any bone which has become invaded by osteomyelitis and surgical drainage of any abcess. When infection is under control the ulcer can be treated as an uncomplicated ulcer. Complete rest for six weeks can heal a plantar

ulcer but the expense of prolonged hospitalisation coupled with the inconvenience and tedium for the patient no longer make this a method of choice. Lack of use also encourages osteoporosis, venous stasis and deep vein thrombosis.

2.3. Surgery

> "For chronic or recurrent neuropathic ulcerations of the forefoot without osteomyelitis or joint infection, metatarsal osteotomies can often be successfully employed." (Frykberg RG, 1991)

According to the theory of pressure relief from surgery, a plantarflexed metatarsal can be elevated or a long metatarsal can be shortened by the use of osteotomies. This principle can be applied to a Charcot deformity of the midfoot by excising the bony prominence (if the foot is well perfused).

2.4. Non weight-bearing plaster cast

This consists of a below knee Plaster of Paris cast without a walking base, used with crutches. The problem with allowing the patient home on crutches is the need for patient compliance. The author has met a diabetic gentleman who promised to remain completely non weightbearing and was seen by his chiropodist, later the same week, shopping in the high street without crutches. This unexpected meeting did not deter the patient; the same thing happened on several more occasions!

A prospective study of the causes and treatment of twenty-six long-standing neuropathic ulcers of the foot (Lang-Stevenson *et al*, 1985) found that all but one of the 26 ulcers had healed after an average of ten weeks of non-weightbearing treatment in a light, skin-tight plaster cast. These patients were from the Children's Hospital, Sheffield and their neuropathy was not diabetic. Nevertheless it demonstrates the potential success of this method if patient compliance is possible.

2.5. Total Contact Casting

According to the theory of total contact casting, peak pressures are attenuated when every part of the plantar surface of the foot shares the weightbearing. The first reference to treating plantar ulcers with a below knee walking cast appears to be by an orthopaedic surgeon named Milroy Paul who used them in the 1930's to treat patients with Hansen's disease out in Ceylon (Kominsky, 1991).

A controlled clinical trial (Mueller *et al* 1989) compared the management of diabetic ulcers by traditional dressing treatment (TDT), with that of total contact casting (TCC). There were 19 in the former group and 21 in the latter. There was no significant difference between the two groups with respect to age, sex, ratio of insulin-dependent diabetes mellitus, vascular status, size and duration of ulcer and lack of sensation ($p > 0.05$). Using the Semmes-Weinstein monofilament to judge sensation

all patients in both groups suffered some degree of sensory neuropathy, with two thirds of each group having *no* sensation in the hallux.

In the TCC group, the cast was applied on the first visit and the patients were instructed to restrict mobility to one third of their usual amount. The TDT group were instructed to avoid weightbearing on the affected foot and were prescribed dressing changes and accommodative footwear. An ulcer was considered healed if there was total skin closure and no drainage. If an ulcer failed to decrease in size in six weeks or if hospitalisation for infection was necessary this was declared "not healed". In the TCC group 19 of 21 ulcers healed in 42 +/-29 days; in the TDT group, 6 of 19 ulcers healed in 65 +/-29 days. The conclusion was that TCC is a successful method of treating diabetic plantar ulcers but requires careful application, close follow-up and patient compliance with scheduled appointments in order to minimise complications. For a statistical interpretation, see Appendix 4.

2.6. The Scotchcast Boot

The Scotchcast boot was originally developed as an alternative to total contact casting, allowing treatment to continue on an outpatient basis with the patients able

to remain mobile and work if possible. The savings to the hospitals with this form of care are obvious. The original boot was non-removable and constructed from resin bandages over a well fitting stockinette sleeve and ankle felt, 6mm felt sole with a precut window and a 15cm padding bandage. There was a canvas sandal with a notched heel for weightbearing and a window over the lesion for dressing changes and observation (Burden *et al* 1983&1989).

Many diabetic centres use a removable variation of this boot with the added advantage of comfort in bed at night and a slightly more acceptable appearance when worn in a dressing sandal such as the 'Darby Cast Sandal' (Appendix 3).

2.7. Total Contact Inlays

These are used in the treatment of neuropathic ulcers in the diabetic centre at Hope Hospital, Salford. Initially an impression of the patient's foot is made, using casting boxes for the negative impression and Plaster of Paris for the positive. A cast sandal with 7mm 'Poron' cushioning insoles is fitted and used while total contact inlays are being manufactured using medium density E.V.A. lined with 6mm moulded 'Plastazote'. The inlays are then machined with bevelled edges and a flat base. A removeable walking cast is moulded around the inlay, using 7mm semi-compressed felt and 'X-Lite' thermoplastic, with 'Fleecy Webb' trimmings and 'Velcro' fastenings. The

walking cast is fitted with a heel or fitted into a cast sandal.

The patient is given instructions to:

a) Slow down the normal walking pace.

b) Wear the walking cast *at all times* except: when in bed; with the foot resting on a stool; whilst redressing the foot.

c) Check the foot and leg regularly for any unusual redness, colour change or breaks in the skin. If any of these occur an emergency telephone number is given for immediate advice.

The cast must be kept away from direct heat such as open fires or radiators. Once an ulcer is healed the inlays are fitted into bespoke shoes so that excessively high peak pressures remain a thing of the past. For the full method with details of bespoke shoes and cast sandals, see Appendix 3.

2.8. Functional Insoles

It has been mentioned above (Bevans, 1992) that abnormal foot function can be implicated as a cause of excessively high forefoot pressures and ulceration. It follows that if functional insoles were used in order to return the foot to a more normal function, the risks of ulceration should be minimised.

Gramuglia *et al* (1988) state that the prescription foot orthosis is used:

"Not for the acute phase of a diabetic ulcer, but rather for prevention of future ulcerations".

The article also points out that a functional orthosis can balance the metatarsal heads and shafts while also providing support for the greater and lesser tarsus, which is very important in diabetics who might undergo Charcot changes. The functional orthosis should provide triplanar support.

It is very important to check the orthosis for any rough edges and to prescribe a 'wearing-in' period, gradually building up the use of the orthosis by an extra hour each day. Footwear is of paramount importance when providing orthoses for insensitive feet. Unless there is sufficient room in the toe box, dorsal lesions will develop. Diabetic clinics usually have a budget for footwear and have an orthotist as a member of the team. Bespoke shoes for diabetics usually come with two cushioning insoles supplied. These can be removed in order to accommodate a total contact inlay or functional orthosis. Examples of suitable footwear can be seen in Appendix 3.

2.9. "Rocker Bottom" shoes

Schaff and Cavanagh (1990) examined the effects of the rocker bottom shoe on plantar pressures. Measurements were made using a flexible pressure measuring insole. As there was only one such insole, volunteers for the study were selected according to shoe size (American size 9). The same pair of size 9 shoes was used throughout the

experiment. Eight volunteers who were non-diabetics and free of lower extremity pathology were chosen to participate in the study.

Data was first collected in the shoes without modifications. Although all the volunteers were familiar with treadmill walking, each walked for 15 minutes before measurements were taken. The treadmill was set at a slow walking speed typical of that which older patients might use. Three separate trials were collected for the unmodified shoe and another three for the modified shoe.

The right shoe was modified as follows: The sole was tapered anteriorly and a rigid metal corset was attached to it. Crepe material was then added to the sole of the shoe and a taper was ground from midfoot to toe to allow an angle of 24 degrees between the fore and rearfoot parts of the sole. The resulting thickness under the heel was 5.5cm, compared to 3.5cm under the metatarsal area. The rear border of the midsole was bevelled in order to allow a reasonably natural heel strike. (Picture in Appendix 3). Modifications were carried out by a very experienced physical therapist who was an expert in the design of shoes for those with insensitive feet. The modification had to be suitable for eight different subjects.

It is interesting to note that while this design did on average reduce the peak pressure under the first metatarsal head, it simultaneously increased the heel strike pressure. Schaff & Cavanagh found that each report on the effects of a rocker bottom shoe gives different results. There are many possible designs for this shoe

and the effect of the geometry on deformed feet may vary considerably. For comment on the statistical aspects, see Appendix 4.

An interesting variant of this design is that used by Ravina in Haifa, Israel (1990) where the hindfoot of the shoe is built up by 3-4cm and the sole under the forefoot is completely removed. These have been used successfully in the treatment of planter ulcers. Ulcers of 6-30 months duration healed within 1-3 months. If the ulcer only affected one foot, the other shoe was built up to match and to facilitate walking. For picture see Appendix 3.

2.10. Cushioning insoles

An heroic BSc(Hons) study carried out by Keith Rome at Durham College compared the shock attenuating properties of seven different chiropody materials over forty hours of wear for each pair of heel pads (Edwards & Rome, 1992). A shock meter in the form of a portable accelerometer (manufactured by J P Biomechanics, Newcastle upon Tyne), was strapped to the medial malleolus of the right ankle. This was connected to a meter unit which was worn on a waistbelt. Pairs of heel pads were cut to the same template from seven different materials which are normally used to manufacture shock absorbing insoles. These included Plastazote, Poron, medium and low density closed cell rubber, Cleron, EVA and Sorbothane.

The heel pads were all measured for thickness using a dial gauge micrometer, both before and after the 40hrs of

wear. Barefoot shockmeter readings were taken and repeated in shoes and socks only before the heel pads were fitted into the shoes with adhesive tape. Further readings were taken once the pads were in place and again after 24hrs and after 40 hrs of wear. Barefoot readings were taken on each day in order to validate the results. The student author walked on a treadmill set at a constant speed of 4km/hr, 8hours a day, five days a week, for seven weeks. He wore the same shoes throughout the test and socks of a design and weight similar to one another. The thickness of the materials was again measured after 40hrs of wear.

Some very interesting results came out of this study, as can be seen from Fig.1. overleaf.

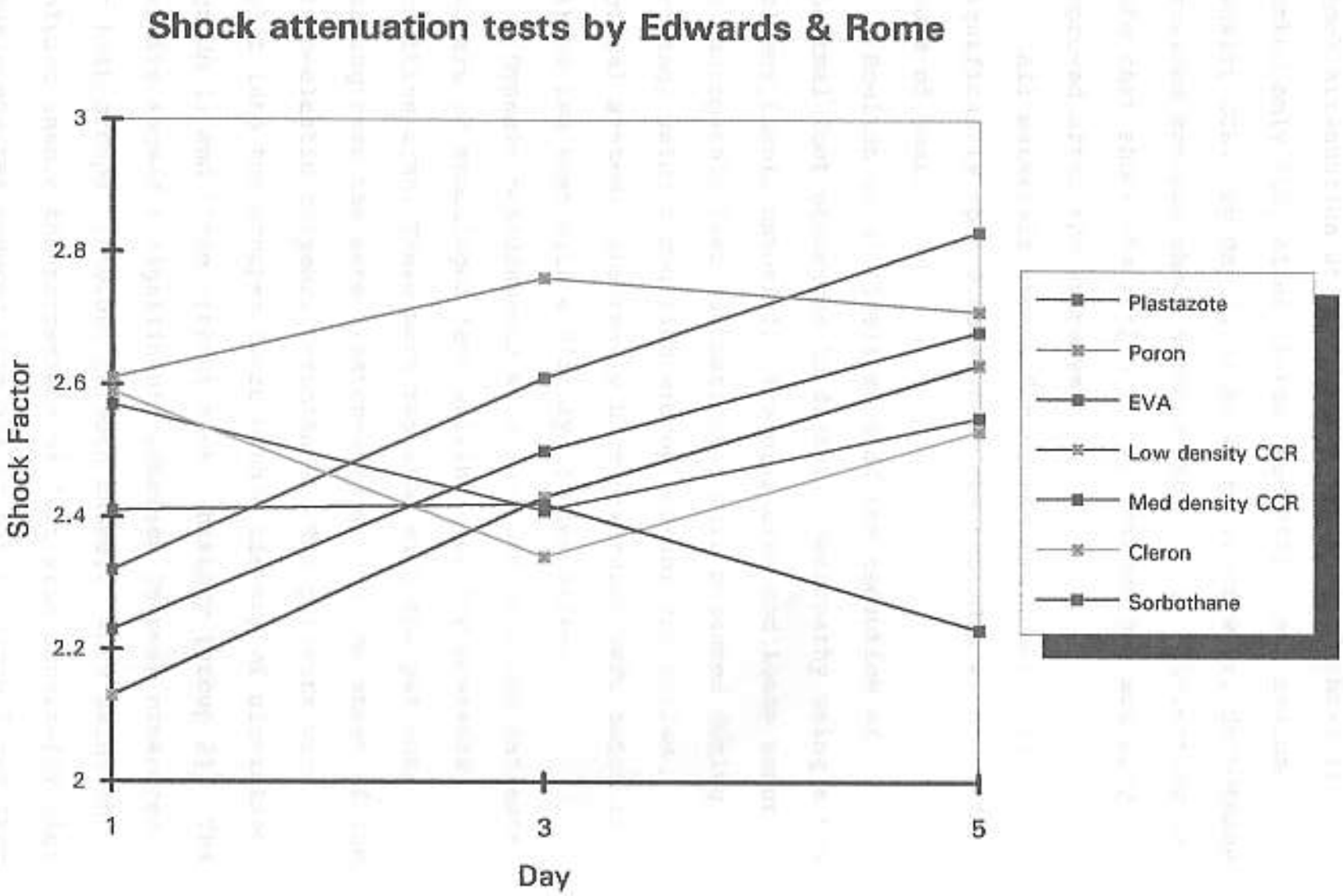
Shock attenuation tests by Edwards & Rome
Shock Factor
3
2.8
2.6
2.4
2.2
2
1
3
5
Day
Plastazote
Poron
EVA
Low density CCR
Med density CCR
Cleron
Sorbothane

Figure 1

Although low density closed cell rubber provided optimum shock attenuation at the outset, by day 3 (24 hrs) it ranked only 4th, after Cleron, Sorbothane and medium density CCR. By day 5, after 40 hours of wear, Sorbothane provided optimum shock attenuation. It is interesting to note that shock absorption for Sorbothane had actually improved after the extra wear.

All materials except Sorbothane had compressed significantly ($p < 0.005$-0.025, see Appendix 4) after 40 hours of wear.

Boulton *et al* (1981) studied the reduction of abnormal foot pressures in diabetic neuropathy using a new polymer insole material. The pressures and loads under 69 neuropathic feet (35 patients) were measured during walking, using a modified microprocessor controlled optical system. Abnormally high pressures were noted in 94% of the feet with a history of ulceration.

Dynamic measurements were recorded with the patients in bare or stockinged feet walking over the pressure sensitive area. These were repeated with the patients walking over the same area covered with a 5mm sheet of the visco-elastic polymer, Sorbothane. The patients were split into two groups: those with a history of ulceration (group 1) and those without such a history (group 2). The results showed a significant reduction in peak pressures in both groups ($p<0.001$ in both cases). When using the polymer insole the percentage of feet with abnormally high peak pressures reduced from 94% to 47% in group 1 and from 33% to 6% in group 2 (see Appendix 4 for statistics).

Barrow *et al* studied the effects of wear on the pressure-relieving properties of 'PPT' and 'SPENCO' insoles. Seven subjects, each known to have a high peak pressure under the second metatarsal head, wore a series of four insoles in random order, for a month each. Two insoles were made from each material on test, each with a 'U' to the second metatarsal head. One of each type had a latex sponge button inserted in the 'U' at the area of peak pressure. Plantar pressures were recorded using a pedobarograph, comparing barefoot recordings to those with insoles strapped to the feet with 'Micropore' tape.

It is difficult to understand why the experiment was set up in this way. If it was to test the pressure reduction capabilities of certain materials it is somewhat questionable to cut a 'U' at the point of peak pressure. To fill the 'U' with latex sponge introduces yet another variable and tests the pressure relieving properties of small pieces of latex sponge.

The stated conclusion that "This study has shown that both insole materials reduce *peak pressure* under the foot", is not strictly correct.

Micropore tape is an unyielding tape and might interfere with the compression capability of the insoles. Boulton's idea of placing a sheet of the material on test over the pressure measuring system and having subjects walk over it seems preferable.

It would be interesting to see a study comparing the pressure attenuating properties of certain materials with those of the same materials with a 'U' cut to the area of

peak pressure. This particular study appeared to raise more questions than it answered.

Chantelau *et al* (1990) conducted a clinical study involving 50 diabetic patients with severe peripheral neuropathy and/or peripheral vascular disease, who were supplied with therapeutic footwear and cushioning insoles. Nine patients died during the period of the study, eight from unrelated causes. Of the 41 remaining participants, only 15% were being treated for foot lesions at follow-up compared with 78% before cushioned footwear was provided.

2.11. Running Shoes

The role of callus in the aetiology of diabetic neuropathic ulcers is undisputed. In Salt Lake City a study was carried out involving 108 diabetics with plantar callus (Soulier, 1986). While all the volunteers wore their normal shoes, plantar calluses were measured monthly for several months. All volunteers included in the study fulfilled the following criteria:

1) Volunteers must be diabetics treated with insulin or oral hypoglycaemics.
2) They must have at least one measurable plantar callus.
3) They must be willing and able to wear running shoes for 80% of the time, including at work.
4) They could not have any active infections or ulcers at the time of admission to the study.

5) The volunteer must have been wearing only ordinary shoes prior to the study.

During the period of the study calluses were shaved by a podiatrist if the patient complained of pain in the area or if the callus was considered too thick to ethically allow it to go untreated.

Once the participant was fitted with running shoes the monthly measurements and callus reduction continued as before, but for a further six months. The measuring system was consistent. The transverse diameter of the lesions was measured in millimetres and the thickness graded as 'thick' or 'thin'.

The running shoes dispensed possessed the following features:

1) Width sizing which facilitates proper fitting;
2) A removable insole which provides cushioning and the ability to fit any felt redirective pads under this insole instead of directly onto the foot;
3) Soft midsoles which provided extra cushioning;
4) Soft upper materials to reduce the possibility of dorsal irritation;
5) Smooth outer sole pattern to reduce the likelihood of tripping indoors;
6) Wide availability without prescription;
7) Relatively low cost;
8) Aesthetic acceptability;
9) Comfort.

Statistical analysis of the data showed:

1) That the size of plantar calluses will diminish with time when running shoes are worn;
2) The wearing of running shoes has a more positive effect than routine podiatric palliative care alone;
3) Participants in the study virtually unanimously described running shoes as being more comfortable than their ordinary shoes.

2.12. Special Hosiery

Padded hosiery has been reported to reduce plantar pressures in patients at risk of ulceration. Veves *et al* (1990) used the optical pedobarograph to study ten patients with high peak forefoot pressures during a study in which they regularly wore experimental padded hosiery for a period of six months. At commencement of the study mean pressure reduction was 31.3% ($p<0.05$). After three months it was 15.5% ($p<0.01$) and after six months, 17.6% ($p<0.01$). A comparison was made with commercially available hosiery designed as sportswear. Although these socks (with high or medium density padding) provided significant pressure reduction versus barefoot (mean 17.4% and 10.4%, $p<0.01$), this was not as great as that seen with experimental hosiery (27%, $p<0.05$).

The conclusion drawn by Veves *et al* is that the use of socks designed to reduce pressure stress on diabetic neuropathic feet is effective and continues to be so for

some considerable time. Commercially available padded sports socks can also be of value in the management of the diabetic insensitive foot.

2.13. Injection of silicone

Between 1966 and 1989, 30 diabetic patients received fluid silicone injections as a subdermal soft tissue prosthesis (Balkin & Kaplan, 1991). This was used as a treatment for 39 neuropathic foot ulcers and 16 painless plantar keratoses without prior ulceration. In the neuropathic foot plantar keratoses commonly precede ulceration; there was a history of previous keratoses at 38 of the 39 ulcer sites. During treatment 29 plantar ulcer sites were injected after healing and 3 before healing. Each of the 7 digital ulcers were injected after healing. After injection there were no activity restrictions and weightbearing was allowed immediately. A greater proportion (79%) of the plantar ulcers injected after healing had no recurrence during the following 13-201 months of observation (mean 6.3 years). One patient who had been previously subjected to multiple episodes of ulcerations where callus had preceeded ulceration, had no further recurrence in the 16 years following injection. Of the six ulcers which occurred post injection, four recurred in the first year. These were all smaller, shallower and less indurated than previously, were treated with plantar pads, healed quickly and remained closed. One ulcer returned after 2 years at

a first metatarsal base under a Charcot's joint. The final recurrence was as an angiopathic ulcer after 16 years. The 3 plantar ulcers which were injected prior to healing showed little or no improvement in two years post injection.

When some of the sites showed signs of incipient tissue breakdown, they were given top-up injections with good effect. No such top-ups were required in the treatment of the seven digital ulcers which had previously been sites for corns. None of the painless keratoses recurred or ulcerated over 1-139 months of observation (mean 4.7 years).

Although there were no complications observed in this study, there have been two reported adverse reactions to silicone injected into the foot. These were asymptomatic fluid migration and skin discoloration over the injected site. The only serious reported medical complications followed the injection of large quantities of silicone of impure or unknown origin for cosmetic augmentation. There have been no adverse experiences in 97 diabetics' feet during 24&5 years of follow-up. Only medical grade fluid was used and in small quantities.

Since many diabetic neuropathic ulcers occur where there is a lack of fibro-fatty padding and/or at sites of hyperkeratoses this would appear to be a very successful preventative technique. The advantage of the patients being able to wear ordinary shoes is considerable as it avoids the stigma attached to the wearing of surgical shoes. Risks appear to be slight and minimal compared to

the very serious risk of ulceration, infection, osteomyelitis, gangrene and amputation.

3 DISCUSSION

After more than a hundred years of research into diabetes mellitus and its complications, diabetes is still the commonest reason for amputation; foot problems are responsible for more diabetic inpatient days than any other factor. There is, therefore, a definite role for the podiatrist in keeping diabetics out of hospital, able to function as members of the community.

In diabetes mellitus there is a picture emerging of the link between good metabolic control and the delayed onset of microangiopathic complications. The causal relationship between abnormally high plantar pressures and diabetic ulceration has been definitely proven.

The podiatrist can only offer advice about the value of metabolic control. Nevertheless he has the ability to prevent some of the later complications such as ulceration, infection and gangrene through observations, good chiropody and the relief of pressure.

It is usual for diabetic clinics to be held where the doctor, dietician, podiatrist, orthotist and nurse collaborate and design treatment plans. With a working knowledge of the available pressure relief methods the podiatrist can play a very important part in improving the quality of life of the diabetics with (or at risk of) neuropathic complications.

This has been shown without any doubt by a number of authors over a prolonged period. Thirteen different methods of pressure relief have been examined in this

study. The methods are not necessarily mutually exclusive; neither is any one method the perfect answer in every situation.

Ideally prevention and protection from ulceration are far preferable to treatment after the event. Several factors come in to play if prevention is to be possible.

The first problem is the identification of those at risk. Known diabetics should be regularly tested for signs of neuropathy: motor, sensory and autonomic. The podiatrist has more than one role in this case - the actual testing, accurate record keeping and the all important task of involving the patient in his own treatment plan. This is attempted through explanation and discussion, increasing his awareness of risks and how to avoid them.

Once there are definite signs of neuropathy, ulceration risks soar. Even before there is any measurable sign, the podiatrist should have noted any biomechanical abnormality which could cause abnormal loading through the foot and where possible have treated it with orthoses. Advice is given early about suitable footwear with adequate room in the toebox

The other person at risk is the undiagnosed diabetic. The podiatrist may be the first person to notice signs of diabetes and if in any doubt should liaise with the General Practitioner. Even the inspection of footwear can supply clues: white spots on mens' shoes can occur with the drying of sugary urine. Since diabetes can develop at any stage in life it is also important to

remain alert to changes in the general health of one's longstanding patients.

The suitability of any method of pressure relief must be judged according to the clinical picture. The table overleaf shows appropriate methods for three different situations: the prevention, treatment and after healing of plantar neuropathic ulcers.

Bearing in mind the information on suitability shown in the table, it is then necessary to consider the available resources in combination with the patient's personal needs.

The injection of silicone shows up very well on many counts: it has been used successfully in America for twenty-five years, with no obvious side effects, although it cannot be used in the treatment of ulcers. In terms of cost effectiveness for the prevention of ulcers and their complications; ease of administration; the obvious benefit to the patient of being able to resume a normal working and social life with few constraints on footwear, there are no apparent drawbacks. There are two main problems: the drug licence and public and medical acceptability. With the recent adverse publicity about the migration of silicone from the breast area to various abdominal organs

Pressure Relief Systems Divided into Categories Indicating Suitability

PREVENTION OF ULCERATION	TREATMENT OF ULCERATION	AFTER HEALING OF ULCERS
Suitable footwear	Rest	Suitable footwear
Functional insoles	Total contact casting	Functional insoles
Padded hosiery	Scotchcast boot	Total contact inlays
Cushioning	Total contact inlays	Cushioning
Running shoes	Rocker bottom shoes	Padded Hosiery
Silicone	Footwear modification	Running shoes
Total contact inlays	Cushioning/ running shoes	Silicone
Surgery	Surgery	Surgery

and the many reported cases of infection it is not considered a safe substance by members of the public. This migration is partly caused by gravity, which is why fluid migration in the foot is both limited and asymptomatic. In the American study there were no infection problems, perhaps because they used highest quality silicone fluid. A spokesman from Dow Corning Ltd. (Britain's leading supplier of silicone products) said that there is no silicone fluid injection of this type being performed in this country at present, neither is there a supplier. She speculated that the reason might be lack of recognition of the technique by the Federal Drug Administration.

There has been one small trial conducted in this country but there is a general reluctance on the part of the medical profession to inject silicone directly in case there should be a severe antigen/antibody response.

Surgery is absolutely necessary in cases of osteomyelitis but otherwise is generally used only when conservative methods have failed. The author has heard an orthopaedic surgeon teach that feet should be operated on **as little as possible** because of the likelihood of giving the patient alternative functional problems.

The treatment of neuropathic ulceration needs a definite dressings policy, so any pressure relief system must complement this and facilitate the changing of dressings.

It is important to consider the patient in the wider context of his normal life. General aims should be to maintain both mobility and his ability to earn a living. In this respect the admission to hospital or the use of non - weightbearing casting are undesirable. Patient compliance, however, is of paramount importance so the ability of the podiatrist to 'sell' the chosen method and its benefits cannot be undervalued.

Plaster of Paris produces fairly quick results but deprives the patient of the ability to observe the limb, with the result that further pressure points and lesions may develop. In this respect the 'Scotchcast Boot' is far preferable in its modified, removable form.

The Hope Hospital system of total contact inlays is a treatment which gives fairly rapid healing times and a follow-up system which is effective in prevention. There is a certain elegance about a system which is designed for both short and long term management.

Rocker bottom shoes seem to have a number of variables in the geometry of the shoe which may unexpectedly delay healing or transfer peak pressures to another risk area in certain patients. The shoes appear normal at a quick glance but would not be cosmetically acceptable in the long term.

While running shoes or 'trainers' are both popular and fashionable they provide a low cost alternative to bespoke shoes for the fitting of functional insoles, total contact inlays or good depth cushioning. In this respect

they have a definite place in both the prevention and treatment of ulceration.

Part of long term management should be the prevention or slowing of the Charcot process by the correction of biomechanical abnormalities which cause excessive loading of the metatarsal heads. To this end functional orthoses can be used but the Hope Hospital casting method can incorporate this idea in the manufacture of total contact inlays by taking impressions with the foot in (or close to) subtalar joint neutral position. The limiting factors are the quality and range of motion of the joints but even if these are limited, total contact inlays will provide support for the entire foot.

Padded hosiery in conjunction with suitable footwear provides a very low cost form of prevention, especially for the young men and women who appreciate wearing sports shoes and socks. Many people do not wish to be thought disabled by their colleagues and want to lead as normal a life as possible. On the other hand, someone who is already severely disabled will accept any system which will improve mobility. Nevertheless it is still the part of the podiatrist to find the best compromise between function and acceptability.

An interest in the Hope Hospital work with total contact inlays prompted the author to experiment with the method and apply it to a case of intractable plantar pain caused by a neurovascular corn and several hyperkeratotic pressure areas. This patient has had painful feet for thirty years and her first comment on having total contact

inlays fitted to her training shoes was, "This is the first time I have felt comfortable in thirty years". Although in the following few weeks it was not possible to increase the chiropody treatment intervals, one longstanding area of apical callus disappeared completely and the patient's mobility increased sufficiently to allow her to take a part time job.

Another patient has severe rheumatoid arthritis which affected his feet so badly that he was unable to walk more than a few yards. When he tried the total contact inlays in his surgical shoes he said, "Smashing". He now claims to be able to walk comfortably for several minutes at a time.

This is surely an area which merits further research.

4 CONCLUSIONS

Most of the conclusions drawn from this study are related to the role of the podiatrist in the care of diabetics.

a) Routine observations are of paramount importance if new diabetics are to be diagnosed. Quantitative testing for neuropathy is an important part of the care of known diabetics and can be a predictor of ulceration.

b) The manufacture of functional insoles for diabetics with biomechanical abnormalities will reduce peak pressure areas which are caused by abnormal loading. They may also slow the Charcot process in patients with autonomic neuropathy. Insoles can only be fitted to appropriate footwear with adequate room in the toebox.

c) Screening diabetics for unusually high peak plantar pressures can be a good predictor of ulceration.

d) Pressure reduction methods can be used in both the prevention and treatment of plantar neuropathic ulcers.

e) It is necessary to involve the patient in his own treatment plan: if he understands the principles of the proposed treatment and the dangers associated with his condition he is more likely to be cooperative.

f) Any treatment plan must have both short and long term attributes. It must be tailored to the patient's lifestyle and aim to maintain his mobility and function within the community.

g) The podiatrist should have information available on where to buy padded socks and suitably cushioned footwear for diabetics who do not need surgical or bespoke footwear.

h) The knowledge gained from research into the treatment of diabetic neuropathic ulceration should be applied to other conditions involving excess plantar pressures.

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APPENDIX 1

CLASSIFICATIONS OF NEUROPATHY

APPENDIX 1

Classifications of neuropathy

A. ACUTE MOTOR NEUROPATHY WITH VARIABLE SENSORY INVOLVEMENT:

1. Guillain-Barrie Syndrome.
2. Polyneuropathy associated with: Hepatitis; Mononucleosis; Diphtheria; Porphyria.
3. AIDS -progressive poyneuropathy of lower extremities with sphincter loss.

B: ACUTE MOTOR NEUROPATHY:

1. **Diabetic** Multiple Mononeuropathy.

C: ACUTE ASYMMETRIC SENSORIMOTOR POLYNEUROPATHY:

1. Polyarteritis Nodosa
2. Wegener's Granulomatosis
3. **Diabetes Mellitus**
4. Other angiopathies
5. AIDS

D: SUBACUTE SYMMETRIC SENSORIMOTOR NEUROPATHY:

1. Toxic:
   a) Heavy metals e.g. arsenic, mercury.
   b) Drugs:
      1) Antibiotic e.g. streptomycin.
      2) Antineoplastic e.g. methotrexate.
      3) Cardiovascular e.g. hydralazine.
      4) Other e.g. gold salts, chloroquine & phenylbutazone.
   c) Industrial chemicals e.g. acrylamide,

methyl bromide.

2. Nutritional deficiency:

   Vitamin B_{12} ; niacin (pellagra); thiamine (beriberi); pyridoxine; chronic alcoholism; Vitamin E (chronic biliary cirrhosis or malabsorption syndromes).

3. Uraemia

E. SUBACUTE TO CHRONIC, PREDOMINANTLY SENSORY NEUROPATHY:

1. **Diabetes Mellitus**
2. Drugs e.g. chlorambucil, metronidazole.
3. Leprosy
4. Paraneoplastic
5. Pyridoxine toxicity
6. AIDS - small fibre axonal, large fibre.

F. SUBACUTE TO CHRONIC, PREDOMINANTLY MOTOR NEUROPATHY:

1. **Diabetes** - proximal diabetic motor neuropathy (amyotrophy).
2. Lead neuropathy

G. CHRONIC SENSORIMOTOR NEUROPATHY:

1. **Diabetes** (mixed sensorimotor and autonomic)
2. Multiple myeloma
3. Dysproteinaemias e.g. macroglobulinaemia.
4. Paraneoplastic
5. Uraemia
6. Leprosy
7. Amysoidosis
8. Sarcoidosis

H. HEREDITARY MOTOR AND SENSORY NEUROPATHIES (HMSN):

Types I-III (e.g. Charcot-Marie-Tooth syndrome)

I. HEREDITARY SENSORY NEUROPATHIES (HSN):

Types I-V

J. HEREDITARY NEUROPATHIES WITH KNOWN METABOLIC DEFECTS:

1. Fabry's disease
2. Metachromatic leukodystrophy
3. Refsum's disease
4. Adrenomyeloneuropathy
5. Tangier
6. Krabbe's disease

K. OTHER HEREDITARY NEUROPATHIES:

1. Familial amyloid neuropathy
2. Hereditary predisposition to pressure palsy
3. Giant axonal neuropathy
4. Friedrich's ataxia

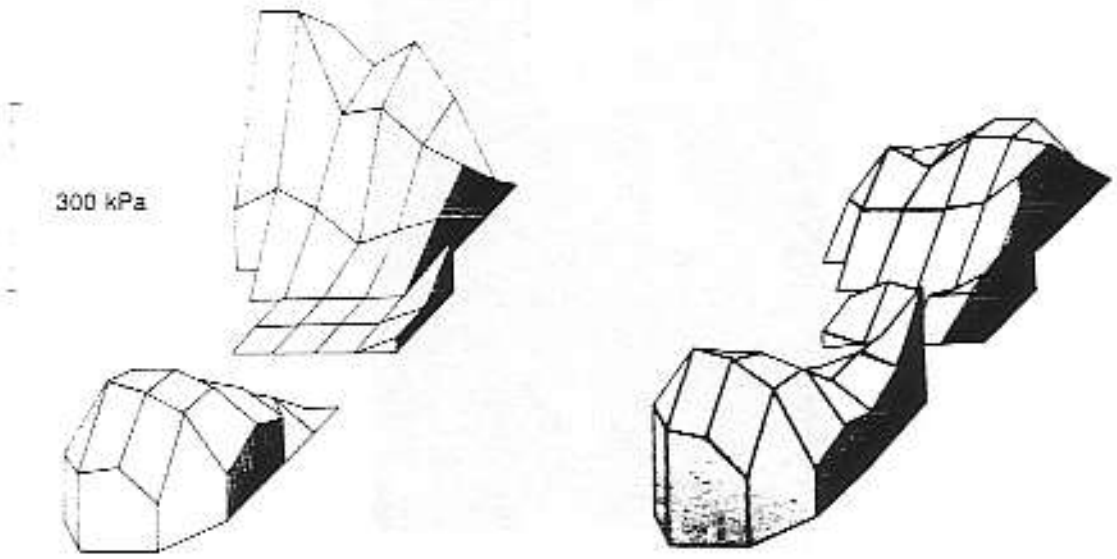
L. MONONEUROPATHIES:

1. Trauma
2. Entrapment
3. Carcinomatous infiltration
4. Vasculitis
5. Leprosy

APPENDIX 2

EXAMPLES OF PLANTAR PRESSURE READOUTS

List of illustrations *Page*



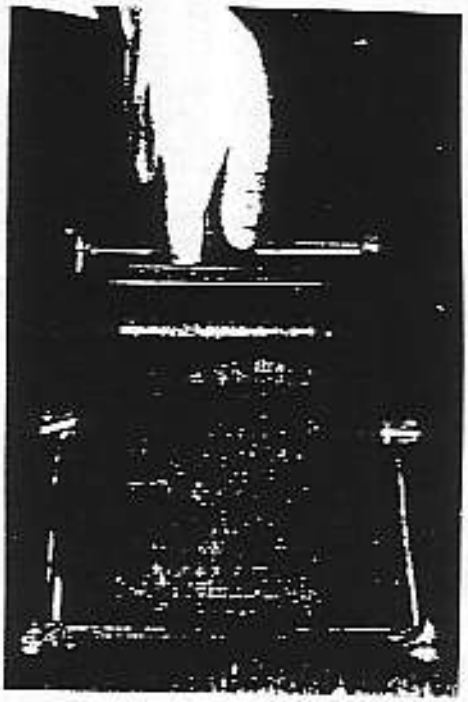
1(a) Ordinary shoe

1(b) Modified "rocker" shoe

Flexible pressure measuring insole result

(Schaff & Cavanagh, 1990)

Harris mat

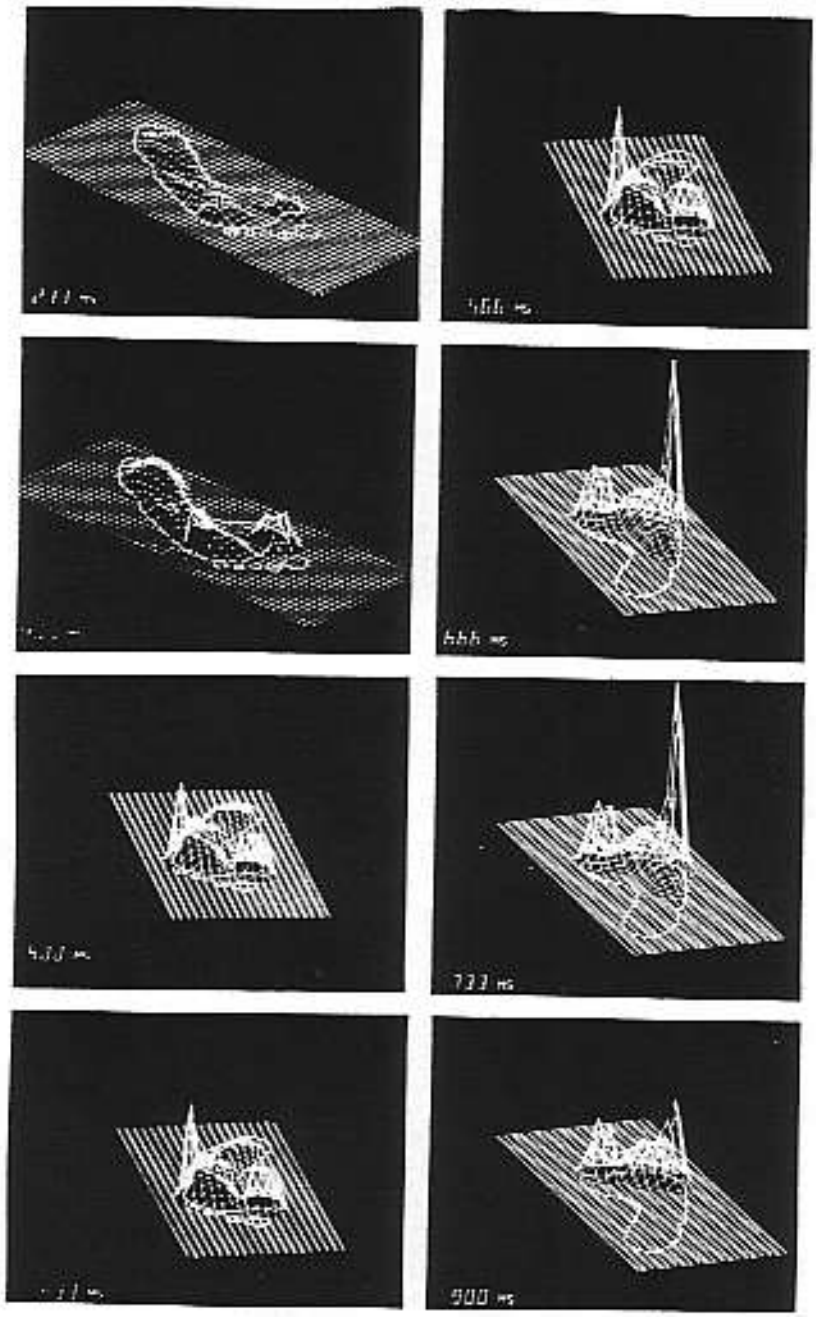


2(a) Inking the mat

2(b) Plantar surface of foot

2(c) Standing on Harris mat

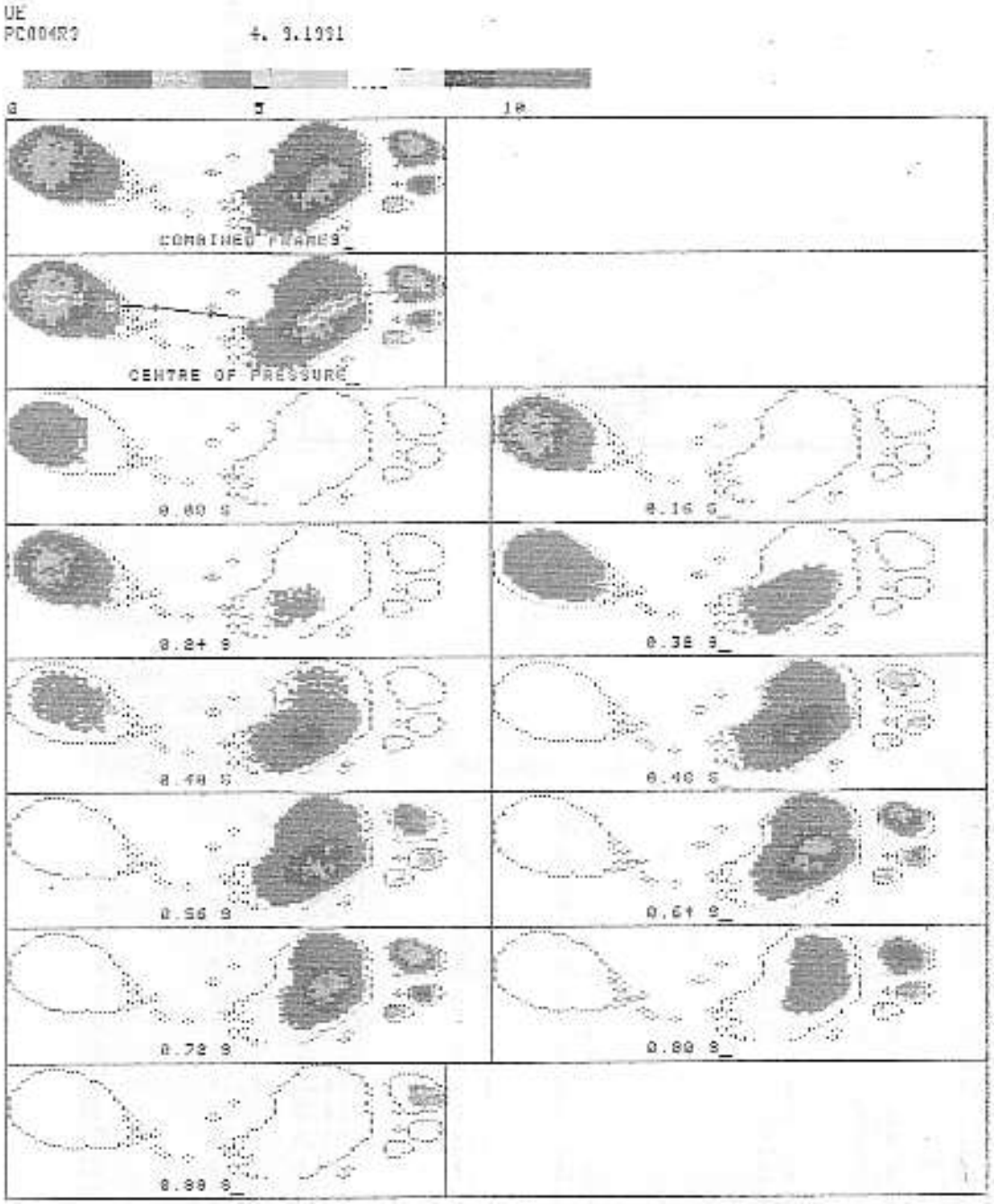
2(d) Pressure result (Cavanagh, 1985)



Computerised result from piezoceramic transducers

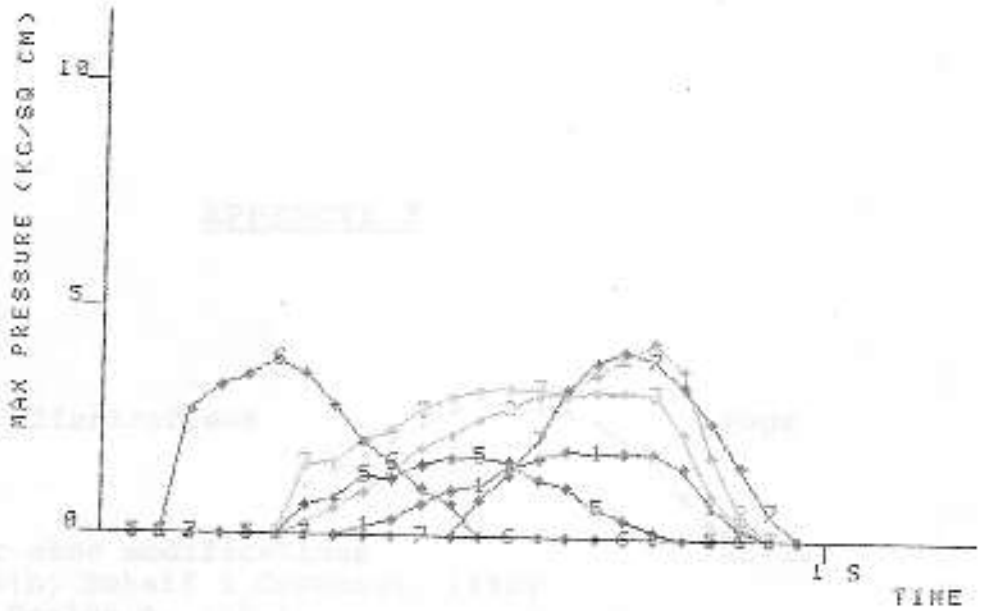
(Cavanagh *et al*, 1985)

Optical pedobarograph result



4(a) Normal patient, no excessive pressures

Areas of interest



PC004R3 4. 9.1991

WEIGHT 0.0
NO. OF AREAS 7
BASE FORCE VALUES 3 15 14 13

FRAME	FORCE	CALFAC	MAXIMUM PRESSURE VALUES 1	2	3	4	5	6	7
1	0.0	0.273	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2	19.3	0.273	0.0	0.0	0.0	0.0	0.0	0.0	0.0
3	46.9	0.273	0.0	0.0	0.0	0.0	0.0	2.7	0.0
4	57.0	0.273	0.0	0.0	0.0	0.0	0.0	3.3	0.0
5	74.7	0.258	0.0	0.0	0.0	0.0	0.0	3.5	0.0
6	90.1	0.241	0.0	0.0	0.0	0.0	0.0	3.9	0.0
7	96.5	0.224	0.0	0.2	1.6	1.1	0.7	3.6	0.0
8	91.8	0.204	0.0	0.6	1.6	1.4	0.8	2.9	0.0
9	85.3	0.184	0.2	0.9	2.1	1.7	1.3	2.1	0.0
10	81.4	0.177	0.4	1.4	2.3	2.1	1.2	1.6	0.0
11	78.6	0.172	0.7	1.9	2.7	2.4	1.5	1.0	0.0
12	82.6	0.170	1.0	2.2	3.1	2.7	1.7	0.7	0.0
13	83.3	0.161	1.1	2.6	3.2	3.1	1.8	0.0	0.8
14	88.1	0.149	1.5	2.8	3.3	2.8	1.5	0.0	1.3
15	93.2	0.141	1.2	3.1	3.3	2.8	1.3	0.0	2.1
16	94.6	0.137	1.9	3.3	3.1	2.7	1.1	0.0	3.3
17	94.6	0.134	1.9	3.6	3.2	2.5	0.7	0.0	3.9
18	91.9	0.133	1.9	4.0	3.2	2.1	0.4	0.0	4.1
19	83.5	0.131	1.8	4.3	3.2	1.7	0.1	0.0	3.9
20	64.3	0.129	1.5	3.7	2.3	0.9	0.0	0.0	3.4
21	39.3	0.122	0.7	1.8	1.0	0.4	0.0	0.0	2.6
22	15.5	0.122	0.2	0.6	0.4	0.0	0.0	0.0	1.6
23	3.3	0.122	0.0	0.0	0.0	0.0	0.0	0.0	0.6
24	4.9	0.122	0.0	0.0	0.0	0.0	0.0	0.0	0.0

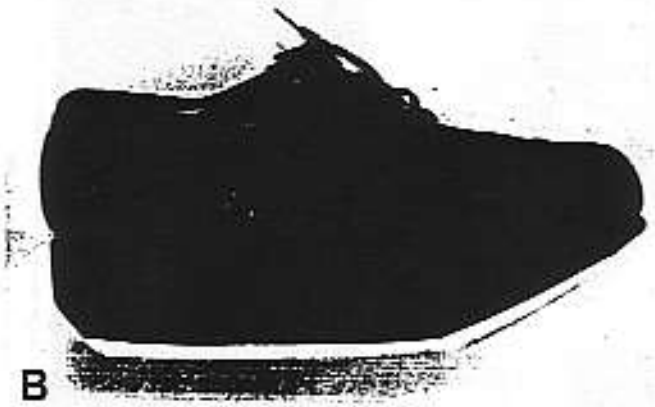
4(b) Normal patient contd.

APPENDIX 3

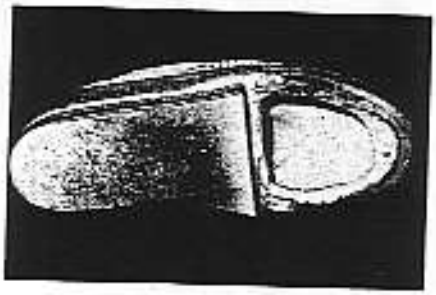
List of illustrations *Page*

Rocker shoe modifications

A



B



C

1(a) Shoe before "Rocker" modification
(Schaff & Cavanagh, 1990)

1(b) Shoe after "Rocker" modification
(Schaff & Cavanagh, 1990)

1(c) "Haifa" rocker modification
(Ravina A, 1990)

The "Hope" X-Lite® Removable Walking Cast

Suggested Procedure

Anita Wiliams, SRCH
Chief Chiropodist
Hope Hospital
SALFORD, M6 8HD

INTRODUCTION

The "Hope" removable walking cast is a method of providing optimum pressure redistribution in the management of plantar foot ulcers. The cast has been used on patients with neuropathic and ischaemic foot ulceration with good results and healing times.

The cast is removable for wound inspection and dressings. Pressure redistribution is achieved by making the whole of the plantar surface of the foot contact the inner surface of the cast.

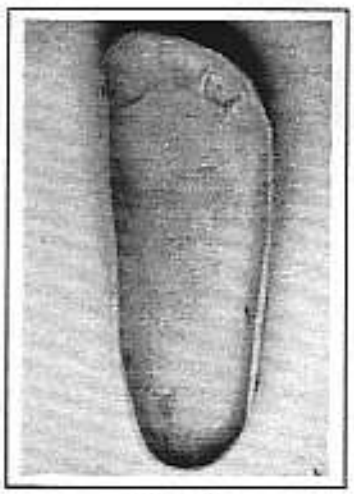
MATERIALS

'X-Lite' thermoplastic sheet 20 x 37.5 cms
Cast heels, adhesive, medium
Plaster of paris powder
Rigid thermoplastic, - 'Resurplus'
Medium density 'EVA', ½" thick
Low density 'Plastazote' 6mm
Semi compressed felt, adhesive one side 7mm
'Fleecy Webb" padding
'Velcro', 30mm or 50mm
Tape, 1" 'Micropore'
Glue, 'Gripsotite'

EQUIPMENT

Heat gun or oven
Grinding wheel
Casting Boxes

(Fig 1).



(Fig 2).

(Fig 3).

METHOD

1. A negative impression of the patients foot is taken using a casting box.
2. From this positive plaster of paris cast is made. *(Fig 1).*
3. A total contact inlay is formed on this cast using medium density 'EVA' and 'Plastazote". The 'Plastazote" being next to the plantar aspect of the cast. *(Fig 2).*
4. The inlay is then machined with bevelled edges and flat plantar aspect.
5. A rigid sole piece of thermoplastic is glued to the plantar aspect of the inlay. A cast heel can be applied at this stage or on completion of the cast. *(Fig 3).*
6. A piece of 7mm semi compressed felt is cut to extend around the back of the lower third of the first and fifth metatarsal heads. N.B. Care must be taken to keep the patients foot at right angles to the leg throughout the procedure. *(Fig 4).*
7. The felt forms the liner to the cast and can be used as a template for cutting out the 'X-Lite' shell.
8. Three layers of 'X-Lite' are cut to the shape of the felt and heated together using a heat gun or oven.
9. The felt liner is positioned around the patients foot, adhesive side outermost and held in place with tape.
10. The total contact inlay is held against the foot using tape with the felt liner overlapping on its outer edge.

11. The 'X-Lite' shell is then reheated and applied over the felt liner and overlapping slightly onto the sole.

12. Once cooled, the whole cast is removed from the foot. *(Fig 5).*

13. A sole piece in one layer of 'X-Lite' is heated, applied and allowed to cool. Also the front of the cast is reinforced with two layers of 'X-Lite'. *(Fig 6).*

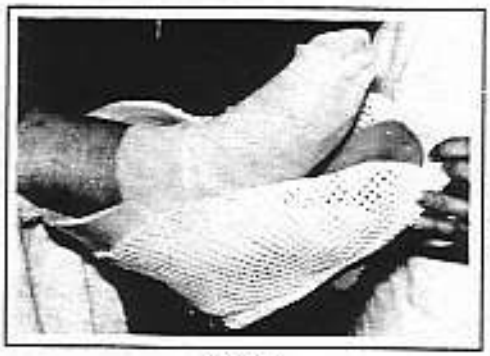
14. The upper edges of the 'X-Lite' are neatened and 'Fleecy Webb' stuck around them.

15. Three 'Velcro' straps are applied by spot heating the 'X-Lite' or using the adhesive 'Velcro'.

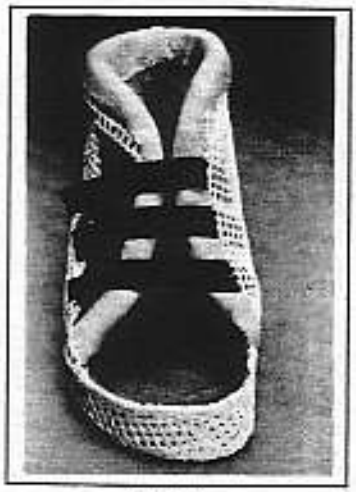
16. A walking cast heel is then heated and applied to the sole or the walking cast can be fitted into a cast sandal.

17. The patient is advised and monitored whilst initially wearing the walking cast and given written instructions. *(see appendix).*

(Fig 4).



(Fig 5).



(Fig 6).

SUPPLIERS

1. 'X-Lite' Sheet No 790
   Walking Cast Heels Small No 20 or Large No 21
   'Velcro'

   ORTHOPAEDIC SYSTEMS
   22-23, Oldgate
   St. Michael's Industrial Estate
   Widnes, Cheshire WA8 8TL
   Tel: 051-420 3250
   Fax: 051-495 2150

2. Casting Boxes, and 'Resurplus'

   PROMEDICS
   Clarendon Road
   Blackburn, Lancshire
   Tel: 0254 57700

3. 'Plastazote' and 'EVA'

   NORTH SEA PLASTICS
   Lillynurn Works
   Glasgow
   Tel: 0360 312 699

4. Felt, 'Fleecy Webb' and 'Tape"

   FOOTMAN AND COMPANY LIMITED
   Grove Hill
   475-479 London Road
   MITCHAM
   Surrey

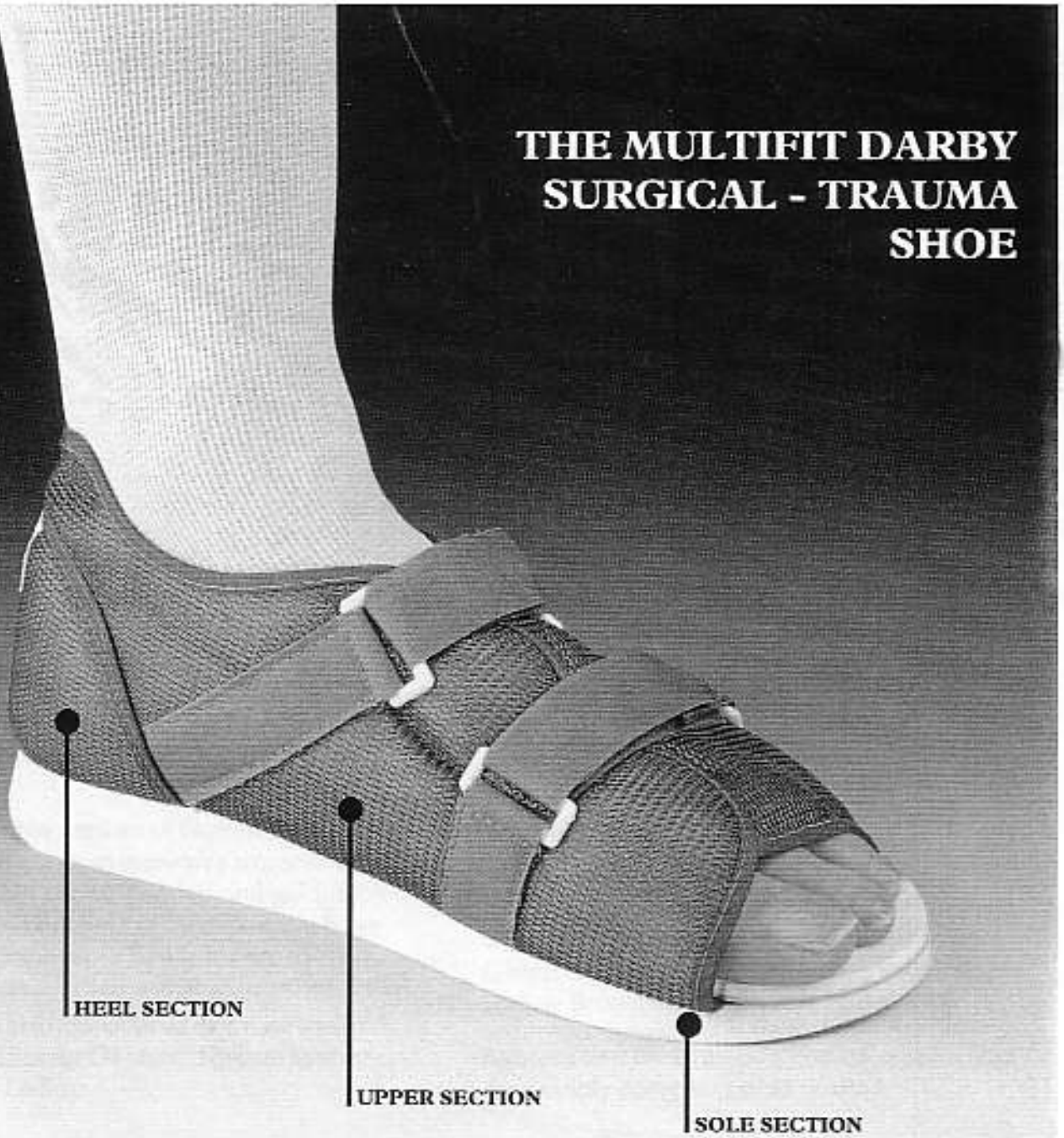
Appendix

Advice Sheet for Patients Using the "Hope" Walking Cast

1. It is necessary to slow down your normal walking pace. (Use crutches or walking sticks if advised to do so).
2. The cast should be worn all the time with the following exceptions:
   a) Whilst resting with foot on a stool
   b) Whilst in bed
   c) Whilst redressing your foot
3. Do not sit close to a fire or radiator or leave your boot near either of these as heat will distort the cast.
4. The exterior of the cast can be cleaned with warm soapy water and a cloth. Do not immerse the boot in water.
5. (As advised) Check your foot and leg for unusual redness, colour change or breaks in the skin. If any of these should occur contact the Chiropody Department for advice immediately.

If the problem occurs outside the departments normal hours contact the Accident and Emergency Department

THE MULTIFIT DARBY SURGICAL - TRAUMA SHOE



Finally a biomechanically sound surgical-trauma shoe that provides controlled ambulation with the comfort and shock-absorbing qualities of a fine running shoe. This versatile trauma shoe is available in an attractive navy colour and is ideal for the conditions listed:-

- CAST SHOE
- ISCHAEMIC FOOT
- DIABETIC ULCERS
- ACUTE DERMATITIS
- SEASAMOIDITIS
- OSTOTOMIES
- TOE FRACTURES
- STRESS FRACTURES
- ANKLE SPRAINS
- RHEUMATOID ARTHRITIS

UPPER SECTION

Made with Tricot foam-lined, nylon mesh which contours to the foot and incorporates a padded dorsal flap. The touch 'n' close fastening accommodates bulky dressings and provides compression.

HEEL SECTION

The reinforced heel reduces slipping and lateral motion.

SOLE SECTION

The shock-absorbing EVA non-skid, outer sole has a 15° declination angle with an inner sole that contours to the foot. Proper heel strike is achieved by the tapered heel.

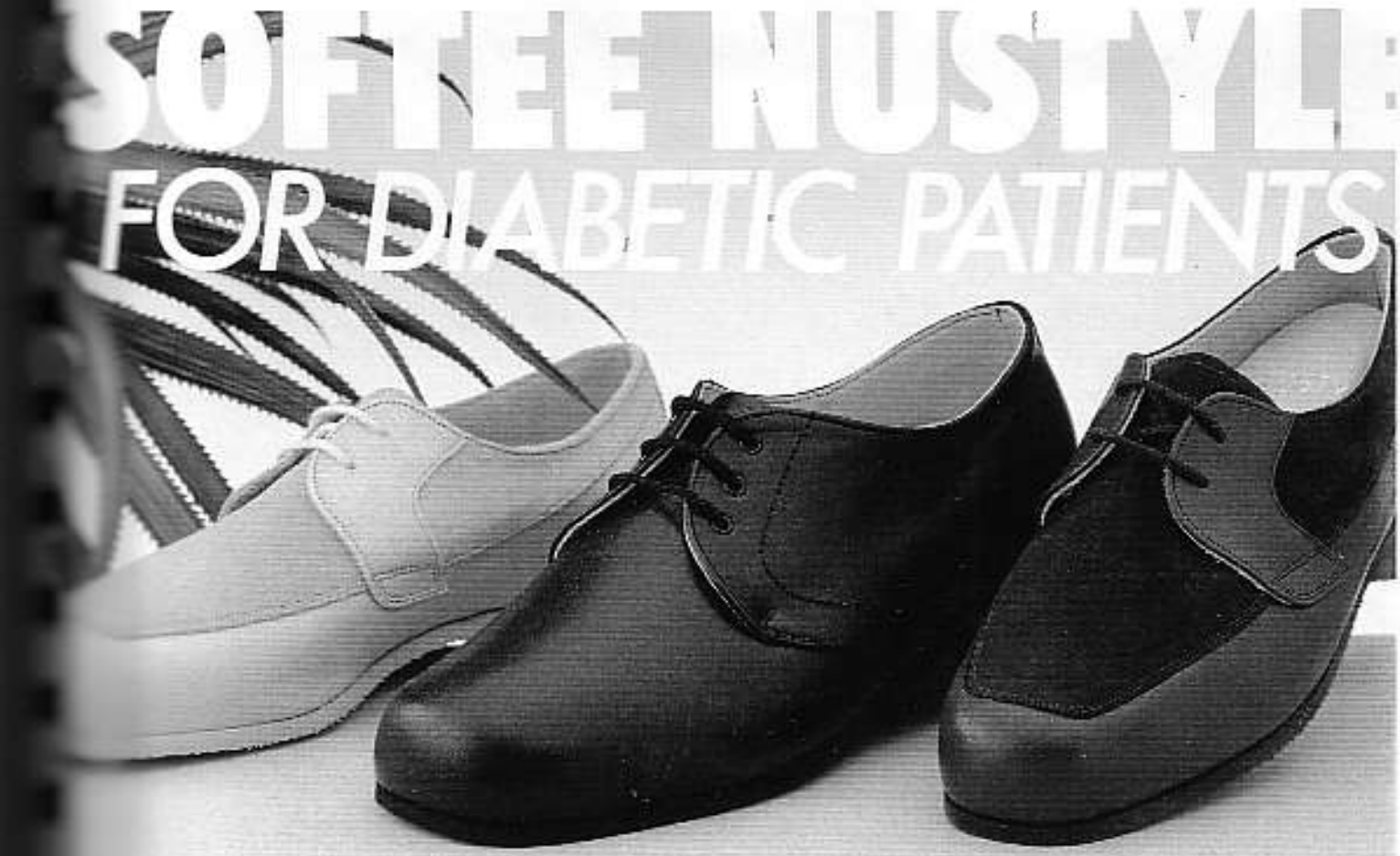
SIZE GUIDE

UK SHOE SIZE		PRODUCT CODE
MEN	5 - 7	DS 1M
	7½ - 9	DS 2M
	9½ - 11	DS 3M
	11½ - 14	DS 4M
WOMEN	3 - 4½	DS 1W
	5 - 6½	DS 2W
	7 - 9	DS 3W
CHILDREN	12 - 1	DS 1C

A3-V

V-M MARKETING
St. Peter's · Ubbeston · Halesworth · Suffolk · IP19 0EX · England · Tel: (0986) 798120 · Fax: (0986) 798040

SOFTEE NUSTYLE FOR DIABETIC PATIENTS



This softee version of Nustyle shoes is the product of our own extensive experience in diabetic foot clinics. Assisted and guided by specialists in this field of medicine, we have designed shoes specifically for the foot problems associated with this disorder.

These are now offered as a new line of stock shoes in Black for Gents and Brown leather and suede for Ladies.

SPECIFICATION

Design
Ladies – as for Standard Nustyle.
Gents – Plain vamp 3-tie Gibson.
All linings have a minimum of seams and stitching.

Uppers
Softee leather uppers for maximum foot comfort.
Vamp linings in 6mm soft foam-backed nylon material. Quarter linings are of soft lining leather which is reversed for ladies to provide an even softer finish.

Toe Puffs & Stiffeners
Wall puffs only at the front to avoid toe abrasion. Heel stiffeners are situated 15mm lower than regular Nustyle shoes.

Insoles & Shanks
As for Regular Nustyle shoes.

Soles
Ladies – EVA Micro unit as Standard.
Gents – EVA soles with Micro heel.

Inlays
Two 6mm layers of Poron microporous polyurethane cushion.

Joint Girth Measures
As for Regular Nustyle shoes.

Size Range
Gents: 4-11 Wide and Very Wide.
Ladies: 1-9 Wide and Very Wide.

Stock Colours and Sizes
Gents: Black only 4-11 Wide and Very Wide.
Ladies: Brown only 2-7 Wide and Very Wide.

Bootees and other colours can be supplied but on a strictly non-returnable basis.

The Nustyle Shoes

A3-vi

Ken Hall Footwear

Ken Hall Limited, Newman Street, Kettering, NN16 0TG Tel:(0536) 522468

APPENDIX 4

AN APPRECIATION OF THE STATISTICS USED IN SOME OF THE DOCUMENTS REVIEWED

APPENDIX 4

An appreciation of the statistics used in some of the documents reviewed

Clinical trials are an objective method of appraisal. Paired groups are randomly assigned or if there is a chronic stable condition the effect of a treatment can be measured by "before" and "after" readings involving the same patients. This is known as a crossover trial.

The parametric statistical tests of significance (e.g. standard t-test) have been designed around the standard distribution in a number of thirty or more. Smaller samples would therefore give an erroneous result. In these situations the non parametric tests may be used (e.g. Wilcoxon, Mann-Whitney 'U' test).

"For clinical trials with matched pairs of subjects the Wilcoxon test might well be suitable and with matched groups the Mann-Whitney test would be appropriate." (Reid & Boore,1990).

Mueller et al (1989)compared traditional dressing treatment with total contact casting. This was a controlled clinical trial and there was no significant difference between the groups. A chi-squared test was conducted on the results comparing the number of ulcers healed in each group then the number of infections contracted during the course of the trial. This is a

non-parametric test which explores the relationship between two variables, comparing the diatribution with the values expected if the variables were truly independent.

There is a simple check to see if this is a suitable test of significance, which involves setting the results out in a table as follows:

	TCC	TDT	TOTAL
Ulcers healed	19	6	25
Ulcers not healed	2	13	15
Total	21	19	40

An expected value is calculated for each cell by multiplying the row total by the column total, dividing the product by the grand total. The test statistic is applied, using 5 degrees of freedom. The test cannot be used if a) the expected value in any cell is less than 1 or b) more than 20% of cells have an expected value ofless than 5. Expected values are 13.1, 11.9, 7.9, 7.1 reapectively, so this is an appropriate test to us with this data.

Boulton et al (1981), in testing a new polymer insole material, took barefoot pressure readings on two groups of neuropathic patients - those with a history of ulceration and those without. The chosen test (Mann-Whitney 'U' test) is quite suitable, but it was

unnecessary to plot a linear regression line when it was not subsequently used to derive an equation.

Bevans (1992) in the research into biomechanics and plantar ulcers also calculated linear regression. It is over elaborate to plot a regression line (and not use it) when correlation gives adequate information on the strength of association.

Edwards & Rome (1992) used a students t-test on 7 shock factors but this was probably correct as they had several readings for each material. There is no obvious information in the article to state which test of significance is used for the 'before and after' measurements of thickness.