Neuropathic Feet

● G. WARREN

"Without pain we destroy ourselves. Pain is the most important natural therapeutic. …..Pain makes us rest and protect the affected area, allowing healing to occur".[14] Hilton, 1863

INTRODUCTION

Neuropathic feet can be defined as feet with secondary impairments due to motor, sensory or autonomic nerve function impairment.[7] The main secondary impairments are plantar ulceration and neuropathic bone disintegration. The primary and secondary impairments may be clinically obvious, or, as yet unsuspected by patient or health care professional. The patient with neuropathy is often unaware of trauma because pain, the natural protective mechanism, is absent. Pain is an unlikely cause of presentation for diagnosis or treatment. Many clinicians do not take seriously complaints of pins and needles or burning sensations that these patients sometimes report. These are often the patients' only means of voicing that there might be a problem as these patients rarely complain of pain. The numbness, 'heaviness' or 'deadness' of an anaesthetic hand or foot, however, can be a real discomfort.

Symptoms that 'alert' the patients' minds are: 1) 'paraesthesia', indicating a change in nerve function or an early warning of a pending ulcer. 2) motor impairment such as weakness in dorsiflexion of the foot or the development of claw toes. Patients are unlikely to report any impairment associated with autonomic nerve function impairment.

At the Time of Diagnosis

Often patients with leprosy and diabetes are first diagnosed with the disease or a neuropathy when there is said to be sensory loss or a 'non-healing' ulcer.[10] Learn to observe patients as they walk into the clinic. If there is a normal gait in the presence of an ulcer it is obvious that sensory impairment is present. If the foot is very cold to touch there is probably also autonomic dysfunction. Once the nature of the neuropathy is known it should be determined if any therapy is indicated to slow down or reverse the neuropathy.

Patients with multiple deformities who have obvious loss of pain perception may well be labelled 'leprosy' in leprosy endemic countries. It is essential that (para) medical workers know about the medical conditions that may result in neuropathic limbs. The only satisfactory method of long term help for patients with diminished pain perception of limbs is to involve the patient in 'self care' and injury prevention. This is best commenced at diagnosis.

For social reasons a clinician in a leprosy endemic country may deliberately not want to tell the diagnosis or he may not have been taught to make a diagnosis. This may result in inadequate treatment till obvious deformity or irreversible neuropathy occurs. Some patients may only report symptoms that will not reveal leprosy as a cause.

Neuropathy is not uncommon, but is frequently not recognised and hence the patient may receive inadequate treatment. Some people are born with a neurological impairment that may not become obvious for many years. Others develop neuropathy as the first sign of a

disease like diabetes or secondary to diseases such as acute infection or malignancy. In leprosy, neuropathy may be a presenting sign or it may develop during or after treatment.[20]

Presentation of Neuropathy

A motor impairment often attracts immediate attention. The patient may have a drop foot or clawed toes, which results in an altered gait. There may also be ulceration, or thick callus that causes no discomfort because of the sensory impairment. The patient may have paralysis or loss of sensation in some other area of the body, with the foot not previously presenting a problem. Be aware of shortened fingers or toes. Look also for burns scars or signs of trauma on all limbs. These impairments (when painless) in leprosy endemic countries are often accepted as leprosy. Be aware of the possibility of congenital indifference to pain (Fig. 13-1).[16]

FIGURE 13-1 Young boy with congenital absence of pain.

Patients are not likely to present for treatment with autonomic impairments only, though dry cracking skin, lack of hair growth, and capillary lability may be present especially in patients with signs of neuropathy affecting motor or sensory nerves.

EXAMINATION AND RECORDING OF NEURAL FUNCTION

It is recommended that at the time of diagnosis a full neurological examination be done to assess the extent and severity of neural impairment (see appendices A-B). This should at least include a basic *clinical* motor and sensory assessment.[7] Many patients with diabetes have already some reduction in nerve conduction velocity at diagnosis.[12] Clinical testing should be repeated at regular intervals, the frequency depending on the aetiology and expected course of the disease. The standard instrument of choice for testing sensory impairment is nylon monofilaments.[2,5] This tests only one modality and while a useful tool for recording touch perception, it may not be related to impaired pain or thermal perception. In one study monofilaments were supplied to patients with diabetes for them to assess the sensory status of their feet.[4] Sensory loss, previously undetected by providers, was reported by 23 out of 145 subjects. Self-administered tests, whenever feasable, provide patients an opportunity to share in the responsibility for preventing foot problems, but should not replace routine evaluation by a professional.[4]

Skin Care–Self Care (see also Appendix C)

Because of the *lack of pain perception*, the limbs are prone to injury and trauma. To prevent permanent impairments a patient should practice self-care. The time of diagnosis is the time to start self-care. Skin care is important in the prevention of (re) ulceration and includes the trim-

ming of scar tissue and callus. A daily routine should include inspection of the skin for abrasions, blisters, swelling and redness (Figs. 13-2-4). Hydration of the autonomically deprived skin (soaking) and oiling is important to maintain skin suppleness. Persons 'at risk', with sensory impairment, that have remained ulcer free for a long time are usually the best facilitators

FIGURE 13-2 Self-care: Foot inspection.

FIGURE 13-3 Deformed hands can still 'care' for anaesthetic feet.

Figure 13−4 a. The patient arrived in the clinic saying his feet were "no problem". On examination a hard plaque of callous was found (marked A). **b.** After a thorough soak and scrape the plaque of callous was placed on the clean foot beside the ulcer which had occurred under the callous and first became obvious when the callous was removed.

for patients that need to learn and understand about skin care.

'Hot-spots' are localised areas of increased temperature, indicating an underlying problem, which needs to be addressed. They can be detected by the patient.[15] Hot-spots are of such significance in the pathogenesis of Neuropathic Bone Disintegration (NBD) that a few paragraphs will be devoted to this topic.

Hot spots: warning sign of 'break-down'.

A neuropathic limb may feel cold but still has the ability to become hyperaemic after trauma or infection. Areas of localised heat and swelling are known as 'Hot-spots'. Once neuropathy has developed the A-V shunt may become more pronounced over a period of months or years. The foot feels cold but the veins may be engorged. This is not due to poor circulation but to the absence of the neurological control of the capillary lability. Patients should learn how to look for hot spots and understand that their presence indicates that something needs to be done *now!*

The natural physiologic responses to trauma are pain, heat, swelling and redness. When neuropathy compromises pain perception the other three signs become more important. A 'Hot Spot' is an easily detected sign of deep tissue trauma. Most people can detect temperature differences of 2 degrees centigrade, and with training 1.0-1.5 degrees C. Often 'Hot Spots' will show a difference of 3-4 degrees when compared to other parts of the foot or preferably the same area on the contralateral foot. Swelling may not be prominent and swelling alone without heat
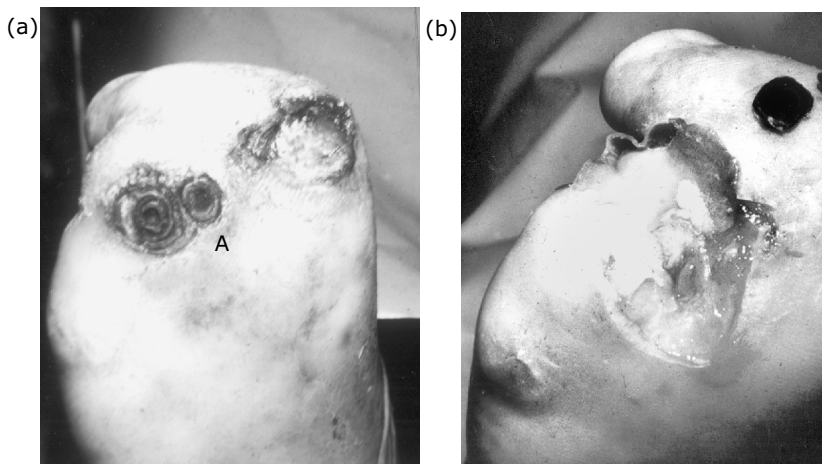
may be due to vascular problems. If a patient had an ulcer or has a bone deformity there may be excessive pressures on these areas. The patient needs to learn to monitor these sites each day, preferably at night before going to bed.

NEUROPATHIC BONE DISINTEGRATION

In the sensate foot any trauma will cause the person to alter the use of the foot, or make the person rest the foot.[14] This may be just enough to protect the limb and allow the tissues to heal. If there is a fracture, pain attracts the attention to prevent any displacement of bones. However, in the foot with reduced pain sensation the discomfort may not be great enough to encourage protection (Fig. 13-5).

As the bones decalcify they are more liable to further trauma. Commonly the decalcified bones rub on each other till they literally fragment. *This is termed Neuropathic Bone Disintegration or Disorganisation (NBD).* Alternately, the softened bones impact during weightbearing and the body weight rests on relatively small localised osteopaenic areas, such as the talo-navicular or Lisfranc areas during take off (Fig. 13-6). This is still neuropathic bone *disintegration* but as fragments of bone are not seen some clinicians prefer to call it *disorganisation*.

Often this is called 'Charcot foot' which unfortunately, through tradition, usually conveys the message that treatment is not effective. This is a pity when it is just a neglected fracture that has not been given the chance to heal. Figure 13-7 shows a foot in which the bone fractures were in the cuneiform area. The bone has now deformed and partly disintegrated but will still heal, in whatever shape it is maintained in the cast, if immobilised for an adequate length of time. Neuropathic feet often feel cold but a fractured area will become warmer than normal. This shows that the blood supply is adequate, when needed, for tissue maintenance and repair.

Clinical Findings in Neuropathic Bone Disintegration

Clinically there may be very few findings in NBD. There may be swelling, heat and redness but usually no pain. The end result is often deformity but by that time the heat and swelling may have gone. A cool non-swollen foot may present with a deformity, that may be fixed or hypermobile, indicating that fracture and deformity has occurred, but that the bone
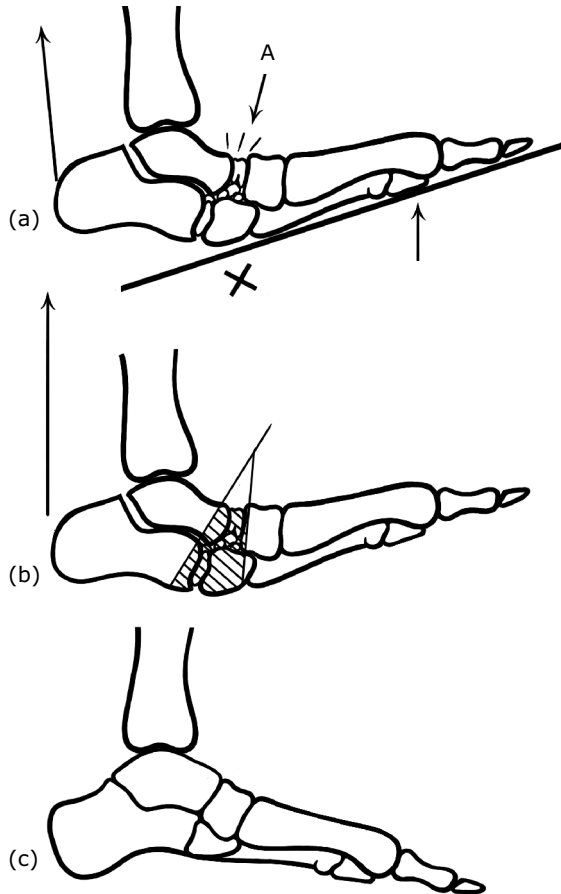

FIGURE 13-5 a. This hot, swollen, painless foot was diagnosed as an abscess. **b.** The radiograph showed it as a well-advanced disintegrating bone lesion. Attempt to drain an abscess was made before the first radiograph was taken. An advanced neuropathic disintegrating ankle joint on which the patient was walking without problems!

FIGURE 13-6 Diagrams to show how damaged bones on the dorsum of the foot may be impacted by the force of walking, producing a plantar deformity, or 'boat shaped' foot. **a.** The impacted navicular is indicated (A). The stress of walking has caused mid-foot flexion producing a boat-shaped foot. **b.** A wedge is removed as indicated by the broken lines and on fusing this has produced the functional foot shown in **c**.

is now healed and that it will not progress further. In many leprosy programmes radiographic examination is not available or affordable. Feet with NBD can be managed clinically, without radiographic control. If these lesions are treated with a Total Contact Cast (TCC) the patient can continue to walk while the fractures and any associated disintegration, or other tis-

FIGURE 13-7 A plantar bulge deformity frequently seen in diabetes. This is usually due to bone disintegration.

sue pathology, heals. If the patient continues to walk without a cast there is likely to be progressive disintegration and deformity. The hot spot usually occurs in association with some osteopaenia and to continue to walk on a hot swollen foot is asking for a stress fracture to occur.

If a (suspected) neuropathic foot has a persistent hot spot it should be treated as bone disintegration till proven otherwise.

The Stages in Neuropathic Bone Disintegration

There are three distinct presentations in NBD.

First stage: There may be a slight degree of heat and swelling. The patient may be unaware of a problem. A radiograph may sometimes show a fracture, a disintegrating bone lesion, or a stress fracture (Fig. 13-8, 13-9). These may only be visible 6-10 weeks following the 'traumatic' event. The situation may persist for weeks or months until the heat and swelling are more obvious or deformity and/or instability develops (Fig. 13-5).

Sometimes, after a lengthy period of heat and swelling, the radiograph may show areas of sclerosis and lucency but no obvious disintegration (Fig. 13-10). This patient had a fracture in the body of the talus, which was not recog-

FIGURE 13-8 Radiograph showing a fracture dislocation of the cuneiform area that would produce a similar external appearance to Fig. 13-7. Note bulge on plantar surface.

figure 13-9 Early disintegrating lesion of navicular; clinically only mild heat and swelling present.

nised. The patient was allowed to continue to walk, while given repeated doses of antibiotics. The bone slowly deformed and in a few months the talus had collapsed (Fig. 13-11). Treatment for 'osteomyelitis' was continued till 5 years later there was no ankle joint left (Fig. 13-12). Then it was reported that the patient had a 'Charcot joint' requiring amputation. However, the application of a TCC resulted in full bone healing.

Second stage: The foot has developed deformity and demonstrates signs of inflammation and ongoing destruction. The foot may become hypermobile. Marked disintegration and progressive deformity of the talus is shown in Figure 13-13a. Radiographic examination may show very osteopaenic bones. The decalcification may be gross – the skeleton appearing as a mere shadow of itself (Fig. 13-13, 13-14).

The deformity that occurs predisposes to local ulceration, which can result in infection. This is often a complication of the deformed neuropathic foot. Midfoot deformity and osteopaenia are shown in Fig. 13-15. It is difficult to tell how much of the deformity is the result of infection and how much due to osteopaenia and disintegration. Normal anato-

FIGURE 13-10 A hot swollen painless foot of a patient with diabetes. The swelling had persisted for two years. This radiograph shows increased sclerosis and lucency.

FIGURE 13-11 The same foot (Fig. 13-10) 18 months later after long treatment with antibiotics but no restriction on walking. The foot was still hot and swollen.
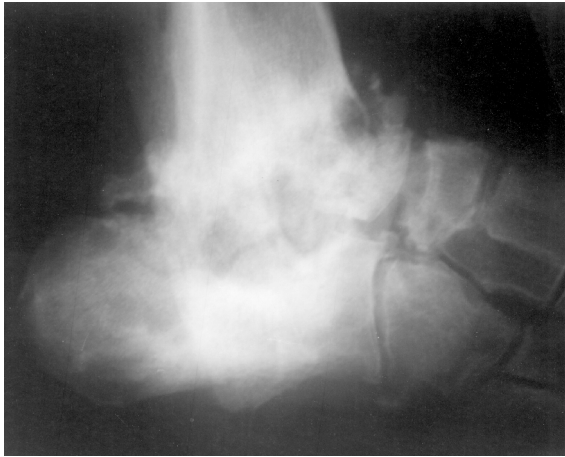
FIGURE 13-12 The same foot 5 years later.

my no longer exists, yet, a functional shape can still be achieved by adequate treatment. This second stage indicates that softened bone has been deformed and/or has disintegrated until the normal anatomy is no longer present. Along the fractured edges the bone will appear hazy (Fig. 13-13a). Sometimes the cortical bone is hazy and 'breaks' in continuity can be detected. Continued use will increase the bone destruction and deformity- but adequate immobilisation will result in healing in the

position the bones are held in the cast. Once the bones have fully healed they are usually sclerotic and strong enough to stand up to the normal stresses of daily use (Fig. 13-16).

Third Stage: consolidated stage. The foot will no longer be warm or hot. It will, however, often be cold due to neuropathy, and may be deformed. The bone edges will once more be clear cut lines as seen in Fig. 13-16. It may have consolidated in a position that is basically normal (Fig. 13-17). There may be some degree of deformity that is compatible with normal usage (Fig. 13-18). It may be unstable and/or hypermobile. Radiographic examination will often show that the bones have recalcified (Figs. 13-14b, 13-15b). The fractures may be healed in a deformed position or, if not fused, the bone edges may show firm lines indicating healed bone, compared to earlier haziness and irregularity. If there is deformity the foot may re-ulcerate and secondary infection may determine the final outcome. The foot that initially had only a minimal fracture, neglected because of *lack of pain*, may become increasingly deformed, and a chronic liability.
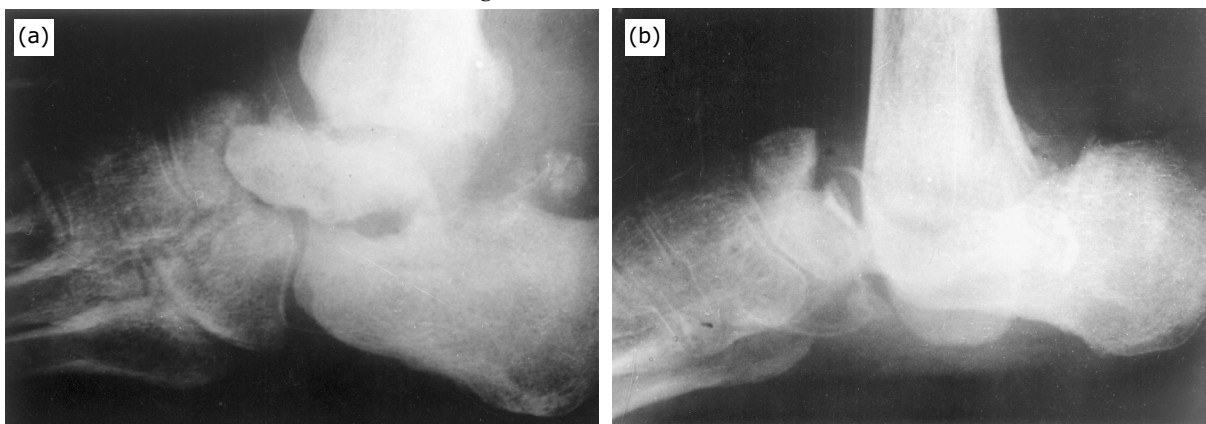

FIGURE 13-13 a. The disintegration of the talus resulted from a minor trauma. **b.** Without treatment it progressed till the tibia had gone through the calcaneus and the foot was amputated.
The foot was not treated because it had been stated that a 'Charcot' foot could not be "cured".
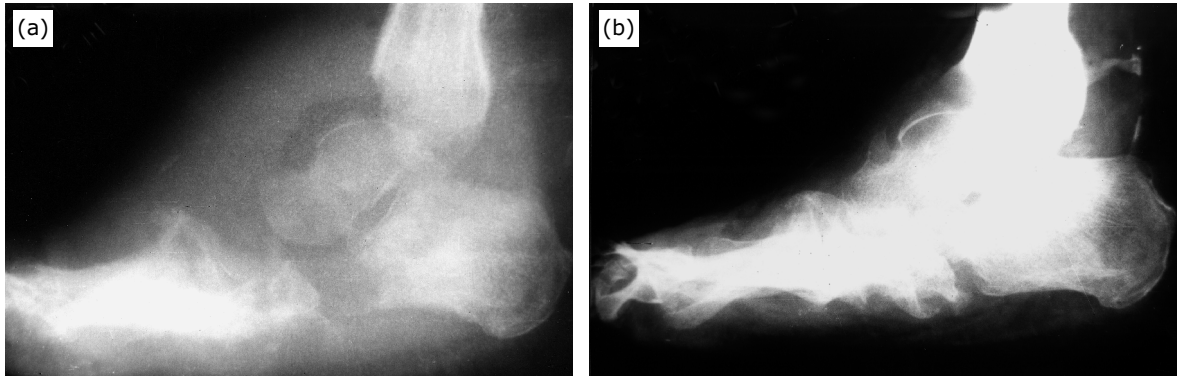
FIGRE 13-14 a. Markedly osteopaenic bones but no obvious infection. The bones went with to re-calcification on adequate rest and protection. **b.** Twelve months later showing the recalcified bones.
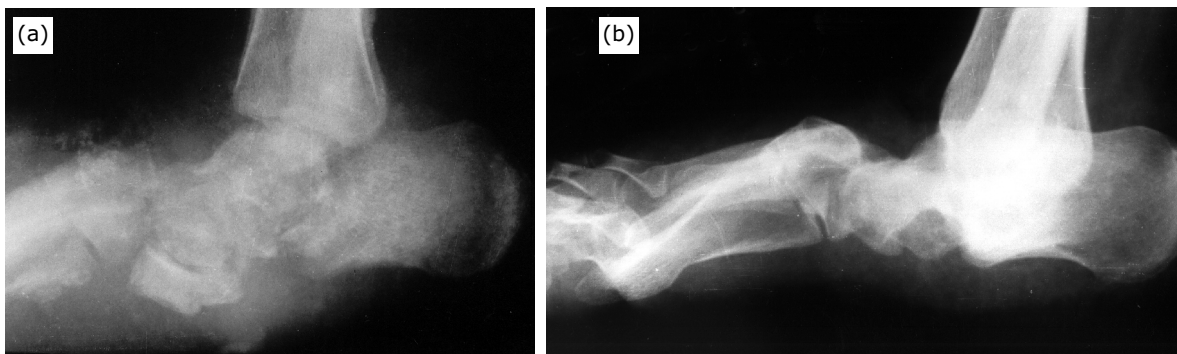

FIGURE 13-15 a. Osteopaenic disintegrating bone with marked midfoot deformity. There is obvious infection that has gained entry through ulcer sites. **b.** Twelve months later the bone is sclerotic and healed.

FIGURE 13-16 Healed bones. Same foot as in Figure 13-5, after 12 months of healing in a TCC. Note well-calcified and firm edges of all bones.

FIGURE 13-17 Healed navicular bone after 8 months in plaster cast. Same foot as in Figure 13-9.
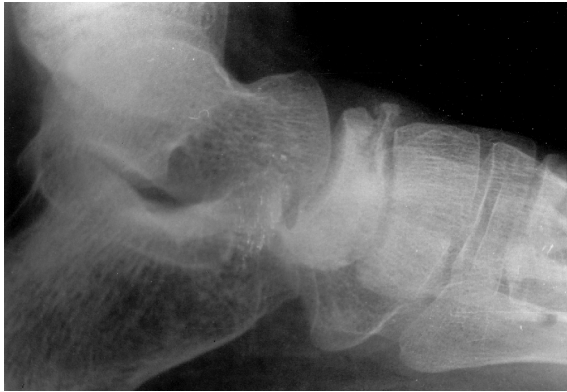
Figure 13-18 Slightly deformed navicular that is obviously healing without requiring any special therapy.

Diagnosis of Neuropathic Bone Disintegration

"Hot Spot"

A hot spot in a sensory impaired foot should be regarded as a sign of a bone lesion, until proven otherwise. It may be a fracture that, if neglected, may develop into a so-called 'Charcot' joint or foot. The term *neuropathic bone disintegration* (NBD) is preferable, as often the joints are not involved, at least initially. It is rare to see a typical 'Charcot' foot as originally described by Charcot (Fig. 13-5).

Hot spots may also be due to:

1. Minor infections.
2. Sprains and strains.
3. Burns, including friction burns on the feet.
4. A recent fracture, which has not yet disintegrated. Stress fractures may not be visible on radiographs for 6-8 weeks and are often missed. Such fractures should heal as well as a fracture in a sensate foot if it is immobilised for an adequate length of time.

The patient with neuropathy and reduced pain perception does not realise the presence of a fracture and continues weight bearing. Movement of the broken bones causes fragments to be rubbed off the bones until disintegration or disorganisation becomes obvious.

The search for the cause of the hot spot is often assisted by white cell counts, full blood counts and ESR tests. These tests are sometimes supplemented by radiological scans, which will confirm the hot spot but are not informative about the cause. A scan confirms what your hand has already told you. It may say which tissues are hyperaemic but not why. Hetherington discusses tests to determine the cause of the hyperaemia but warns about relying too greatly on such tests. He recommends a bone biopsy. However, if the skin is intact a biopsy may often be negative, possibly because the wrong tissue is taken. If there is a hot spot in a neuropathic foot without an ulcer it is unlikely to be septic osteomyelitis. For diagnosis, therefore, radiography is not essential, not reliable and in many places not available or affordable.

Clinical testing for hot spots is the most effective and cheapest.

Most 'hot spots' are distal to the ankle. The patient is given complete rest of the affected part- preferably complete bed rest. The foot is elevated and rested in a splint that will prevent movement of the ankle and toes. If the heat and swelling are due to a fracture they will settle in a few days but rapidly return as soon as the patient resumes walking. If the heat and swelling are due to infection they will not subside quickly unless the patient is given antibiotics. Once settled it should not return quickly when walking is resumed. When in doubt, especially in a patient with marked neuropathy and/or diabetes, it is best to give appropriate antibiotics for a prolonged period, rest till the swelling has subsided and then use a Total Contact Cast (TCC) for 6 to 12 weeks (appendix D). At that time any fracture should be seen on

an X-ray, and may already have healed without displacement. Any infection will probably be completely eliminated, especially if antibiotics have been given.

Radiography

Ideally every neuropathic foot should, for future reference, have routine radiographs taken at diagnosis.[26,27] The best radiographic views are:

A. True standing lateral from toes to heel (Fig. 13-19).

B. Antero-posterior view of the forefoot taken with 15 degrees of obliquity from the verti-cal. (APO view; Fig. 13-20b). This will show the metatarsal bases more clearly than the standard AP view (Fig. 13-20a).

These two views are cost effective and if radiographs are routinely taken in these positions it will facilitate comparison when a lesion is suspected. Any suspect lesions should be further investigated by other views if necessary.

Early signs of bone lesions may include chips, cracks (Fig. 13-21), haziness (Fig. 13-22), obvious disintegration and lucidity (Fig. 13-10), increased calcification (sclerosis), impaction (Fig. 13-18) and decalcification (Fig. 13-14a). Impaction may be associated with loose frag-
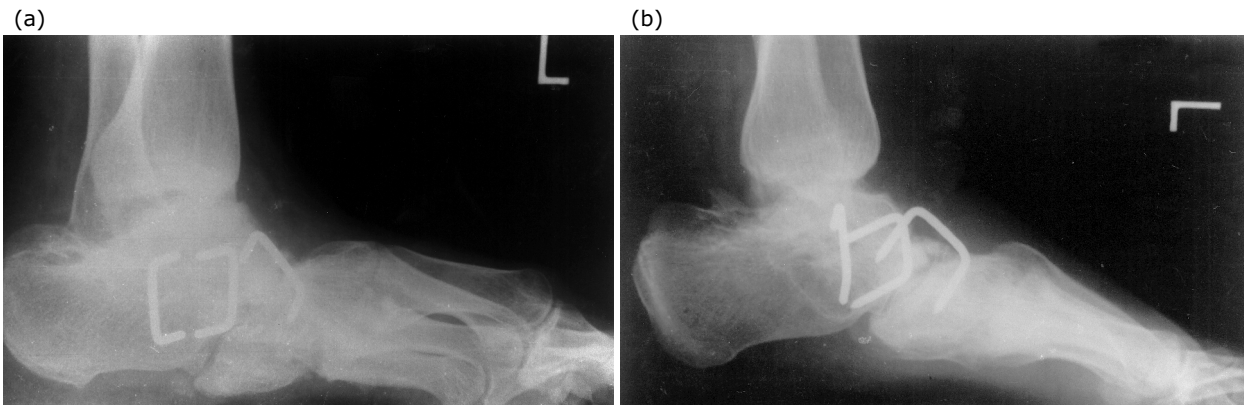
(a)   (b)

FIGURE 13-19 a. Standing lateral view. **b.** Compare with non-standing lateral view of the same foot which may give a wrong impression of stability.
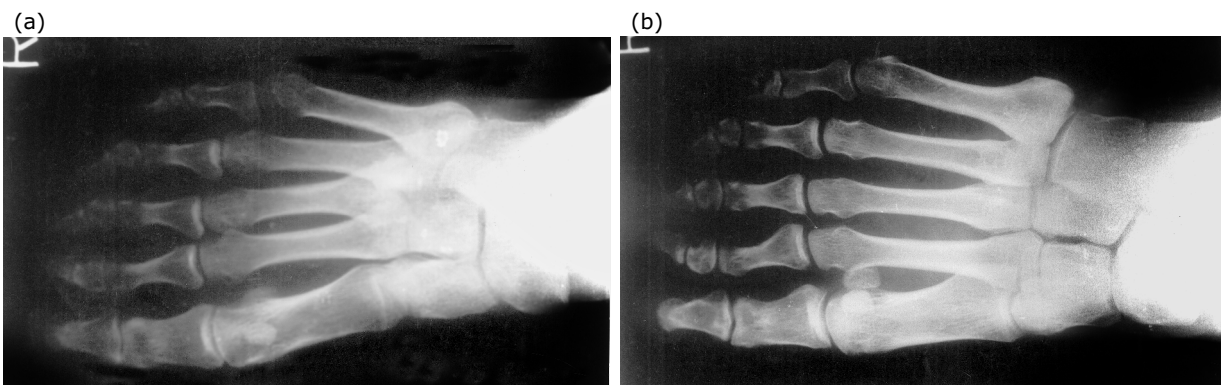
(a)   (b)

FIGURE 13-20 a. Standard AP view. **b.** Oblique (APO) view that gives a much better view of the bases of the metatarsals.

FIGURE 13-21 Large crack through navicular.

FIGURE 13-22 Mid-tarsal fracture showing haziness and disintegration along the fracture line.

ments of extruded bone lying free beside damaged bone (Fig. 13-23). There may be obvious fractures with disintegration (Fig. 13-9), fragmentation and general haziness of the bone edges (Fig. 13-13a), loss of trabeculae and irregularity of cortical bone. The foot in which the radiograph suggests abnormality should, if possible, be further examined by different radiographic projections. A neuropathic limb that shows a suspicious change on a radiographic examination should be treated as if there is NBD. A radiologist who knows that the patient has a neuropathic foot may report any bone lesion as a 'Charcot' joint. The most common early lesions are simple fractures and if the limb is adequately immobilised these

Figure 13-23 The head of the talus has been fractured, impacted, and partially displaced. The cortex of the navicular is irregular and may be disintegrating.

should heal as well as they would in a sensate foot. When the lesions have healed, their edges will appear clean cut and no longer hazy or fuzzy. NBD usually starts as a fracture, not a joint lesion. The joint may be involved when a fracture extends into the joint or when the subarticular cartilage collapses.

Other Factors Affecting the Development of Neuropathic Bone Disintegration

Harris and Brand presented a new concept on the basis of analysing the patterns of NBD lesions, and stated that lesions will heal if adequately protected.[13] Before that time it was believed that the neuropathy was the cause of NBD. Brand summarised their findings by saying: "We have been looking for weakness of the tissues when we should have been looking outward to prevent excessive forces and high temperatures". Delbridge also stresses the mechanical factors that predispose to ulceration in patients with diabetes.[10]

Brand states that although leprosy may render an insensate foot more vulnerable to mechanical forces, the denervated tissues are only marginally less competent in wound healing. The 'misuse' of the limb due to reduced pain perception is the main factor in prevention of healing and hence the development of the so-called non-healing ulcer and 'Charcot'

foot. If special care is given, the anaesthetic limb need not develop perforating ulcers and loss of digits. The basic problem is one of mechanics, not medicine. The principles of care for insensate feet in leprosy are equally applicable to other neuropathies such as diabetes, familial motor sensory neuropathy and spina bifida.[3,23] Coleman and Brand discuss the acute care of neuropathic feet including osteomyelitis and fractures.[8] They point out that the key to treating the fractures is early diagnosis. They emphasise the use of the TCC, and the extra time required for healing of neuropathic bones.

Hetherington states that there are definite osseous changes associated with neuropathy and that these include radiographic osteoporosis, atrophy, destruction and disappearance of bone structure. It has been observed that the neuropathic foot with chronic ulceration becomes more and more osteoporotic due largely to the hyperaemia, which itself causes increasing osteoporosis. Osteoporotic bone is more prone to stress fractures. One study showed that 10% of patients who had a walking cast for 6 weeks or who were otherwise immobilised developed a stress fracture if allowed to resume unrestricted walking at the end of this time.[25]

Borssen recommends the use of plaster casts in treating neuropathic diabetic foot lesions.[6]

TCC's are excellent for healing neuropathic, ulcer or bone, lesions. Shaw has shown that the weight is spread over the foot with some 40% being carried through the inner shell of the cast.[22] This means that the pressures on the leg and foot are relatively low. Movement inside the cast is eliminated so any tissue trauma, bone or soft tissue has a chance to heal without being constantly disrupted by movement.

However, many clinicians, especially diabetiologists are very hesitant to leave a neuropathic limb in a TCC. They feel that the risk of pressure ulcers and necrosis within the cast outweighs the advantages of the casts. I have great success treating ulceration of diabetic feet with TCC, especially when it is associated with bone involvement. This has resulted in prevention of amputation in many patients with diabetes.[11,17,19,21] Because of the reticence of some clinicians for a TCC a modified *bivalved TCC* has been devised. This cast can be removed to check the skin for rubs or pressure areas and to treat any ulceration that is present. It is replaced with Velcro and therefore is still virtually total contact when correctly applied (Appendix D). The main problem with its use is compliance. Patients have to learn that they do not take the cast off, except when instructed by the physician. By using a TCC, an ulcer of about 2-3 cm will usually heal within 6 weeks. Where there is a bone lesion the time will be much longer. When the cast is discarded it is important to ensure that trial walking (graded weight bearing) is started (Appendix E). Ulceration has been included here because of its intimate association with the development of osteoporosis and the complications that may result from it.

Osteopaenia and Osteoporosis

The degree of osteopaenia is one of the main factors in the development of NBD. Osteoporosis is not uncommon, especially in the older age group in whom stress fractures frequently occur. There are other factors that are important in the aetiology and management of NBD.

1. Acute neuropathy in itself may lead to osteoporosis. This may happen in Type I leprosy reactions. Osteoporosis can predispose to a stress fracture, especially in patients with an unstable gait.
2. Reduced activity such as prolonged bed rest. The use of a TCC instead of bed rest for foot ulceration helps reduce the severity of osteopaenia.
3. The use of plaster casts. One study reports that 10% of patients with neuropathy, who wore a walking plaster cast

for 6 weeks or longer, developed a stress fracture when allowed to return to unrestricted walking the day the cast was removed.[25]

4. The use of corticosteroids.
5. Poor nutrition, especially in combination with hormonal factors, or in growing children.
6. Cigarette smoking and excessive use of alcohol.
7. Osteoporosis is also found when there is long standing hyperaemia, such as occurs in chronic infection, fracture or trauma (Fig. 13-14).
8. Age. The development of osteoporosis in older age groups is well accepted.

Initially the stress fracture does not show on an X-ray. As a first step towards healing the body removes the calcium from the edges of the fracture until it becomes possible to visualise the fracture on an X-ray. After 6-8 weeks it is usually possible to see a stress fracture. The fracture makes the bones initially softer and they are therefore more prone to deformity. The edges of the bones may 'impact' during walking. If the area is mobile the edges may wear each other away. Heat and swelling persist until the bones are united, that is when removal of calcium ceases and deposition of new bone and calcium occur. This may take several months of immobilisation. If a decalcified limb is not adequately protected it may continue to exhibit a low degree of activity for many months slowly changing its shape while adapting to the stress of use. The patient should not undertake unprotected walking when there is still heat or swelling. All patients should undergo trial walking when returning to unprotected walking (Appendix E).

Once osteoporosis is present it is not likely for the bone density to return to the pre-trauma level without medication. Immobilisation in a TCC does cause osteopaenia but such immobilisation is essential for bone healing. At best, healing may occur in association with increasing deformity. Once healed the patient can walk without the cast, in suitable footwear and this will encourage further re-calcification provided the amount of walking is 'guarded'.

Management of Neuropathic Bone Disintegration

If a disintegrating bone is not immobilised, the disintegration may continue (Fig. 13-13) but if adequately immobilised healing will occur. Healing can occur spontaneously in lesions that are not subject to marked movement. If the bone edges are already well defined at diagnosis it is unlikely that fusion of the fragments will occur. Even with lesions that are grossly decalcified or disintegrated, healing is possible, though in severe cases it may require 12 months or more, of total immobilisation. (Figs. 13-14, 15)

Disintegrating bone requires immobilisation for a prolonged period to ensure that healing will be complete. There is no reason to believe that neuropathic bone takes longer to heal than bone that is innervated. However, bone with reduced sensory perception is more prone to injury than normal bone. If normally sensate newly healed bone is subject to excess stress, pain will protect the foot. If pain perception is reduced the patient may not be warned of impending trauma and continues 'stressing' the healing bone until a new fracture may occur. Hence, the rule that a neuropathic bone lesion is immobilised for 2-3 times as long as would be required for the same lesion in a sensate foot.

Basic Routine Management of NBD

Following examination and recording:
1. Rest in a splint, foot elevated. Not one step! Complete bed rest if possible. Foot elevated on pillows or frame with the

knee bent. Many patients already have weak dorsiflexion and shortening of the Achilles tendon is NOT wanted. Advise the patient about toileting etc. One step on the foot may nullify the good done by the previous 24 hours.

2. After 5-7 days, earlier if all swelling has subsided, check mobility and, if mobile, mould foot into optimal functional position.

3. Apply Total Contact Cast (TCC) to maintain optimum position. (See appendix D)

4. After 3-6 weeks, if the cast becomes loose, remould the foot and replace the cast.

5. Patient should be encouraged to walk in the cast as this helps to stimulate recalcification. It also helps to maintain the strength of the muscles of the affected leg.

6. Immobilise for full length of time according to schedule.

Initially, review the cast weekly. Some choose to delay the first complete cast, (during which time the patient does not walk on the foot), for about a month in the hope that all swelling will have subsided before the cast is applied. Then there will be no need of a new cast in a few weeks. If this is done the foot should be adequately splinted so the ankle and toes are not easily moved or bumped. When the diagnosis of bone disintegration is not certain at cast application, it is advisable to re X-ray the foot about 8 weeks after the initial cast is applied.

The bones will heal in the position in which they are immobilised. Therefore, an attempt should be made to manually reshape the foot before the TCC is applied. It is not advisable to try to mould the foot into a functional position once the first layers of plaster of Paris are applied. When a better position is achieved by moulding, and the TCC is applied, excessive pressures should not result. If the foot is moulded after cast application there are likely to be pressure points where ulceration may occur. Many clinicians working with neuropathic feet of patients with diabetes are reluctant to apply a TCC due to fear of pressure sores. This fear is unwarranted provided proper cast application has been done and the patient is well informed.

By careful application of TCC it is often possible to obtain a functional foot without the need of surgery. In some patients the initial degree of deformity is so great - or increases as the swelling subsides that the healed position is not functional to ensure ulcer free walking. In these patients some form of wedge osteotomy, or other bony surgery, may be indicated to restore the foot to a shape that is functional and near normal. The object of the reshaping of the foot is to produce a functional unit, not to restore original anatomy. When a patient presents with a grossly osteopaenic deformed bone try and mould it to a better shape and then after 3-4 months in a plaster cast review with an X-ray. If the bone is no longer disintegrating and showing defined, albeit osteopaenic bone but the anatomical shape is obviously incompatible with ulcer free walking, surgery should be contemplated (Figs. 13-14b, 13-15b, 13-16, 13-17).

Healing Time for NBD

The time required for healing depends on the site and extent of the bone lesion(s). The times suggested below were determined by 'trial and error' and use of trial walking on a large group of leprosy patients over a 15-year period.

1. The *patient who presents early, with a hot swollen foot in whom there is no evidence of bone lesions* on the radiograph should wear a TCC for 6-8 weeks. A new X-ray should be taken at that time. If there was a stress fracture the bone lesion should then be visible. If there was no lesion, start trial walking to test if there is other

pathology. Immobilise for 3-4 months if no X-ray can be made before reviewing the clinical condition. The patient should not continue trial walking (TW) if the foot repeatedly becomes warm and swollen.

2. Patients *who show minimal fractures (excl. tarsal bones) without obvious disintegration* require 3-4 months in TCC. Phalangeal fractures or disintegration of metatarsal heads or phalanxes need at least 3 months in the cast.

3. Patients with *major fractures of tarsal bones but no disintegration need 5-6 months in TCC.* If in doubt *give 7-9 months TCC.* If only mild disintegration but no gross fracture or marked displacement, trial walking can be started in 6-8 months.

4. Patients with definite mid-foot fracture/disintegration require 8-9 months. Most lesions occur in the talo-navicular-cuneiform area.[25] The Lisfranc area also requires 8-9 months immobilisation.

5. Metatarsal osteotomies or disintegration need 6-9 months. If they involve the Lisfranc area they need 8-9 months.

6. Gross disintegration involving many bones or large areas may need 12-18 months in a TCC.

If the patient with NBD requires tendon transfer these can be done towards the end of the bone healing time. This is 4-6 weeks prior to ending the TCC. However, return to locomotion by TW and a split TCC should be slower than normal for a tendon transfer.

Many have used callipers, braces and moulded footwear to encourage healing of NBD. This may be effective in minor lesions. However, if the appliance allows any movement it is unlikely that the optimum functional position will be achieved. Healing will be slower than if completely immobilised in a cast.

RECONSTRUCTION OF THE DEFORMED NEUROPATHIC FOOT

Because of reduced pain perception many NBD lesions result in deformity that may not be compatible with ulcer free usage. Minor deformities can be 'accommodated' in footwear or with orthotic appliances to prevent ulcers. However, the provision of footwear-devices is not practical, and costly for the patient. Walking without shoes, even for a short period may undo all the good achieved by special footwear. In addition other questions are relevant. Where does the patient bathe? In the river? Does he need to remove the shoes for this? If he has to take off his shoes for these reasons then these are valid reasons to consider reconstruction of the foot in such a way that barefoot walking for short periods is tolerated.

The Badly Deformed and/or Ulcerated Foot

Some patients have had a dropped foot for many years and the lateral border is 'worn away' through recurrent ulceration. If a foot drop correction is not appropriate and it is not possible to passively place the ankle in a functional position it may be best to do an ankle arthrodesis (Fig. 13-24). Patients can walk well when the ankle is arthrodesed if there is adequate dorsiflexion (5-10 degrees). If there is too much dorsiflexion the patient may need to stand with the knees slightly bent to get the soles flat on the ground. This is not a real disability and often improves stability as extensor and flexor muscles of the hips and knees are firing all the time. The ankle arthrodesis may be combined with a midtarsal wedge if there is rotation of the midfoot or turning in of the forefoot. Alternately, a modified triple arthrodesis may correct the basic deformities of the hind and midfoot but this will not provide ankle stability if a foot drop is present.

FIGURE 13-24 Pan-talar fusion of grossly deformed ankle.

There are also patients with a fixed inversion of the heel in whom a foot drop correction will not produce a good result. These patients do well if a sub-talar fusion is combined with a tendon transfer to correct the foot drop.

For patients with marked deformities it is often more effective to reconstruct the foot so that special footwear or orthotic devices are not needed. Patients can then use regular footwear with a microcellular insole to provide extra resilience. The surgical alternative is often major surgery, requiring good hospital facilities and prolonged periods in a TCC. When the cost of such procedure is compared with that of providing a new pair of shoes every 6-12 months for a lifetime, then costs favour a reconstruction. Years ago it was generally accepted that healing after reconstruction of neuropathic bones would not occur. However, it has now

been shown that it does occur.[24] Banks has reviewed various techniques but emphasised the need for adequate fixation.[1]

Osteo-Ectomies

After neuropathic bone has healed there will be some degree of deformity. In many patients this is a slight deformity and compatible with function especially if the patient wears suitable footwear. A common deformity is a prominence on the planto-medial surface of the longitudinal arch, usually at the talo-navicular joint or the Lisfranc area (Fig. 13-8). This 'bump' often ulcerates. It is possible to do a 'bumpectomy' (an osteo-ectomy) to remove the prominence but it is essential to know that adequate 'healthy' bone is present (Fig. 13-25).

An oste-ectomy for removal of a deformity resulting from major disintegration is best done after about 6-7 months in a TCC. By that time the position is stabilised and handling the foot on the operation table is not likely to increase the deformity. Bone should be ossified enough to be able to assess where trimming is necessary. The 'bump' is usually approached from the lateral or medial side of the foot. The incision is made on, or a little above the level of the plantar surface of the bone. The plantar tis-

FIGURE 13-25 a. Typical plantar ulcer caused by a deformed cuboid. **b.** Radiograph of the same foot.

sues are peeled back at the periosteal level and the 'bump' is chiselled off and rasped smooth. If possible, saucerise the area so that the actual bump becomes a depression roughly the shape of a saucer, created under the old scar so that the patient's weight is carried mainly on the edge of the saucerised area. The scar will then not be subjected to as much pressure as would happen if the bone were trimmed flat. Be careful to remove any rough or irregular periosteum and bone fragments as these may otherwise recalcify and cause new problems. Do not plan the 'bumpectomy' through a plantar ulcer, as that will require a much larger incision and will leave a poor scar line. After removal, the scar should be closed with everting deep mattress sutures of large (0 or 2x0) monofilament nylon to produce a good quality surgical scar. The surgical approach scar is left open except for one or two mattress sutures at each end, as a relieving incision, that provides drainage, can be packed open and allowed to heal by secondary intention. If there has been an ulceration that has left poor quality skin it is advisable to excise that skin, by an elliptical incision, at the same time as the bump is flattened. In that case the incision to approach the 'bump' should be dorsally, high enough to ensure survival of the bridge of tissue left between the definitive surgical incision and the original scar (ulcer).

Following surgery the patient can start walking in 5-14 days, using a bivalved TCC. A windowed cast is not recommended if the wound is on a weight-bearing surface. Scars on the weight-bearing surface should be avoided except when:

a) There is poor quality skin on the weight-bearing surface that is likely to break down again. The scar can be excised and closed to leave better quality skin. The cavity left after removal of the bump is loosely packed with a layer of Vaseline gauze padded out by saline soaked gauze. The relieving incision should be long. This pack is not changed for 5-6 days, when it will be determined if there is any residual infection. The pack can be changed once or twice a week till the wound has fully healed from the bottom. The granulation tissue will help to increase the depth of plantar tissue. The patient should be on bed rest, in a cast or back slab, for the first week or two and then may have a bivalved TCC which can be removed, usually weekly, for dressings. Do not allow the skin edges of such a cavity to heal before the bottom is fully granulated. Do not allow the cut edges to invert so that the wound heals as a deep crack.

b) a periosteal irregularity on the plantar surface that keeps causing ulceration. These can often be managed by orthotics and regular removal of callosities but for long-term care it is often better to do a 'bone trimming'.[9]

Sometimes a roughened metatarsal head that is prominent is best approached through the dorsum. Let the wound heal by secondary intention. It is advisable to excise the scar that was under the head and close it with deep mattress sutures to improve the quality of skin. The small decrease in plantar weight bearing area is offset by improved quality of skin. This is especially the case if it is possible to bring the toe(s) down. In some patients it will be an advantage to shorten the prominent head so that no one head is taking excessive stress during take off. Ideally, all the metatarsal heads ought to be in a curved or straight line so that no one is prominent.

After these procedures the patient should continue to use the bivalved cast for a total of

6-10 weeks or two full weeks after all the scabs on the long lateral incision have come off by themselves (which ever is the longer period). There is no practical way of assessing the healing of deeper tissues. If bone has been cut or trimmed it may be better to use the cast for 3-4 months especially if the bone was osteoporotic or infected before surgery. Radiographs will not indicate if healing is complete. Once the patient goes on to trial walking he will require another 2-4 weeks of guarded walking. The patient should keep the cast and use it when 'at risk' e.g. on trips where the foot will be in use for a long period. The cast should be worn if heat and swelling occurs.

WEDGE OSTEOTOMIES & ARTHRODESES

For a marked deformity moulded footwear is often recommended. However, it is possible to reconstruct the foot by wedge osteotomies or an arthrodesis that could make the foot less prone to 'break-down' and the patient less dependent on custom made footwear. The common osteotomies or arthrodeses are in the sub-talar, ankle, midtarsal or metatarso-tarsal areas (Fig. 13-26 a-c). A flat foot is perfectly acceptable as long as the patient does not have plantar prominences, or hyperextended clawed toes that can be corrected by Girdlestone tendon transfers (Chapter 14).

In some patients with flat feet it is possible to do one or more small wedges e.g. talo-navicular or naviculo-cuneiform to correct over pronation. Pandy's osteotomy is a helpful procedure to correct over pronation.[18] The plantar part of the calcaneum is shifted laterally to assist in preventing inversion of the calcaneum. If necessary, the forefoot can be rotated on the hind foot. It is usually more practical to do one big wedge osteotomy across the midfoot and use internal fixation to ensure the position is maintained. With good internal fixation the patient can walk in a TCC 10-14 days after surgery, when the post-operative swelling has subsided. I would not bivalve this cast, especially if the patient is going home. I do not recommend Charnleys clamps or similar external fixateurs because their removal means that
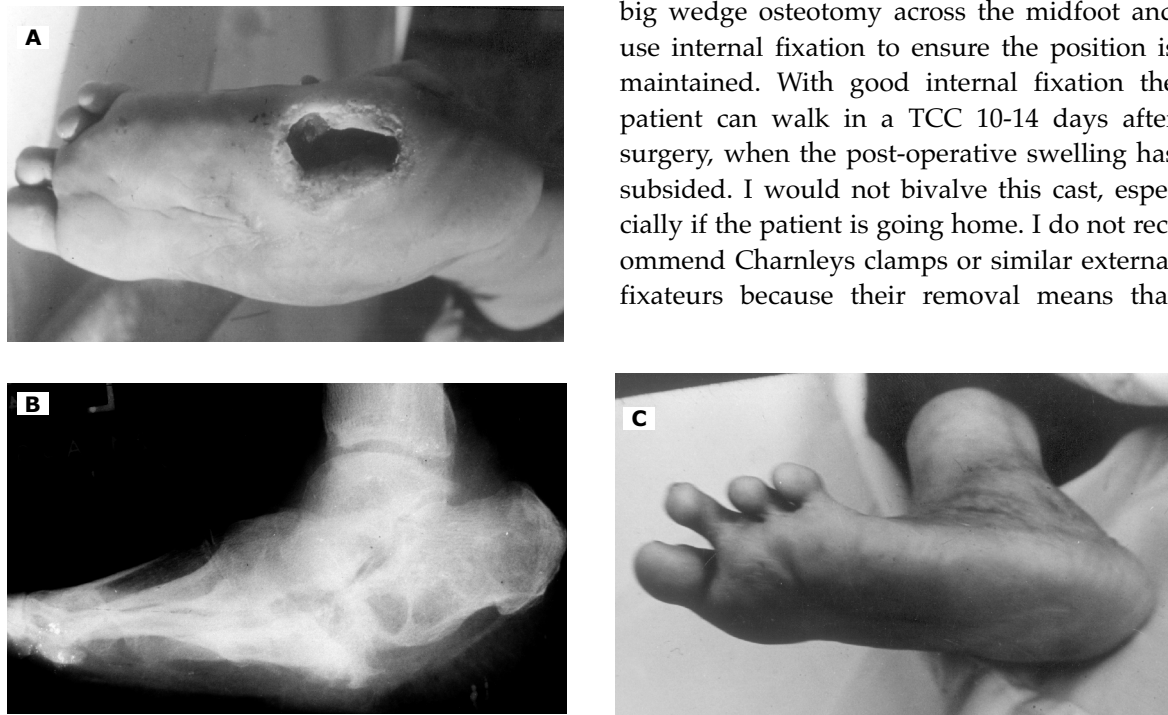
FIGURE 13-26 a. A chronic ulcer present for over 5 years. **b.** The radiograph indicating there had been neuropathic bone disintegration with impaction that had caused a boat foot. **c.** The sole 5 years after wedge osteotomy to remove the bony prominence of the sole.

there is no longer internal fixation maintaining the position. Even when covered by casting material there is an increased risk of soft tissue infection or osteomyelitis.

Wedge osteotomies can be done to reshape a badly deformed foot after NBD once the bone appears to be healing. This could be at 3-6 months after TCC. If a wedge osteotomy is attempted early, in the acute stage of the disintegration, the bone is so osteopaenic it can be cut with a scalpel and any fixation will not hold. Some have tried to insert bone grafts to restore the foot to normal size. This is effective only if a small area is involved and the foot is maintained in a TCC for 9-12 months. However, it is usually safer to slightly reduce the plantar area of the foot and fuse without a graft as the addition of a graft may cause excessive stretch on the already damaged dorsal skin. After the osteotomy the TCC is used for a further 7-9 months before trial walking is used to test the integrity of the structures. If the toes are clawed, or not making good ground contact, they should also be corrected before trial walking commences.

Mid Tarsal Wedge Osteotomy

A wedge osteotomy of the midfoot can correct a boat shaped foot, a medial displacement of the forefoot or other obvious deformity of the plantar surface (Fig. 13-6). The external shape of the foot has to be considered and the type of wedging, size and direction determined, bearing in mind that the internal anatomy may be completely abnormal. There will be no arthrodesis of existing joints. Following severe NBD there may be complete loss of all joints and sometimes the whole foot fuses into a solid structure.

Technique

The approach is through an incision that curves up from the cubo-calcaneal joint area across the dorsum to about mid dorsum. Sometimes a second incision is advisable on the medial side over the talo-navicular area.

The most common wedges are transverse through the talo-navicular, naviculo-cuneiform, or through the Lisfranc area with the widest part of the wedge plantar grade to re-create an arch.

The osteotomies are best fixed with long crossed screws, or K-wires, that extend from the base of the first and the fourth metatarsals so the heads can be counter sunk but the ends of the screws reach to the cortex of the calcaneum. Blount's staples can also be used. This type of wedge will be subject to great stresses during weight bearing and postoperatively the TCC must be well moulded to fit the new arch for at least 8-9 months (Fig. 13-27). The principle behind the arthrodesis or osteotomy is that the patient can go straight into normal, or custom-made, footwear, eliminating the need for orthopaedic appliances.

Ankle Arthrodesis

In the deformed, often plantar flexed ankle it is convenient to perform an ankle arthrodesis

FIGURE 13-27 Post midtarsal wedge osteotomy. Patient walked without protection before healing was complete. This caused the wire to bend with resultant loss of some position.

through a transfibular approach (Fig. 13-28). Elevation of the lower part of the fibula provides an excellent view of the ankle joint. By fixing the fibula to the tibia and talus and/or calcaneum there is no need to use extra bone chips. An alternative approach is anterior,

either mid-ankle or with an incision on each side just anterior to the malleolus. With both of these approaches it may be difficult to remove the cartilage from the inside of the malleoli and certainly in the plantar flexed foot it is difficult to free the posterior surface to obtain good dorsiflexion and remove enough cartilage. If the talus is deformed it may be very difficult to get good opposing surfaces. If a bone graft needs to be inserted, then a midline anterior approach may be easiest. It is probably better to arthrodese without a graft. With the transfibular approach it is easy to obtain good apposition and fixation by wires and screws.

Technique

For an ankle fusion I recommend the transfibular approach. The surgery is easiest if the patient is lying on his side with the ankle to be
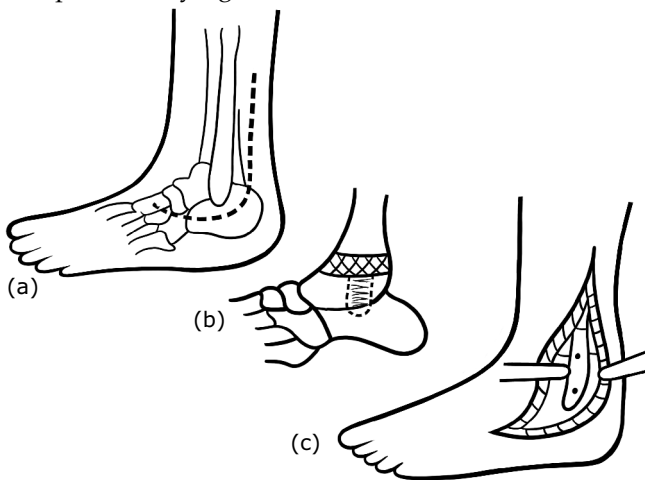

FIGURE 13-28 Diagrams of the transfibular approach to an ankle fusion.
a. Line of skin incision. **b.** The area to be removed is cross-hatched. Lines mark the site where the fibula will be replaced. **c.** The finished fusion. The fibula is held with two screws; one may be adequate or none needed.

operated on uppermost and the other leg flexed at the knee and hip. A sandbag can be placed under the upper leg. A tourniquet on the thigh is advisable.

The incision is made just behind the fibula starting about 10 cm above the lower end of the fibula and turning at the lower pole of the fibula to run parallel to the sole for a further 10 cm. This will allow a flap to be raised. The tendon sheath of the peroneal muscles may need to be opened to give a good approach to the back of the fibula. A gigli saw- or electric saw is used to section the fibula about 8 cm up its shaft. A chisel is then inserted behind the lower end of the fibula to free the fibula from the tibia. The fibula is then lifted forward still attached to the skin and deep tissues to expose the ankle joint. This exposure gives excellent access to the ankle in any degree of dorsi-plantar flexion. It allows section of the posterior capsule if that is needed and lengthening or sectioning of the Achilles tendon. It also allows removal of the posterior cartilage of the talus and tibia easier than by the standard anterior approach. The operator can easily determine the cuts needed to put the foot in a suitable slightly dorsiflexed position. It is always better to provide slight dorsiflexion. Walking with a dorsi-flexed foot is preferred to walking with a foot in plantar flexion. The cartilage of the tibial surface of the ankle joint is removed and then the talus is trimmed to fit the space. If necessary, part of the medial malleolus can be removed to wedge the upper section of the remains of the talus into the tibial malleolus. If the foot is badly deformed it may be advisable to do a subtalar fusion at the same time or a pantalar fusion. Using this approach it is easy to use Blount's staples, tibio-talar and talo-calcaneal to hold the bones in the desired position. Alternately, screws or K-wires can be used but they require separate incisions for insertion. If there is difficulty holding a position, a Steinmans pin or 2.0-mm K-wire can be inserted vertically through the calcaneum into the tibia. Once the ankle is in a satisfactory position the fibula is split by removal of the cortex so that there is cancellous bone adjacent to the tibia. The tibia

is also roughened and the fibula placed as an onlay graft and held by staples, K-wires or screws to the talus or calcaneum. This provides a good lateral strut to help ensure stability of the arthrodesis. Bone chips are not needed if the talus has been cut but could be added either before or after the fibula is attached. If pieces from the fibula are used to add chips make sure it is cancellous and not cortical bone. A drain is inserted into the main cavity and the skin closed with interrupted everting mattress sutures. A plaster is applied to hold the foot in the desired position without undue pressure on the incision area.

Remember that the neuropathic patient may not have any real pain sensation and so it is important to ensure the new arthrodesis is not stressed before the TCC is made. Post operatively it is best to insist on complete bed rest-no toilet privileges for 3 full days till most of the swelling has subsided. The drain can be removed about 48 hours after surgery. A walking TCC can be made at day 6 and the patient can be discharged at day 10. If nylon sutures are used there is no need to remove them before the cast comes off. The plaster can be changed, when it becomes loose. The cast will be on for 6 full months and then bi-valved when trial walking is started. Because of the fibula overlay with internal fixation there is usually excellent lateral stability. There is no need to radiograph the foot. Radiography will never tell if the fusion is complete. That must be tested clinically by trial walking.

Subtalar Wedge Osteotomy

A wedge osteotomy of the subtalar joint may result in a better functional foot than an ankle fusion when the ankle joint range of motion is compatible with walking but the heel inverts at the sub-talar joint (Fig. 13-29).

Technique

The patient lies on his side, prepared as for an

FIGURE 13-29 Typical inverted foot in which the deformity occurred at the subtalar joint. The ankle movement was functional.

ankle fusion. The incision passes from just behind the lateral malleolus along the lateral border of the foot to the calcaneo-cuboid junction and then up towards the dorsum. Direct approach to the bone should display the subtalar joint (STJ) with minimal disturbance of the ligaments of the ankle joint. Wedges are cut from the joint surfaces to achieve the desired degree of eversion. This will not be one straight wedge the whole length of the STJ. If the inversion is very marked it may be necessary to do further wedges of adjacent joints (see triple arthrodesis). Blount's staples are very convenient and after 3 days elevation and removal of the drain (at 48 hours) the patient can have a walking TCC cast applied at day 10. Total immobilisation needs to be only 3 months as walking impacts the bones.

In young children such fusion is not recommended. A similar result can be obtained by a Grice operation designed as an extra-articular arthrodesis of the subtalar joint to stabilise the unstable STJ without sacrificing growth plates (Fig. 31-30). The block of bone (B) is taken from the iliac crest (A) and driven in to the subtalar

joint (C) to stabilise the position. It can be combined with an Achilles lengthening to provide a stable plantar grade foot in talipes equinovalgus or planovalgus. It may be of use in the child with early loss of anterior tibial function that tends to go into pronation but that cannot be adequately corrected by Tibialis Posterior transfer.

Triple Arthrodesis and other Midtarsal Wedge osteotomies and Arthrodeses

Triple arthrodesis is recommended for uncorrectable severe flexible pes planus with heel valgus. There are many variations in technique. It is essential that the deformity of each joint be considered. Various degrees of wedges are removed from each joint to create a solid heel block that passively plantar-flexes and dorsi-flexes at the ankle to provide a functional stable unit.

Technique

Place the patient on his side as described for ankle fusion. The incision commences behind the fibula, about 2-3 cm above the lower pole of the fibula and swinging round well below the lower pole of the fibula parallel to the sole of the foot to reach the calcaneo-cuboid area when it can turn up towards the dorsum. Cut deeply directly onto bone and elevate the flap. Do not damage the ligaments of the ankle itself. The subtalar joint is easily accessible without moving the fibula if the talus has not collapsed. The cartilage is removed from the subtalar joint and a wedge is removed from the upper surface of the calcaneum to evert the calcaneum to stop any tendency to inversion of the foot at the subtalar joint. This is especially so with a badly inverted heel. The talo-navicular joint is usually arthrodesed and this joint can also be
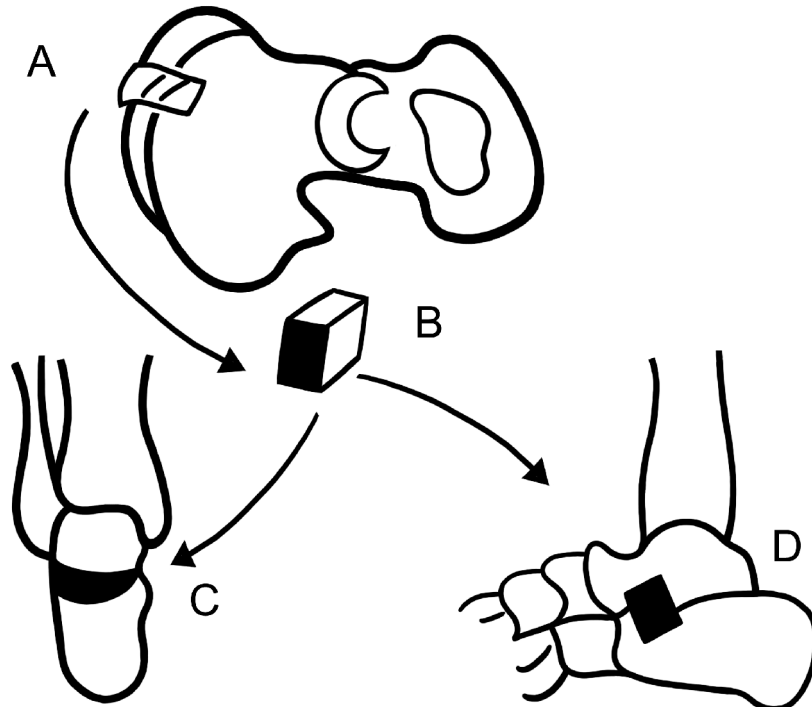

FIGURE 13-30 A piece of iliac crest graft can be inserted according to the Grice technique to stabilise the subtalar joint without damaging the growth plates.

wedged to improve the functional shape of the foot. The cartilaginous surfaces of the calcaneocuboid joint are removed and this joint can be wedged to improve the shape and function of the foot. This means that there can be complete arthrodesis through the midfoot at the site of the talonavicular joint. Those cuts can provide a wedging to reshape the arch or correct the turning in of the foot (Fig. 13-31).

Staples or screws can hold the arthrodeses. In grossly deformed feet it is sometimes advisable to use many K-wires (Fig. 13-32). Note that the midsole area that was initially the ulcer site over the peak of the boat is now a depressed
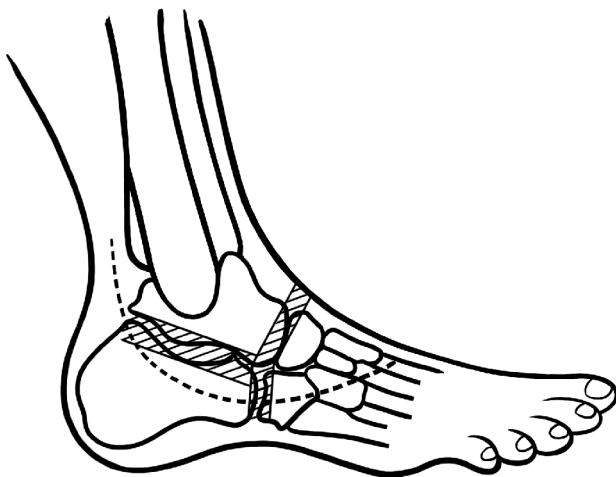

FIGURE 13-31 Diagram shows skin incision (broken line) for triple arthrodesis and the hatched areas that may need to be resected.

area so that in standing the weight will not be on the scar tissue. After drainage and closure the foot is placed in a cast. Postoperative care is the same as for the ankle arthrodesis. If there is a marked arthrodesis right across the midtarsal area it is advisable to keep these patients in a TCC for 9 months before trial walking. Figure 13-33 shows a left foot with a reasonably normal ankle but twisted midfoot. Triple arthrodesis followed by a Tibialis tendon transfer produced a satisfactory foot with near normal gait.

Closed wedge osteotomies of the midfoot require 7-9 months in TCC.

Management of the Shortened Equinovarus Foot

Many patients when first seen may have a deformed forefoot. This is often due to a motor deficit of the anterior tibial muscles. Much can be done surgically to treat the ulceration of the forefoot, in addition to a footdrop correction. However, this is not always practical because of the shortness of the dorsum of the foot, which may be shortened as far back as the head of the talus. A tendon transfer to provide dorsiflexion is difficult to attach and would have little mechanical advantage against the strong plantar flexors.
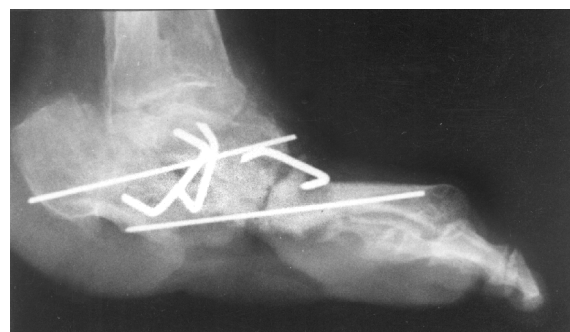

FIGURE 13-32 a-b Pre and postoperative radiographs of the gross deformity of the foot that has had many fractures and much disintegration. This foot has been reconstructed by multiple osteotomies requiring many wires and staples for stabilization.

FIGURE 13-33 Pre and postoperative photographs of a deformed left foot, corrected by triple arthrodesis and tibialis posterior transfer.

Technique of Salvage of the Short Foot

If the tissues of the heel pad are still reasonable, the best long-term results will be obtained by stabilisation of the hind foot. Trim the scar off the front of the foot for shoe fitting and see to it that new surgical scars are not as prone to trauma as the irregular scars left by ulceration. The basic procedure is an arthrodesis of the ankle to provide stability and removal of any prominent bumps on the plantar surface that may cause increased pressures during walking (Fig. 13-34 a-b). In some patients there is no need to do the arthrodesis. It may be enough to cut the Achilles tendon, remove part of it, to prevent plantar flexion and trim the front of the plantar surface so the stump is virtually a nice short rocker bottom. In some patients it may be better to tenotomise the tibialis posterior tendon if it is not practical to do a tibialis posterior transfer. In many patients there is no need of prosthesis as the malleoli are still present. They will be stable in a high lacing canvas boot if tied onto the stump. The foot would need 6 months in a TCC to obtain full healing. The patient should have minimal further problems as long as he maintains skin care and wears suitable footwear. If a prosthetic workshop is available a prosthesis can be made to fit the stump and provide a toe filler so the shoe does not crease.

This type of reconstruction reduces the plantar surface up to about 50% of its original size but is very practical when the patient has already lost most of the forefoot (Figs. 13-35 a-b).

Pirrogoffs procedure

There will be many feet in which the loss of the plantar surface is so great that the above adaptations are not adequate. These feet may benefit from a Pirrogoff procedure or the Boyd modification (Chapter 18).
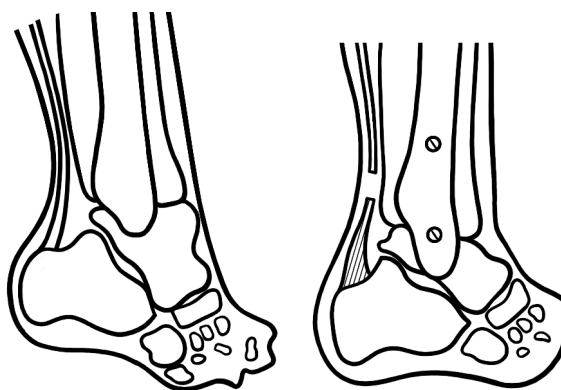

FIGURE 13-34 Diagram of a typically destroyed forefoot with odd fragments of bone that cause repeated ulcerating. At ankle fusion good dorsiflexion needs to be achieved and the worst scars on the forefoot can be tidied up, if needed, to leave a practical stump as seen in Fig. 35.

FIGURE 13-35 a. Deformed feet on which an almost blind patient could walk without ulcerating. **b.** After realignment he managed very well.

Forefoot Reconstructions

There are many surgical procedures to improve the foot function by removing the worst of the ulcerated forefoot, or reconstructing what is left so that the maximum amount of weight bearing tissues are preserved. The bones of the tarso-metatarsal (Lisfranc) area are often deformed and displaced downward into the plantar area and this becomes the site of frequent ulcers and scars. It is often the aftermath of major fractures or infections of that area but the toes and even most of the metatarsals escape. Often it is possible to do a wedge osteotomy right across the area and re-align the bones as described under wedge osteotomies, preserving the toes and whatever remains of the metatarsals. This provides a more socially acceptable foot than a Lisfranc or trans-metatarsal amputation, and preserves a larger amount of weight bearing surface.

An alternative management is to trim (ostectomy) the under surface of the Lisfranc area and correct the clawing of the toes (as by the Girdlestone procedure) so that the toe pulps become weight bearing again. This will help to reduce the pressure on the trauma prone Lisfranc area or metatarsal heads. The bones are frequently badly deformed and cause ulceration because of pressure from inside the foot. In this way it is possible to use the toe pulps to help protect the scarred area of the foot and so prolong the life of the foot, provided the patient takes adequate care of the foot and wears suitable shoes and resilient insoles. In some patients it is obvious that a trans-metatarsal or Lisfranc amputation is necessary (Chapter 18). In some patients if the plantar surface is trimmed to remove bony irregularities a pedicle skin graft may solve the problem of chronic ulceration.

Metatarsal Head Resection

It is not uncommon for an ulcer under the head of the great toe to result in osteomyelitis. Frequently this results in removal of the first ray, in part or entirely. In the neuropathic foot the removal of the first metatarsal head frequently results in ulceration and then removal of the adjacent metatarsal head and a domino effect results. To preserve as much weight bearing surface as possible and keep the second metatarsal head for as much length of time as possible it is advisable to remove the infected metatarsal head through a dorsal or dorso-medial incision. Ensure that the plantar surface of the remains of the metatarsal is bevelled to prevent undue pressure during walking. At

the same time excise and close any tracks connecting the ulcer to the bone and any plantar ulcer as described in the section on ostectomies. After removal of the infected bone the cavity is packed open and the foot elevated for about a week. Then a TCC is applied that ensures that the great toe is correctly placed for suitable function after healing. The TCC should have a window on the dorsal or medial side through which the cavity can be packed as frequently as necessary to encourage healing from the bottom. It will take about 6-8 weeks for the dorsal incision to fully heal from the bottom and during that time the plantar ulcer scar should also fully heal. However, as it was infected bone it is advisable for the patient to use the TCC for at least 3 months after surgery. The proximal end of the proximal phalanx of the great toe is often unaffected and can be the basis for a pseudarthrosis ensuring that arthrodesis does not occur. If the phalanx is also affected it is best removed completely but the toe tip and pulp can still be preserved. The great toe will be displaced proximally, perhaps until it is occupying the place where the head of the first metatarsal was. However, even in this situation the pulp of the toe should still take weight during walking. If it is not plantar grade enough then a Girdlestone type transfer of the FHL should be done to assist. This will prevent plantar flexion of the great toe IP joint and assist the great toe pulp to absorb some of the stresses of walking, and hence help to protect the second metatarsal head. If possible Girdlestone procedures on all the toes will assist in spreading the weight better and help to maintain a functional ulcer free unit.

Even in a foot with badly affected metatarsal heads it is often possible to preserve the toe tips or pulps and have them in suitable position to help spread the weight of the forefoot. If the metatarsals are rough or prominent they can often be removed while the toes are preserved. The use of the Girdlestone proce-

dure will frequently enable the surgeon to bring the toe tips down till they are weight bearing, even after the metatarsal heads are removed. However, when a few metatarsal heads are affected make sure that one head is not left protruding distally beyond the others. It may be better to trim a little off each head so that the remaining metatarsals end in an almost straight line to eliminate a prominent head that will almost certainly become the seat of an ulcer in the future.

Return to Free Walking

Whenever a patient following surgery and a TCC returns to walking it is advisable to institute a program of 'trial walking' to test the integrity of the tissues. For example, a newly healed ulcer will leave a scar on the sole of the foot. By 'trial walking', the stability of that scar is monitored. We allow the patient to increase the duration of walking at regular intervals, watching after each walk to check if there is any evidence of skin breakdown that would indicate that the newly healed tissues were not strong enough for weight bearing. If there is any evidence of tissue damage during a trial walking period the correct course of action would be to rest the limb in the cast again, till the hot spot settles and then the trial walking is again instituted. Recurrence of hot spots is best managed by use of a TCC walking cast, for a few weeks, or months, depending on the initial pathology. The cast can be bivalved, so it can be replaced after each test walk, until the foot has proved itself suitable for prolonged use.

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