WARREN, Agnes Grace
M.S. August 1972

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PATTERNS OF TARSAL BONE DISINTEGRATION IN LEPROSY PATIENTS

being a study of the prevalence and the normal progression of tarsal bone lesions and the variations achieved by treatment

by

Agnes Grace Warren

for presentation for
the degree of Master of Surgery, University of Sydney

It is impossible to know perfectly the part, if one is not acquainted with the whole, even in a gross way; so it is impossible to be a good surgeon if one is not familiar with the foundations and generalisations of medicine. On the other hand, as it is impossible to know the whole perfectly if we are not acquainted in a certain measure with each of its parts; so it is impossible for anyone to be a good physician who is absolutely ignorant of the art of surgery, with a knowledge of its possibilities and its limitations.

Henri de Mondeville
(1260 - 1320)

SUMMARY

In 1959, when the investigator first undertook the regular full time treatment of leprosy patients, foot problems were generally accepted as inevitable and part of the disease itself. Often, early lesions were allowed to progress until the foot was grossly deformed, and then amputation was advised. Antileprotic drugs eliminated the infection, but did not prevent the development of disability. The neuropathic basis for the development of foot deformities was still being investigated and, at this time, the literature contained few references to this aspect of leprosy.

A study of radiographs at Hay Ling Chau in 1959 showed that many patients, with gross foot deformities, secondary to tarsal bone disintegration, had initially presented with small, clinically undetectable lesions, which had only rarely provoked comment in radiographic reports. Over a period of time, these lesions had undergone alternating phases of regression and progression, resulting in healing of bone with some degree of deformity or in intermittent activity with increasing deformity and disability.

In some patients these periods of repair could be correlated with episodes of limited physical activity, when new bone was deposited and increased calcification occurred, but often the healing process was disrupted by the resumption of normal activity and a phase of further breakdown followed. Many of the radiographs examined showed progressive breakdown of the tarsal bones with minimal callus formation. Others indicated that all lesions did not progress to marked disintegration but that some did heal spontaneously. Differentiation of prognosis was impossible at diagnosis.

In this dissertation the term "Tarsal Bone Disintegration" will be used to include all tarsal bone lesions that on radiographic

examination show definite evidence of disintegration, either acute or healed. The term "Tarsal Bone Lesion" will be used for any other abnormality of the tarsal bones. In some patients an abnormality may initially be described as a lesion but will later show definite evidence of disintegration.

In 1959 it was decided to evaluate the effects of prolonged immobilisation on early osseous lesions; the results were reassuring, healing appeared to be encouraged. It was soon realised that plaster immobilisation to achieve healing of a bone lesion in an anaesthetic foot was required for a much longer period than in the normal foot. In addition, it also became apparent that if plaster immobilisation was discontinued before healing was complete, then disintegration proceeded more rapidly than before immobilisation was commenced.

Tarsal bone lesions may result in gross deformity to a degree that is incompatible with normal usage of the foot and renders the patient ulcer prone. Surgical correction of these deformities was attempted in 1964. With prolonged immobilisation healing was found to be satisfactory and the results were encouraging. Over-correction was essential to prevent recurrence.

The work so far described had slowly evolved as a response to the needs of the individual. To standardise therapy for easy application elsewhere, intensive study was necessary to determine the best therapeutic procedures to conserve good feet in those as yet undeformed, and to reconstruct deformed feet so as to improve function and prevent eventual amputation. This study has now been undertaken and its results are summarised here.

Tarsal bone lesions usually commence as minor traumata, possibly stress fractures, often neglected because of the reduced pain perception of leprosy patients with nerve involvement. Some degree of nerve involvement of the feet and legs can usually be detected

clinically in 84% of patients who develop these lesions.

The earliest clinical signs are painless heat and swelling of the foot or ankle. There is usually no relevant history of trauma. Clinical examination may elicit a neurological deficit as the only other sign.

In the untreated lesion there may be development of a minor deformity which is still compatible with normal function. As the lesion progresses more marked deformity occurs until the foot may be grossly deformed, hypermobile or unstable, but there is usually no heat or swelling at this stage.

In the extreme case the patient may walk into clinic, weight bearing on the end of the tibia, with the remnants of the foot as an appendage flapping beside it.

Osteoclastic and osteoblastic activity continue side by side and the relative proportions depend on the degree of activity of the patient and the resultant continued trauma of the involved bone. Some patients present with almost complete disintegration of the tarsal bones and very few signs of healing. In others, spontaneous healing occurs so that a stable but deformed foot results.

A systematic radiographic appraisal of tarsal bone disintegration showed that the lesions may not be detected radiographically until four or six weeks after the development of clinical signs. The earliest detectable radiographic lesions were minimal fractures, decalcification, mild fragmentation and partial collapse.

Later lesions showed obvious fractures with decalcification, marked fragmentation sometimes with absorption of displacement of fragments, and collapse of affected bones.

Healed lesions were characterised by increased calcification associated with deformity.

Chronic progressive lesions showed areas of disintegration in association with healed areas and increasing deformity.

The radiographic evidence supports the theory that microtrauma is a precipitating cause. Conditions that are associated with osteoporosis and hyperaemia frequently precede tarsal bone disintegration. Sepsis may be a complication but is not usually a precipitating factor.

Arteriography has not increased our understanding of the disorder.

Biopsy has not shown any specific pathology, but tends to support the theory that trauma is an important factor.

The treatment of choice is immobilisation in a weight bearing cast until clinical and radiological healing is complete. The early mild lesions will heal within six months. More severe lesions may take much longer.

In moderately severe lesions, if there is hypermobility, it is essential that the foot be moulded into a satisfactory functional position before the plaster is applied.

Gross fixed deformity may need surgical intervention to provide a functional foot. Healing does occur after surgery but immobilisation may need to be continued for twelve months or even more.

Sepsis may complicate disintegration, but if the foot is held in a satisfactory position bone healing will commence even before the sepsis is cleared and a final satisfactory outcome can be expected.

The healed bone appears sclerotic and withstands trauma very well. If a second primary lesion occurs in a foot, a different bone is involved. On no occasion has one bone been seen to be affected twice.

A programme of "Trial Walking" has been devised to check whether healing is complete and so minimise the possibility of reactivation of partially healed lesions when walking is resumed.

This study has shown that tarsal bone disintegration is a progressive disorder which, if untreated, leads to marked deformity and disability. The patients at risk have been determined and the modifications in general leprosy therapy that could help reduce their number. If early clinical signs are detected it is possible to obtain healing of the lesions without the development of deformity. It is possible to reconstruct a deformed foot to provide a functional unit and reduce the patient's dependence on others. Amputation should not be necessary if patients and staff are alerted to recognise in time and adequately treat this disorder.

It has been possible to formulate specific recommendations for the care of the feet of patients with leprosy. These "rules of daily living", if observed, will do much to reduce the disability arising from minor deformities and so enable the patient to lead a more normal life, even after he has suffered from tarsal bone disintegration with resultant residual deformity.

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This dissertation has been composed entirely by myself and is based on my own observation, reading and investigation at the Hay Ling Chau Leprosarium where I have been the Medical Superintendent since 1960.

A. GRACE WARREN
M.B.B.S., D.T.M. & H. (SYD)
30TH AUGUST 1971

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INTRODUCTION

In this dissertation the investigator will: -

1. Summarise the modern ideas in the relevant aspects of leprosy as a means of understanding the disease processes that result in deformity and disability.

2. Review the literature relevant to tarsal bone lesions as they are met with in leprosy.

3. Describe the lesions of tarsal bones observed in Chinese leprosy patients, including the clinical and radiological findings.

4. Describe investigations carried out by the investigator with a view to determining the aetiology.

5. Present the prevalence rates for tarsal bone lesions in Chinese leprosy patients and analyse them in an attempt to elicit any predisposing factors.

6. Describe methods of treatment adopted and the results obtained by their use.

7. Make practical suggestions as to the application of these findings for the benefit of patients.

Chapter 1
RELEVANT ASPECTS OF LEPROSY

Because tarsal bone disintegration is a local manifestation of a general disease the investigator thought it desirable to review in some detail the development and progress of leprosy. Special attention will be given to the complications of the disease which seem to predispose to bone disintegration.

"Leprosy in Theory and Practice", edited by R. G. Cochrane (2nd Edition, 1964), is the standard work on this disease and is the source of much of the information in this section. This work will not be specifically referred to, item by item. References of material from other sources will be given.

A. DEFINITION

Leprosy is a chronic infectious disease caused by Mycobacterium leprae, affecting mainly the skin and the nerves and subject to episodes of activity and quiescence over a period of many years. The skin lesions often dominate the clinical picture so that leprosy is frequently regarded as a dermatological disease and the nerve lesions are considered as a secondary manifestation.

But leprosy can also be regarded as a disease of peripheral nerves with cutaneous manifestations. A study of the histopathology of the nerves will aid in the classification of the various types of leprosy and in the understanding of the disease as a whole.

The manifestations of leprosy vary with the natural resistance of the patient. The clinical signs and the histopathological changes can be correlated to classify the disease into two polar types - lepromatous, the low resistance type; and tuberculoid, the high resistance type; and two less well defined groups - dimorphous and indeterminate leprosy.

B. HISTOPATHOLOGICAL CLASSIFICATION

The earliest histopathological lesion is an inflammatory cellular exudate in association with the finest nerve fibres. As the lesion progresses this exudate becomes focalised around the small blood and lymph vessels, the pilosebaceous apparatus and the hair follicles. It consists mainly of lymphocytes, histiocytes and plasma cells and gives very little indication of the future development of the disease. This histopathological picture may be associated with pink or hypopigmented macules or weal-like papules. This is indeterminate leprosy and may be regarded as an early, mild, transient and non-contagious form of the disease. This form of leprosy results in spontaneous recovery in 75% of persons so affected.

From this undifferentiated inflammatory condition two different patterns of histopathology may develop - the lepromatous and the tuberculoid types of leprosy. The dimorphous form of the disease shows a blending of these patterns though there may be a tendency for one or other pattern to predominate. This blending is often spoken of as borderline type leprosy.

1. LEPROMATOUS LEPROSY

As the disease progresses towards the lepromatous type, Mycobacterium leprae will be found in the cytoplasm of the Schwann cells. Here the M. leprae can multiply and may eventually burst into the endoneural spaces where they are phagocytosed by histiocytes which may gradually become vacuolated and form Virchow cells (lepra cells) typical of lepromatous leprosy and identified by their physaliferous or foamy appearance.

M. leprae are also found in the nerve axons and in some of the nerve fibres; the perineural spaces are packed with M. leprae but there is very little cellular reaction to this bacillary invasion. However, the high density of lepra cells increases the pressure in the nerves so that a relative ischaemia develops. There is also degeneration of the axoplasm, collapse of the Schwann tubes and disintegration of the myelin sheaths, but M. leprae are still to be found in unaltered Schwann cells.

Nevertheless the basic architecture of the nerves is unchanged and they are easily identified on section. The presence of M. leprae does not seem to prevent the regeneration of the damaged axons. In any involved nerve only some fasciculi are affected but this is accompanied by a hyaline degeneration of the endoneurium and a proliferation of the perineural reticulum fibres which may eventually lead to a firm fibrosed nerve with impaired function.

By this stage there will be definite clinical evidence of nerve damage. This is usually irreversible. During this slowly developing destruction of nerves there is a progressive invasion of the skin. The lesions are characterised by granulation tissue forming extensive sheets of granuloma packed with lepra cells and M. leprae, and containing groups of mononuclear and plasma cells. By now leprosy is a systemic disease and bacillaemia occurs. This,

together with the migration of histiocytes containing acid fast bacilli, results in granulomatous lesions of bone, bone marrow and many internal organs, but these lesions of internal organs do not usually produce clinical signs.

At the height of the disease the skin lesions show gross infiltration with or without nodule formation and usually some paraesthesia and erythema. As lepromatous leprosy regresses the granuloma gradually disappears. There is some regeneration of elastic and collagen fibres but the skin never resumes its normal texture and resilience and is more prone to traumatic damage than normal skin.

In lepromatous leprosy true anaesthesia is a late development as is motor paralysis; nerve involvement often increases as healing of the nerve lesions by fibrosis occurs. There may also be residual paraesthesia from damage to small nerve endings in the skin itself, even if stocking anaesthesia is not present, so that the limb is more prone to be traumatised and the trauma to be neglected because of diminution of pain perception.

The end result of severe lepromatous type leprosy is large areas of scarred skin which are often anaesthetic and prone to trauma and varying degrees of paralysis and paresis.

2. TUBERCULOID LEPROSY

The first specific manifestation of tuberculoid type leprosy is the presence in the fine nerve twigs of epithelioid cells which become surrounded by lymphocytes and histiocytes. These epithelioid cells may coalesce and form typical Langhans giant cells. This cellular exudate collects within the epineural and perineural sheaths and the normal architecture of the nerve fibres is destroyed, sometimes leaving fragments of the original fibres in an irregular granuloma. On histopathological examination of the skin in tuberculoid leprosy the cutaneous nerves are not always easily recognisable but may be represented by a few degenerated nerve bundles.

In the early stages the nerve fibres are swollen and irregular in shape but with an increase in epithelioid cells there is increasing degeneration and dissolution and Wallerian degeneration of the nerves. There is also an attempt to replace the degenerated nerves by regeneration.

The skin lesions may or may not be a prominent feature of the disease but will be well demarcated and will exhibit paraesthesia early, due to the early involvement of local nerve fibres. In mild cases there may be complete recovery of sensation but this is not usual when there are gross lesions of main nerve trunks. In these the granuloma around the nerve fasciculi is gradually replaced by connective tissue and compression of the nerve results. A central area of necrolysis may develop, followed by softening and the formation of a so-called abscess on the edges of which nerve fibres may sometimes be detected. Normal nerve architecture is destroyed.

Tuberculoid leprosy is characterised by a few well localised lesions. Acid fast bacteria can only be demonstrated in these lesions during a reactive phase (see page 158). There is early nerve involvement and patients may present with acute paralysis with

or without pain.

Tuberculoid leprosy resolves much more rapidly than lepromatous leprosy and due to the marked localisation of the granulomatous lesions in the sub-dermal tissues there may be comparatively small areas of anaesthesia. However, there may be damage of a main nerve trunk resulting in glove and stocking anaesthesia of the skin supplied by that nerve and frequently associated with paralysis.

In acute paralysis, recovery can be expected in a large proportion of patients who receive adequate treatment. This recovery may take months or even years suggesting that some nerve fibres have regenerated and every effort should be made to encourage nerve recovery.

3. DIMORPHOUS LEPROSY

Dimorphous leprosy shows a combination of the above two histopathological pictures in the same patient at the same time or at different times. The cutaneous nerves may be either free of acid fast bacteria and containing epithelioid cells or packed with acid fast bacteria lying singly and not in groups with a round cell infiltrate between the nerve fibres.

This form of the disease is very unstable and after acute nerve lesions of the tuberculoid type the disease may progress towards lepromatous leprosy with resultant fibrosis of the nerve and further impairment of function.

Some patients may show atypical features of lepromatous or tuberculoid leprosy and not the true intermingling of characteristics that occur in dimorphous leprosy. The term borderline is clinically used to include these patients as well as those with dimorphous features.

In 1962 a classification of leprosy for research purposes was published by Ridley and Jopling. This classification is used routinely in our work and in this thesis. For convenience the abbreviations listed in that article will be used in the text and the case histories of selected patients.

The abbreviations are: -

Typical tuberculoid leprosy	TT
Borderline-tuberculoid or atypical tuberculoid	BT
Borderline or dimorphous	BB
Borderline-lepromatous or atypical lepromatous	BL
Typical lepromatous leprosy	LL

C. IMMUNITY IN LEPROSY

It has recently been shown (Turk, 1970; Turk and Waters, 1969) that the ability of the patient to develop a cell mediated immune response (CMI) to M. leprae is an important factor in determining the type of leprosy developed and the final outcome. If the patient can produce only a humoral antibody reaction he will develop LL leprosy and may be subject to hypersensitivity phenomena which manifest themselves as Erythema Nodosum Leprosum (ENL) and other forms of lepra reaction. If the patient has a high CMI there will be a more rapid elimination of M. leprae but it may be associated with acute inflammation. This inflammation constitutes the lepra reaction of TT leprosy and may be associated with acute ulceration of skin lesions and acute paralysis.

In patients with BL-BB-BT leprosy the levels of immunity may alter from time to time and give rise to up-grading and reversal reactions as described by Ridley (1969).

D. REACTIONS IN LEPROSY

The term Reaction has been used to include a number of different medical entities. Broadly, it includes all acute exacerbations of the disease which may be associated with neuritis, iritis, arthritis, oedema, swelling, an increase in the size or activity of the skin lesions, and possibly ulceration. This "lepra reaction" may continue for many months and the patient becomes generally debilitated. Reactions may be either spontaneous or precipitated by therapy; judicious treatment of reaction may reduce the possibilities of permanent deformity by minimising the incidence of skin ulceration and the severity of permanent nerve damage. Treatment is dealt with in Appendix B, page 148.

Some BL-BB-BT patients show a lability of the immune response as mentioned previously. As a result, they are more prone to episodes of reaction than other patients.

In borderline leprosy there are multiple lesions, and each one may be affected during reaction so that many ulcers occur. This is particularly common in Chinese patients with borderline leprosy who frequently present with multiple ulcerations and continue to develop new ulcers. These ulcerations are often accompanied by polyneuritis which may be of acute onset and result in multiple paralyses which may affect all the limbs within a few days.

Intensive physiotherapy is essential for these patients to prevent the contractures which may rapidly develop because of general malaise and the patient's disinclination to exercise himself. Borderline leprosy that tends to reaction is probably the most crippling form of the disease as seen in Chinese patients.

In lepromatous leprosy the commonest form of reaction is erythema nodosum leprosum (ENL) which appears as small rose-coloured spots on inapparent skin lesions. These ENL lesions may progress to large

lesions and may ulcerate and become secondarily infected. They may continue for many months or even years and be accompanied by fever and general malaise. During episodes of ENL there may also be neuritis, arthritis, lymphadenitis, iritis and osteitis, which all combine to debilitate the patient.

In tuberculoid leprosy there is usually only one lesion that may be markedly swollen and erythematous, and may ulcerate. There is usually involvement of one or more associated nerves. Reaction may occur very rapidly and acute paralysis, resultant from reaction, may be the presenting sign.

The reactive lesions when present on the fingers and toes are known to be associated with localised bone rarefaction without the presence of sepsis, though secondary osteomyelitis is also a common cause for bone lesions associated with ulcerated reaction. In rarefaction associated with reaction the bone lesions will usually heal if protected from fracture and specific antileprotic drugs are given.

E. THE MECHANISM OF THE INVOLVEMENT OF NERVES

The earliest nerve symptoms may be numbness and paraesthesia which may be very difficult to evaluate and are often not associated with palpable or tender superficial nerves. There may be no obvious anaesthesia or even an abnormal two-point perception test or mis-reference at this stage, but the patient often suspects his disease although he shows no obvious skin lesions.

In lepromatous leprosy, neuritis may be acute, chronic or recurrent. Acute neuritis is usually associated with reaction (ENL) and unless intensive therapy is instituted will tend to become chronic or at least recurrent. The ulnar nerve at the elbow is the commonest site of the first episode of neuritis. Early lesions of the lateral popliteal and posterior tibial nerves are also common. The nerves may become acutely swollen and tender and very painful and there may be apparent weakness of the muscles supplied by the nerve. There is, however, rarely a complete acute paralysis and supportive therapy including physiotherapy will help preserve nerve function.

The episodes of lepra reaction may become more frequent until they are virtually continuous. Although this reaction may be accompanied by bouts of acute inflammation and tenderness the tendency is to an increasing fibrosis so that the nerves gradually become harder and more fibrotic. The nerves do not feel oedematous at this stage. Pain may be very severe and may continue for long periods of time and is usually associated with slowly progressive loss of muscle power and sensation.

In tuberculoid leprosy the nerves adjacent to the skin lesion are often acutely involved with or without pain. There may be sudden painless loss of power and on palpation the nerve may be oedematous though rarely will it be very tender. If the patient presents within a few days of onset a very dramatic recovery may

occur following the use of A.C.T.H. or corticosteroid therapy. If therapy is instituted after a longer period of time, there is still a good chance of a slower but adequate recovery of function. In tuberculoid leprosy nerve involvement is usually confined to one nerve or a group of nerves.

In borderline leprosy there may be acute involvement as for tuberculoid leprosy. However, usually more than one group of nerves is involved and it is not uncommon to have acute paralysis of some muscles in all four limbs and the face at the same time. As the disease progresses this acute neuritis is replaced by a more chronic form similar to that of lepromatous leprosy and characterised by constant nagging pain and slowly increasing paralysis - often in nerves that had escaped the first attack.

It is generally accepted that injudicial therapy, such as an over-dose of Dapsone or other antileprotic drug, can precipitate acute neuritis in tuberculoid and borderline leprosy, or aggravate and prolong the chronic forms of neuritis that affect lepromatous and borderline leprosy.

F. THE GENERAL RESULTS OF NERVE DAMAGE

In all types of leprosy the end results of nerve lesions are the same and dependent on the type of nerve fibre involved and the degree of involvement.

1) Motor nerve involvement results in paralysis or paresis of the muscles supplied by the fibres involved. The paralysis follows very definite patterns, affecting the more paripheral nerves and muscles initially. As the disease progresses new muscle groups may be involved.

2) Sensory nerve fibres may be destroyed in the skin, resulting in a patchy anaesthesia or paraesthesia in which some recovery often occurs and this may result in misreference. Main nerve trunk involvement may result in glove and stocking anaesthesia. The first modality to be involved is usually temperature or light touch, then pain perception is impaired and finally deep pressure sensitivity is affected. Deep muscle reflexes are rarely affected and sense of passive movement of joints is usually retained until the disease is far advanced. Some patients also lose deep pain perception in their feet so that trauma and surgery cause no discomfort.

3) Autonomic nerve fibres are involved as they course down the main nerve trunks with which they are closely related.

Their damage is shown by: -

a. Loss of sweating in an apparently normal skin.

b. Failure of hair follicule function.

c. Failure of vasodilatation in an affected limb.

d. Failure of reabsorption of sebum at an early stage and failure to secrete sebum in advanced disease.

e. Impairment of nail growth.

f. Disturbed pigment formation.

g. Lability of capillaries.

G. NERVE INVOLVEMENT OF THE LEG AND ITS SEQUELAE

The nerves in the leg that are usually involved are four: -

1. The lateral popliteal (common peroneal) nerve. This nerve is usually affected just above the neck of the fibula and the neurological signs may be associated with either the anterior tibial (deep peroneal) nerve or the musculo-cutaneous (superficial peroneal) nerve. A complete paralysis of all muscles supplied by both these branches is most common and is usually associated with anaesthesia of part or all of the foot and lower leg.

a) The anterior tibial nerve supplies the anterior tibialis, extensor hallucis longus, extensor digitorum longus, peroneus tertius and extensor digitorum brevis muscles.

It also supplies sensory fibres to the periostium of the extensor surfaces of tibia and fibula and to the ankle joint. The lateral terminal branch supplies the periostium and ligaments of the dorsum of the foot and the tarsal joints under the extensor digitorum brevis muscle and then becomes superficial to supply the skin of the first toe cleft.

b) The musculo-cutaneous nerve supplies the peroneus longus and peroneal brevis muscles and then becoming sub-cutaneous supplies most of the skin of the dorsum of the foot, except that supplied by the saphenous nerve and by the anterior tibial nerve.

2. The medial popliteal nerve may occasionally be damaged in the popliteal fossa so that there is a motor deficit affecting all muscles below the knee and marked sensory loss, both superficial and deep. The sural nerve, the cutaneous branch of the medial popliteal

nerve, supplies the lateral portion of the calf and a narrow strip of skin on the dorsum of the foot.

3. The posterior tibial nerve, the terminal portion of the medial popliteal nerve, is often enlarged immediately above and behind the medial malleolus. The enlargement may sometimes be felt for a few inches above this point. The resultant nerve deficit may paralyse some or all of the small muscles of the foot and cause anaesthesia of part or all of the sole. Sensory fibres are also supplied to the ankle joint. The medial plantar nerve sends fibres to the articulations of the tarsal bones and the metatarsals.

4. The saphenous nerve accompanies the saphenous vein down the medial aspect of the tibia and the foot to supply the skin as far as the first metatarso-phalangeal joint.

H. CLINICAL MANIFESTATIONS OF NERVE DAMAGE IN THE LEG AND FOOT

Many patients present with posterior tibial neuropathy - patchy or complete anaesthesia of the sole and claw toes due to paralysis of the small muscles of the foot. Because of this paralysis, the toes, in walking, do not take the body weight during take off, so that abnormal stresses fall on the metatarsal heads. This is especially so if the extensor digitorum longus is not paralysed and this muscle tends to extend the metatarso-phalangeal joints, causing elevation of the toes. Because of lack of pain there is no warning of impending damage and the first indication of an abnormality may be an ulceration, most commonly on the first or fifth metatarsal head area of the sole.

A lateral popliteal nerve lesion may present as a complete foot-drop resulting from a complete paralysis of all the muscles supplied by this nerve. The patient walks with a typical flapping gait with a tendency to inversion of the heel and foot produced by the unopposed action of the unaffected tibialis posterior muscle. This flapping gait allows the fifth metatarsal head area of the foot to strike the ground first with an abnormal amount of force so that ulceration often occurs at this site and it may be this ulceration that draws attention to the deformity. If the patient leads a sedentary life and no effort is made to keep the ankle and foot mobile he may develop a fixed deformity in the partially plantar-flexed and inverted position with shortening of the tendo Achillis.

This deformity may be even more gross if the tibialis anterior muscle is not paralysed, but the other muscles supplied by the lateral popliteal nerve are. In these patients the inversion is more pronounced so that the sole of the foot may face medialwards and the patient walks on the dorsilateral aspect of the foot which, however, is not grossly dorsiflexed. The skin at the ankle joint of the dorsilateral aspect of the foot usually ulcerates rapidly and

secondary osteomyelitis invades the tarsal bones.

A paralysis of any or all of the muscles supplied by the lateral popliteal nerve and the associated anaesthesia renders the foot more liable to trauma as a result of abnormal stress during every-day activities. Even if no paralysis is present, anaesthesia may allow misuse of the foot. Because there is no pain the resultant trauma is left untreated and ulceration, infection and loss of tissues may follow. These deformities make the foot more vulnerable to further trauma and may set up a cycle of increasing deformity and disability.

I. BONE INVOLVEMENT IN LEPROSY

The presence of damaged bones has long been accepted as a common finding in leprosy patients. Recently investigations have been undertaken on the osseous remains of graveyards where leprosy sufferers were buried and these have thrown light on the character of the disease in the Middle Ages (Møller-Christensen, 1953 and Andersen, 1969).

For a long time, however, it was not appreciated that many of the bone lesions were due to secondary complication and were not specific lesions of leprosy. This point was made by Paterson (1961) as a basis for the possibility of preventing increasing deformity in patients with anaesthetic limbs. As a result of his work the bone lesions of leprosy can be divided into the following types: -

1. Specific changes due to the presence of M. leprae and usually involving the small bones of the hands and feet.

These changes are: -

(a) Honeycombing in the distal ends of the phalanges.
(b) Small pseudocysts.
(c) Reticular disorganisation of a phalanx.
(d) Decalcification, especially of a patchy nature, causing sub-periosteal and sub-articular lesions.
(e) Sub-periosteal new bone formation especially on the sub-cutaneous surface of the tibia.

Job (1963) stated that lepromatous granulomatous tissue had been found in biopsies of the destructive lesions of small bones of the hands. These destructive lesions invade and destroy the bony trabeculae. Healing takes place by fibrosis so that the bone marrow may be replaced by dense fibrous tissues. Radiographically these lesions

have been seen to resolve within one year of commencing regular Dapsone therapy (Paterson, 1961). They rarely appear after the patient has been stabilised on antileprosy drug therapy (Erickson and Johanson, 1948). If the affected digits are protected from trauma while antileprosy treatment is given, healing will occur. Deformity is often the result of trauma, especially to the fingers, which may fracture with normal usage during periods of osteoporosis that may accompany lepra reaction or inflammation.

2. Non-specific changes are associated with abnormal stress factors or with secondary infection that has entered through areas of ulceration or trauma and established itself.

These changes are: -

(a) Hazy areas in the cortex with loss of trabeculae and decalcification usually associated with ulceration and infection. Early therapy can stop the advance of osteitis and osteomyelitis but healing may be associated with the development of further abnormalities.

(b) Irregularities of bone. Due to stimulation of the periosteum associated with the above, bony spurs may develop that will in turn lead to further ulceration and more osteomyelitis.

(c) Irregularities of joints with ankylosis or hypermobility - frequently the result of septic arthritis from trauma to the joint.

(d) Pathological fractures of weakened bones, usually metatarsals or phalanges.

(e) Concentric absorption of phalanges caused by constant usage of a foot with chronic osteitis. A slow absorption of the sub-periosteal layers of the cortex occurs and new bone is laid down on the medullary side of the cortex until the phalanx is markedly thinned and may fracture under usage.

3. Osteoporosis, which may become very marked in patients requiring prolonged immobilisation or bed rest and may predispose to a pathological fracture.

4. Neurotrophic joints are described as occurring in association with a muscular palsy and stocking anaesthesia of the leg. The lesion at an early stage may show as a minimal crack fracture, a hazy area, or an osteophyte, but without therapy may develop into complete dissolution of the tarsal bones. The appearance and behaviour of these joints while under observation is similar to that which occurs in diabetes and syphilis and congenital absence of pain. These lesions do not appear to be due to leprosy as such but to be the result of neglecting traumata that would in a person with normal sensation be rested and protected from further trauma.

J. ULCERATION IN LEPROSY

In certain phases of the disease the skin lesions may break down and ulceration result. This usually only occurs during a reaction and is the true ulceration of leprosy. It may be severe and prolonged, resulting in marked scarring, or secondary infection may occur and possibly also osteomyelitis.

Diminished pain perception of the foot, and especially of the skin of the sole, combine with the disturbed mechanism of function caused by muscle paralysis to produce abnormal stresses on the tissue which cause necrosis.

The earliest manifestation of tissue necrosis may be a blister, a haematoma, or only a mild degree of oedema which may not be easy to detect. In the foot early minimal trauma may occur around the toes and metatarsal heads and resultant oedema may cause a slight increase in the distance between the toes (Brand, 1963). Complete rest at this stage results in healing in a few days but if no rest is taken and the patient continues to walk he may present with a blister within forty-eight hours and an ulcer within a few days.

In most leprosy patients one can consider that healing proceeds normally if the limb is rested, but because of the lack of pain perception the lesion is often neglected and the patient may not even be aware of its presence unless he has been taught to examine his feet regularly, looking for trauma. Even obvious ulcers may be neglected until frank infection has intervened and it may be a lymphadenitis that attracts the attention to the infected foot.

In leprosy, a trophic ulcer will heal if it is kept clean and is rested but because of the diminution of pain awareness the patient will not spontaneously rest as would a person with normal sensation

and he continues to use or misuse the limb. This continued use of the limb delays healing with the result that the ulcer becomes chronic. After many years of recurrent and chronic ulcers there will be residual scar tissue and healing of these areas may be impaired. The presence of scar tissue on the sole of the foot increases the vulnerability of that foot so that minor trauma tends to produce fresh ulceration much more easily. Hence it is important to try and prevent the first ulcer and to achieve healing, as quickly as possible, of any ulcers that do occur so that the residual scar is kept to a minimum.

Because of the problem of lack of pain sensation it is often advisable to treat chronic ulcers by complete immobilisation of the affected foot in a walking plaster, to ensure rest and to encourage healing (Cochrane, 1947 - page 173; Price, 1959; Bauman, Girling and Brand, 1963; Andersen, 1964). Most ulcers will heal if the foot is kept immobilised in a walking cast for six weeks and this has become the accepted out-patient treatment for chronic foot ulcers (see also pages 99 and 121-2). Ulcers that are more chronic or very large may require longer than six weeks and this period of immobilisation may extend to many months, but persistence should achieve healing in the majority of patients.

The patient must be taught to watch for trauma and to treat it early and effectively if he is going to maintain his feet in good condition.

K. PROGRESSIVE DAMAGE OF THE FEET IN LEPROSY

In leprosy the lesions of the disease itself do not usually cause marked deformity or disability. It is possible for the patient who will take care of himself and take regular therapy to maintain his hands and his feet in a good physical condition in spite of the presence of paralysis. However, due to the combination of paralysis and anaesthesia and bone involvement as outlined earlier, (pages 17-23) many of these patients do develop marked disability. This is often because minor trauma is neglected as it is unaccompanied by pain. Many persons do not realise that much deformity can be prevented.

For example, the patient may walk on a large foot ulcer because it causes no pain when he does so, and as a result the ulcer will progress and healing will be retarded. If the same patient can be persuaded to rest his foot the ulcer will heal in due course.

The patient with a paralysis of the peroneal muscles will tend to walk with an inversion and as a result may develop a fixed inversion deformity with ulceration on the lateral border of the foot.

One of the biggest problems that is encountered in leprosy clinic work is the gaining of the co-operation and understanding of the patient so that he will report ulcers, muscle weakness and other lesions early and accept recommended therapy so that the lesion will heal without increased disability. Continuing ulceration and increasing deformity may eventually destroy the usefulness of the foot, and yet in most cases this deformity and disability can be prevented.

L. RESUME OF RELEVANT ASPECTS OF LEPROSY

The manifestations of disease that follow an infection with M. leprae are many. Tuberculoid leprosy is characterised by rapidly developing skin lesions and early neuritis. Paralysis and anaesthesia develop early and residual stigmata and deformities remain. The disease may quickly become non-active but the patient is scarred and must learn to live with his disabilities.

Lepromatous leprosy, by contrast, may be a slowly progressive disease in which there are no obvious clinical signs for many years. Neuritis is a late complication that can frequently be avoided by early treatment but is inevitable in the untreated patient.

Anaesthesia and paralysis of hands and feet slowly progress until the patient is deformed and disabled. The end results for all untreated badly involved patients are similar as far as hands and feet are concerned - anaesthetic limbs with muscle paralysis or paresis. Because of the resultant disability the patient misuses his limbs and traumatises them. Because of the inability to feel pain the trauma is often neglected and secondary infection follows. Thus a cycle of increasing deformity and disability is commenced.

Patients who come for treatment in the early stages of the disease need not develop marked disability. Adequate drug therapy (see page 148) and education in the care of anaesthetic limbs, assisted by physiotherapy (see page 149) and surgery (see page 150), can do much to minimise disability. However, many patients only seek help when deformity and disability are already present, and more understanding of their needs and problems is required to rehabilitate them.

Chapter 2
REVIEW OF RELEVANT LITERATURE

A. ON TARSAL BONE LESIONS IN LEPROSY PATIENTS

1. Causation of bone lesions

Cochrane (1947 - pages 52-3) referred briefly to the marked trophic changes that occur in neural leprosy and stated that osteoporosis may occur without trophic ulceration. He asserted that necrosis of bone and signs of periostitis and irregular ossification never occur in the absence of sepsis following trophic ulceration. He observed that tarsal bones may be affected when phalanges and metatarsals are not involved but asserted that this is usually secondary to ulceration and may lead to a condition similar to a Charcot's joint.

Barnetson (1951) provided a valuable survey of the literature regarding bone lesions in leprosy. He connected bone lesions of leprosy with nerve involvement, trauma and infection, altered mechanics, and possibly changes in vascular supply. However, he only mentioned the involvement of the small bones of the hands and feet and implied that the most peripheral parts of the most peripheral bones are primarily affected. He did not mention the involvement of tarsal bones or more proximal bones but stated that secondary factors such as trauma may play a causative role in the development of secondary bone lesions.

Paterson (1959 - pages 245-256) discussed the various types of bone lesions that occur in leprosy patients, but drew all his examples from the small bones and did not specifically mention tarsal bone involvement. He compared the bone lesions of leprosy with bone lesions in other disorders of peripheral nerves and disease producing similar radiographic pictures. He accented the hypersensitivity of tissues after the central nerve of supply has been

cut, resulting in hyperkeratoses, easy bruising, sub-cutaneous haemorrhages and then fragile skin. He asserted that true trophic changes are limited to abnormalities of tissue repair and may be manifested by excessive new bone formation or osteoporosis. Also, that irregular bone erosion is related to non-specific soft tissue infection and is the result of trauma which can frequently be prevented if the patient will learn to care for himself. He included neuropathic disintegration of the tarsal bones in his article in 1961.

Price (1960A) also commented on the occurrence of minimal new bone formation in neuropathic joints. He accentuated the fact that the neurotrophic joint of leprosy resembles that of diabetes rather than the classical Charcot joint of syphilis. He stated that its frequency is probably under-estimated, although no literature was then available concerning its frequency.

Paterson and Job (1964 - page 441) stated that disintegration of the tarsal bones had been noted in about 2% of patients in a leprosarium. They suggested that neuropathic bone and joint lesions may be the result of unusual trauma that is allowed by sensory loss, and that the bone may be secondarily involved by sepsis which results in widespread and rapid destruction. They implied that neuropathic bone and joint lesions only occur in association with stocking anaesthesia and muscular palsy, and emphasised the fact that if adequate therapy is not given complete dissolution of the tarsal bones may occur. They also mentioned the possibility of healing occurring during immobilisation of the affected limb in a plaster cast.

It is evident that, according to Paterson, 1961; Andersen, 1961; Brand, 1963; Price, 1964 - page 523; Lennox, 1965; Harris and Brand, 1966; and others, microtraumata are accepted as playing a leading causative role in the development of neuropathic bone

lesions in the feet of leprosy patients. This thesis is difficult to sustain on their published radiographs which are usually of severe or advanced lesions. Radiographs of early lesions are rarely seen in the literature.

In some cases, however, fractures are seen easily and Johnson (1967) stated that in the majority of neuropathic joints that he had examined, in which the aetiology varied, fractures of insignificant magnitude were of major importance in initiating joint change. Even a minor fracture may prove very serious if it leads to joint instability and abnormal stresses.

Microtraumata can result from every-day activity and may not be noticed by the patient. Walking for long distances is enough to cause "march fractures" in normal feet (Bernstein and Stone, 1944) and similar lesions may occur in anaesthetic feet. Lennox (1965) stated that painless fractures of foot bones were relatively common in the anaesthetic foot.

Abnormal mechanisms of walking were implicated by Harris and Brand (1966), as was vigorous activity associated with loss of pain sensibility, yet the incidence of disintegration of the tarsal bones is comparatively low when compared with the number of active persons who have nerve lesions of the lower limb resulting in a degree of anaesthesia and paralysis. Cases are reported where tarsal disintegration has occurred during periods of limited activity for foot ulceration or medical complications.

Cochrane (1947 - page 52); Paterson (1961) and Karat et. al. (1968) mentioned the incidence of osteoporosis in the bones of leprosy patients. Generalised osteoporosis is particularly noticeable in patients who have been in bed for prolonged periods, and localised osteoporosis is frequently seen in the foot bones after the limb has been immobilised in a walking plaster for any reason.

No records are available of the incidence of bone lesions that occur after the removal of a walking plaster or after other immobilisation sufficient to allow osteoporosis to develop. Price (1964 - page 519) stated that osteoporosis may predispose to collapse of the foot.

Paterson (1961) mentioned that talo-navicular collapse may follow plaster immobilisation or result from damage to the bones during tibialis posterior transplant operation (see also pages 150-152 of this thesis).

Lennox (1965) suggested that over-activity while in a walking plaster may predispose to bone breakdown on removal of the plaster.

Writing as the Chairman of the Panel on Physical Medicine and Rehabilitation at the 8th International Congress on Leprology held in Rio de Janeiro, Brazil, in 1963, Brand (1963 and 1964) stated that neuropathic bone changes were the result of microtraumata and often accompany, but are not caused by sepsis or plantar ulceration.

Harris and Brand (1966) studied patterns of disintegration in the bones of the feet of leprosy patients and gave the case histories of seven patients to illustrate the progress of the lesions. Each of these seven patients was under therapy for plantar ulceration and the radiographs that were taken showed bone pathology of various types. No mention is made of the prevalence of bone lesions in patients without ulceration. However, they stated that the role of sepsis is not obvious.

Karat et al. (1968) stated that bone and joint lesions in anaesthetic limbs are almost always associated with ulceration. Their examples also were predominantly patients who had ulceration and in whom later neurotrophic bone lesions were found.

Cochrane (1947 - page 52; 1959 - page 138; and 1964 - page 275)

adhered to this view but Paterson and Job (1964 - page 441) stated that ev n when no plantar ulcer or sepsis was present, degenerative changes of the bones could occur. These changes could provide a focus for infection, the resulting sepsis leading to further bone damage.

Price (1960A) pointed out that sepsis often accompanies a bone lesion but may be a secondary factor rather than a primary one. He made no mention of the role of sepsis in early lesions, but stated that lesions may occur in the absence of sepsis.

Lennox (1965) stressed the importance of the presence of infection that may lead to hyperaemia and oedema which may cause softening of the plantar ligaments. These then may stretch when the patient walks, so allowing the foot to collapse.

Johnson (1967) mentioned that hyperaemia may weaken bone and predispose it to further injury.

Barnetson (1951) suggested that one factor concerned in bone atrophy may be a failure of the blood vessels supplying the bone. He did not specifically mention disintegration. He demonstrated a failure in reflex dilatation of vessels when stimulated by heat or cold, but local stimuli were effective in producing vasodilatation. He assumed leprous interstitial neuritis was the basic causal lesion.

Lechat and Chardome (1958) investigated arteriography of the circulation in "mutilated lepers" in whom scarring may have been a secondary factor in producing abnormalities. Lechat (1962) suggested that obstruction of small arteries and anastomoses between small arteries and veins caused early venous filling and a slow circulation.

Bang and Tiep (1958) reported normal arteriography in twenty-six

out of thirty-four patients with perforating type leprosy of the foot. The patients with abnormal findings showed complete or incomplete obstruction of the vessels and changes in the calibre of the arteries, either constriction or enlargement, especially enlargement of collateral arteries, suggesting a disturbance in the main blood flow.

There is no mention in these papers of tarsal bone lesions as such and it cannot be assumed that any of these abnormalities are present in association with tarsal bone disintegration. These papers were written at a time when it was considered that the small bones of hands and feet were the ones mainly affected.

Paterson (1961) reported on abnormalities in arteriography in lepromatous cases with bone absorption. No reports were found, by the investigator, regarding arteriography in leprosy patients with tarsal bone disintegration.

Barnetson (1951) reviewed the investigations into blood chemistry and concluded that these abnormalities have little if any correlation with bone absorption in leprosy.

No definite association has been found between bone disintegration and abnormalities of blood vessels, blood chemistry or general nutrition.

It is generally accepted that the lesions are progressive and lead to increasing disability, but that little is understood about the mechanisms of destruction and repair in the neuropathic bones and joints of leprosy patients.

There are many factors that have been suggested as playing a causative role in the development of tarsal bone disintegration. It would appear that microtraumata, associated with lack of normal pain perception in the joints of the feet, are the prime factors and that sepsis may play a role either in the initiation or in the progression of the lesions.

2. Treatment

In reporting the findings of the Panel on Physical Medicine and Rehabilitation at the 8th International Congress on Leprology held in Rio de Janeiro, Brazil, in 1963, P. W. Brand (1963 and 1964), as Chairman, summed up the treatment of foot deformities by stating that treatment should be directed to the achievement of undeformed stability by surgical and non-surgical means. However, how this should be achieved is still a matter of controversy.

Paterson and Job (1964 - page 441) suggested that healing of neuropathic bone lesions could be achieved by immobilisation in a weight bearing cast. Price (1964C- pages 523-4) suggested surgery, or a weight bearing hip calliper, so that the muscles of the leg and foot would be made non-functional for a period of twelve to eighteen months. He warned against immobilisation in a plaster cast, suggesting that this would aggravate the condition. He recommended bed rest as the treatment of a suspected lesion but did not state the period of rest necessary for healing.

Arthrodesis was recommended by Price (1964C- page 523) as the treatment giving quickest results: he stated that three months would give sound healing but did not differentiate between the different affected sites or degrees of severity.

However, many leprosy clinics have no facilities for provision of such callipers or for prolonged bed rest and cannot tackle major surgery so some easier routine is needed to save many feet from deformity and disability.

Ross and Maclean (1964) did not recommend arthrodesis for neuropathic joints because of the difficulty of obtaining healing, and they recommended prolonged bed rest as the best therapy without mentioning any form of immobilisation or any method of maintaining a functional position.

Lennox (1965) stated that arthrodesis was the operation of choice and mentioned the use of inlay grafts of cancellous bone. He stated that there should be no delay once diagnosis of disintegration is made and implied that an arthrodesis will heal in twelve to sixteen weeks after which a special shoe should be fitted. He stated that surgery will only succeed if a special surgical shoe is worn and mentioned the risks of operating on some of these poor-risk feet when many surgeons would amputate without hesitation.

Johnson (1967) stated that healing will occur in neuropathic bone lesions if proper treatment is given. He stressed (1) preventive therapy - i.e. the adequate treatment of fractures, sprains and effusion in neuropathic limbs as the first essential. (2) Early diagnosis and (3) adequate protection until all activity subsides. He listed rest in bed, immobilisation in plaster, crutches and braces, but warns against too early resumption of activity. He implied that even grossly involved joints will heal in time and that surgery is only required in the late stages when marked deformity, that requires correction, is present. He stated that an operation it is essential to expose good bone and use some form of fixation to ensure immobilisation. The removal of bony spurs and prominences that interfere with the weight bearing surface of the sole may also be necessary. In neurotrophic feet he recommended allowing the acute process to subside before attempting surgery.

Harris and Brand (1966) stated that the provision of special shoes is the treatment of choice in the early stages of involvement of the posterior pillar and the medial arch but they recommended surgical intervention for lateral arch involvement. When definite bone damage is established they considered full immobilisation necessary and observed that sound union will take longer than in a normal foot. If conservative methods fail they suggested that surgical fusion should not be delayed and the moulded unpadded

plaster should be used for a period of time that is 50% longer than for the same procedure in a normal foot. Even then, care must be taken as walking is resumed, for indications that union is not as firm as it was thought to be.

Ikeda (1965) speaking about the treatment of neuropathic joints and other foot deformities in leprosy patients stated that it may require up to two years to achieve complete union after "talo-crural arthrodesis", and 5.5 months after "talo-calcaneal arthrodesis".

It is obvious that these writers have referred to different stages and degrees of severity in the pathological process and that various methods of treatment may be applicable at different times. The medical officer in charge is responsible for trying to achieve undeformed stability, be it by surgical or by non-surgical means. To this end further study is needed to differentiate the stages of the disease and the methods of therapy applicable in each.

Failure usually appears to result from immobilisation that is inadequate or is discontinued too soon, but no guide is available at present in the literature to correlate the type of immobilisation and its duration, with the site of disintegration, its severity and duration.

B. ON STRESS FRACTURES

The Second World War provides much material for study concerning stress or fatigue fractures.

Stress fractures have been defined as apparently spontaneous fractures that occur in a normal bone as a result of repeated stresses, although any one would not by itself damage the bone. They may also occur as a result of a chance faulty movement, especially as seen in tabetic patients with an ataxic gait (Watson Jones, 1952 - page 350) or in a normal person walking over rough ground (Watson Jones, 1952 -p.343).

Morris (1968) stated that the term "stress fracture" was used to describe three separate but over-lapping responses of normal bone to repeated minor stress. Firstly, a fracture, partial or complete, may occur. Secondly, a microscopic fracture may occur which will not be apparent on radiographic examination at the time of injury but will be detected in retrospect by callus or clinical development. Thirdly, osteoporosis may develop in a bone that is undergoing stress; this may progress to callus formation or if the stress continues may develop into a true fracture.

Watson Jones (1952 - page 344) pointed out that radiographs taken immediately after the fracture may not show evidence of fracture, but that if these were re-examined after six or eight weeks and compared with newly taken films it would frequently be possible to detect the fracture on the first film by the use of a hand lens. Morris (1968) stated that changes may not be observed for fourteen to twenty-one days, when a crack or callus may be seen. He also mentioned the progressive nature of the lesion and stated that a complete fracture may occur some two to three weeks after symptoms first occurred.

In 1944 Bernstein and Stone stated that most stress fractures would show some callus in ten to twenty-one days and that healing

would usually be complete at four months unless malalignment occurred from the continued use of the unsupported foot. These statements indicate the difficulty of an accurate diagnosis even in a normal foot and the importance of making one, if malalignment and deformity are to be prevented.

Watson Jones (1952 - page 347) stated that if there is clinical evidence of a fracture the patient should be treated for a fracture even if the radiograph does not support the diagnosis. He listed the clinical signs as tenderness, oedema and pain on strenuous exercise. He also pointed out that induration of the skin over the lesion may occur and after a few weeks a lump may be felt to indicate the site of callus formation. Burrows in 1940 had described the clinical signs and pointed out that there is usually an absence of any suspicion that a fracture had occurred. Bernstein and Stone (1944) included the fact that crepitus rarely occurred and the pain was of a dull burning or aching character.

The majority of stress fractures occur in the metatarsal bones - but other bones are not completely exempt. Reporting on 700 cases at the Ford Ord Infantry Training Center, U.S.A., Morris (1968) quoted that 51% occurred in metatarsals, and 26% in the calcaneum, and 17% in tibia.

In 1944 Bernstein and Stone recommended the use of a shoe incorporating a metal bar that would keep the sole rigid and prevent pain on walking, while allowing the patient to continue full military training. They stated that this therapy gave excellent results.

Leavitt and Woodward (1944) inferred that the use of short walking plaster produced a high rate of residual disability, but that excellent results would be obtained by complete freedom from weight bearing until all soreness had disappeared.

Thredgill and Hollingsworth (1952) recommended a walking plaster for six to eight weeks and then rapid return to extensive walking and normal duties. Watson Jones (1952 - page 347) recommended the use of a plaster until union was firm and then simple strapping, combined with exercises, to develop the supporting musculature.

Morris (1968) pointed out that an early lesion may be treated by rest or protection but if a complete fracture had occurred immobilisation was necessary, either by internal or external fixation.

Stress fractures are not uncommon in normal feet; they occur in the absence of obvious trauma. These workers have used different methods of treatment yet each claims good results. However, the basic principles are held by all - immobilisation to prevent pain until union is firm and then rapid return to full mobility.

Chapter 3
MATERIALS

A. AVAILABLE AT HAY LING CHAU

The radiographs and clinical histories of over 1,500 Chinese leprosy patients treated personally by the investigator at Hay Ling Chau, Hong Kong, were available for study. These comprise all patients in residence in 1959 and those admitted between 1960 and 1970.

For all these patients routine radiographs were taken of hands and feet on admission and regularly, as required, during their hospitalisation. Periods of hospitalisation varied. A large group of these patients were under treatment for six to ten years.

Routine lateral view radiography of the feet was not commenced until 1964 so that full radiographic studies of the feet are only available for patients under treatment since 1964. Prior to 1964 lateral view radiography was only ordered when a lesion was suspected. Also prior to 1964 many of the dorsiplantar views of the feet did not visualise the talus or the navicular. Hence minimal lesions prior to 1964 may not have been identified.

In addition, the radiographs and histories of about 400 patients discharged prior to 1959 were available for study (see Appendix A, page 141, for Background to Hay Ling Chau and the development of the facilities there. See Chapter 4, page 51 for methodology of radiographic routines).

B. SELECTION OF PATIENTS FOR ADMISSION TO HAY LING CHAU

When Hay Ling Chau Leprosarium was instituted in 1951 (see Appendix A, page 141) the emphasis was on admission of patients with positive skin smears (see Appendix C, page 157) and their retention until the skin smear was negative. No reconstructive surgery was done in the early years. The admissions included most of the patients suffering from multibacillary forms (see Appendix C, page 158) of lepromatous and borderline leprosy, but relatively few patients with tuberculoid leprosy.

Modern scientific knowledge has changed attitudes to leprosy so that fewer patients are now admitted to segregation units and patients are admitted for reconstructive surgery and rehabilitation.

In 1968 only 25% of the newly diagnosed leprosy patients from the Hong Kong out-patient clinics were admitted to Hay Ling Chau for in-patient care. This meant that a smaller number of places were being utilised for contagious patients and patients requiring drug stabilisation, and the total number of patients in the institution was gradually falling. This trend enabled the proportion of disabled patients admitted for reconstructive surgery and rehabilitation to be increased.

Those who have been admitted to Hay Ling Chau since 1968 are: -

1) Patients with multibacillary leprosy of either lepromatous or borderline type (see Appendix C, page 158).

2) Patients with complications such as reaction (page 11), neuritis (page 13) and ulceration (page 24), requiring intensive treatment to prevent or to minimise residual deformity and disability.

3) Patients requesting, and suitable for, reconstructive surgery (see Appendix B, page 150).

Therefore amongst the patients admitted to Hay Ling Chau in recent years there has been a higher proportion of these three groups than in the leprosy population of Hong Kong as a whole.

The figures quoted by the Government out-patient clinics indicate that in the last five years, of the patients who were newly diagnosed:

(1) 55% had tuberculoid leprosy (including TT and BT types)
(2) 25% had lepromatous leprosy (including LL and BL types)
(3) 20% had borderline leprosy (BB type)

on clinical classification at diagnosis. However, admissions to Hay Ling Chau showed:

(1) 10% had tuberculoid leprosy (including TT and BT types)
(2) 53% had lepromatous leprosy (including LL and BL types)
(3) 37% had borderline leprosy (BB type)

on histopathological classification (see page 9 for classification).

As the means of classification used to achieve these figures was different they cannot be expected to be completely comparable. They do, however, show the proportionate severity of the different types of leprosy from the point of view of the need for intensive care to prevent or correct deformity and disability.

Hence the group selected for admission to Hay Ling Chau cannot be regarded as a true representation of leprosy in the Chinese patients of Hong Kong. They may present a higher proportion of deformities than would be seen in a more representative group, because patients diagnosed at an early stage of the disease, and those with less severe forms of the disease, are treated as out-patients.

By contrast, because of the political situation in China and the socio-economic changes in Hong Kong in the last thirty years, there are relatively few severely disabled patients of very long standing in Hong Kong although this type of patient is common in other centres in South East Asia.

C. SELECTION OF PATIENTS FOR INTENSIVE STUDIES

Four hundred patients were selected in order to determine the prevalence rates of tarsal bone lesions in Chinese leprosy patients.

The patients chosen for intensive study were consecutive new admissions to Hay Ling Chau Leprosarium during the years 1963 to 1968 for whom dorsiplantar and lateral radiographs of their feet were available. Those included all patients with admission numbers 1601 to 2011, except patients with numbers 1609,1610, 1613, 1632, 1647, 1674, 1764, 1823, 1864 and 1900, for whom these radiographs were not available. For simplicity, patients will be referred to by the admission number.

The results of the investigations on these 400 patients will be analysed so that the prevalence rate for this series of involvement of the tarsal bones may be determined. An attempt will be made to correlate the incidence of these bone lesions with the types of leprosy, the bacillary index, the nerve involvement of the affected limb and the presence or otherwise of sepsis at the time of diagnosis of the bone lesions, in order to indicate the patients at risk.

D. SELECTION OF PATIENTS FOR BIOPSY OF BONE LESION

(1) At Hay Ling Chau

In 1959 two patients with navicular disintegration were selected for biopsy and arteriography because, during the previous six months, bone breakdown was detected without any known precipitating cause - Patients No. 309 and 780.

In 1965 one further patient was selected for investigational biopsy when he presented with a highly active lesion some four months after foot-drop correction on the same foot (Patient No. 1646).

In two patients biopsy was taken during surgery to correct gross deformity at the acute phase of disintegration - Patients No. 1126 and 1493 (right foot).

In two patients biopsy was taken during reconstructive surgery to correct marked deformity with disability (Patient No. 1493 - left foot, and Patient No. 1656 - right foot).

(2) In South East Asia

Biopsies have been taken from a number of patients in Malaysia and Singapore during reconstructive surgery to correct gross deformities.

E. SELECTION OF PATIENTS FOR ARTERIOGRAPHY

In 1959 four patients were selected for arteriography because of existing lesions of tarsal bones which had recently been active.

Two of these patients had biopsies done at the same time. It was then decided that as the results showed no obvious arterial defect further investigations by arteriography were not warranted. Paterson reported in 1961 on arteriography in lepromatous feet and as a result, (1969) it was decided to examine by arteriography patients presenting with new tarsal bone lesions.

One patient, No. 1650, was examined before the breakdown of the radiographic equipment prevented further investigations along this line. Since the equipment has been repaired no new lesions have been detected in patients under treatment who could be investigated.

F. MATERIALS AVAILABLE ELSEWHERE IN SOUTH EAST ASIA

Since 1962 the investigator has taken part in a consultative service that is now available for leprosy institutions in South East Asia. This has enabled the investigator to examine and treat the feet of many leprosy patients in Korea, Taiwan, Thailand, Malaysia, Singapore and more recently, Pakistan.

Although medical records at some of these centres are not complete and radiographs have not been taken regularly, it has been possible to follow the progress of some of these lesions with and without treatment.

In many instances the bone lesions were not recognised at an early stage and even when visible in a radiograph were not reported on. The significance of lesions that were seen was not always recognised so that most of them were left untreated and have provided controls for comparison with treated patients. This situation is still unchanged in many institutions in the world where radiography is not freely available and where the significance of the lesions and the possibilities of treatment are not realised. This situation is resulting in increasing deformity and disability of the feet of many patients that could be prevented.

The observation possible under these conditions have confirmed the impressions gained at Hay Ling Chau, and have indicated that the lesions do not only occur in Chinese leprosy patients. It is, however, impossible to give any indication of the prevalence rates of tarsal bone lesions in other racial groups.

Most of the material used in this thesis will be that from Chinese patients, but for completeness material from other racial groups will also be used. In these cases the racial group will be mentioned and the centre where the patient was seen.

C. PATIENTS AVAILABLE FOR TREATMENT OF TARSAL BONE LESIONS

1) In the early years of the Hong Kong leprosarium patients were admitted because of contagiousness of their disease. Tarsal bone lesions were not looked for specifically on admission but those detected while under treatment were observed and some were treated.

After 1960 a growing awareness resulted in the detection of more lesions and the encouraging results of early treatment led to the acceptance that all lesions should be treated.

After 1964 a decrease in the number of patients needing hospitalisation for contagious leprosy allowed admission of selected non-contagious patients for rehabilitation and reconstructive surgery. These patients included a number of chronic or severe foot lesions who were admitted specifically for treatment of the foot lesion.

At Hay Ling Chau during the years 1950-1970 the investigator has personally treated or observed about one hundred patients with active tarsal bone disintegration. The numbers of patients under treatment since 1960 are listed on page 147.

2) At other centres the patients seen were those with "problem feet". Some were agreeable to treatment and some not. It was not possible to examine large numbers of patients with apparently normal feet in order to detect early lesions.

Chapter 4

METHODS

A. ROUTINE EXAMINATION OF PATIENTS ON ADMISSION TO HAY LING CHAU

Over the years a routine has been developed so that now on admission every patient undergoes an admission routine which includes:

(1) History taking to ascertain: -

a) Duration of disease; the patient usually under-estimates the length of the disease in lepromatous leprosy because of the equivocal appearances of the early lesions and the late anaesthesia.

B) Previous antileprosy therapy.

c) Intercurrent disease.

(2) Physical examination recorded by: -

a) Charting on a printed diagram of areas of anaesthesia as assessed by "feather touch".

b) Marking enlarged nerves on the diagram.

c) Listing of obvious deformities.

d) Listing of ulceration.

(3) Voluntary muscle testing to record abnormalities of function of the muscles of the hands and feet.

(4) Skin smear examination as described on page 157 to provide the Bacillary Index (B.I.) and Morphological Index (M.I.) readings.

(5) Skin biopsy, taken from the edge of an active lesion and examined to provide a histopathological diagnosis

(6) Radiographic examination of the hands, feet and chest. (For details of the radiographic foot examination see page 51).

While under treatment:

1. The R.I. and M.T. estimations are repeated every three months or more frequently until the R.I. has fallen to zero.

2. Physical examination is recorded every twelve months.

3. Voluntary muscle tests are repeated as necessary depending on the neurological state, but at least each twelve months.

4. The radiographs are repeated as necessary depending on initial findings.

A file is kept for each patient, to which reference can easily be made regarding his progress.

B. RADIOGRAPHIC EXAMINATION OF THE FEET IN PATIENTS AT HAY LING CHAU

Since 1965 two projections of the feet similar to those described by Kleiger (1963) have been taken routinely soon after admission: -

1) Dorsiplantar (DP). The patient sits or lies on the radiographic table with the knees sufficiently flexed so that the sole of the foot can rest on the cassette. The foot is positioned so that the toe tips and the head of the talus will be included. The tube is positioned directly over the tarso-metatarsal joints and the exposure is adjusted to allow adequate penetration of the tarsal bones even though this may cause some over-exposure of the phalanges.

2) Lateral (LAT). The patient lies on his side with the lateral border of his foot on the cassette. The foot is positioned on the cassette so that the tarsal bones will be visualised though the phalanges need not be included. The radiograph needs to be a true lateral view of the ankle joint, even in patients with a distorted forefoot.

In patients in whom suspicious lesions were seen on these radiographs, further films were taken. The commonest of these are: -

3) The dorsiplantar oblique (DPO). The patient is in the same position as for dorsiplantar projection, except that the knee is allowed to lean medially so that the transverse plane of the foot is parallel to the film. The tube is angled so that the ray is at right angles to the general plane of the dorsum of the foot and centred over the cuboid-navicular joint.

The superiority of this view over the standard PP view for visualisation of tarsal bone lesions led to its adoption for routine use in January 1970. A wooden cassette holder has been devised to standardise the angle of obliquity and allow better comparison of films to aid in the assessment of progression of a lesion under observation.

4) Lateral view with patient standing. The patient stands on the examination table with the lateral side of the foot facing the radiographic head, placed horizontally, and the cassette held vertically against the medial border of the leg and foot. A comparison of this view and the standard sitting lateral view will frequently indicate unusual mobility in the mid-tarsal joints and bony prominences that are subject to abnormal pressures when walking. These prominences are frequently not appreciated when the lateral view in the sitting position is the only one examined. The standing view also provides better visualisation of the ankle joint and enables the radiologist to detect abnormalities of the talus at an earlier stage than is possible if the radiograph is taken with the foot in plantarflexion.

Early in 1970 our staff developed a wooden cassette holder (illustrated in Fig. 1) to facilitate the positioning for this view and this position has now replaced the standard lateral view, whenever possible. By use of the standing view a more constant position is obtained for comparing changes in bone shapes.

Radiographic examination is routinely repeated in three to six months if lesions are suspected but not definite and in two to three years if the initial examination shows no abnormalities.

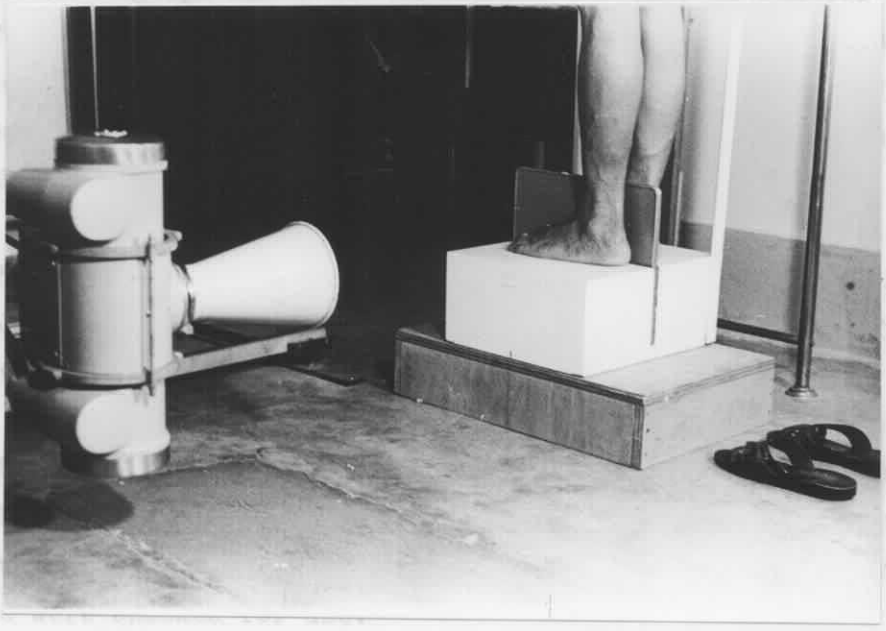


Fig. 1 The wooden cassette holder to facilitate positioning for the "standing lateral" position. This provides a weight bearing lateral view of the foot, with the film in a vertical position and using a horizontal tube projection.

C. EXAMINATION OF MATERIAL USED FOR PREVALENCE RATES

The radiographs of the 400 patients were examined and all definite lesions of the tarsal bones, the metatarsal bases and the lower end of the tibia and fibula were recorded. If the original radiograph showed an indefinite picture the examination was repeated after three months and if necessary again after three more months. If the radiograph was still indefinite the patient was listed as unaffected until such time as a definite lesion was seen on a further radiograph. The lesions of the phalanges and metatarsals that have already been well described in the literature have not been included in this study.

The routine admission examination as described on page 49 was completed and checked for completeness. Appropriate data from it was utilised.

Special note was made of the presence of sepsis on admission or at the time of detection of a new tarsal bone lesion. Voluntary muscular tests are regularly recorded by the Physiotherapist and these were checked for use.

D. BIOPSY

Recent biopsy specimens were removed surgically and fresh smears from the bone were made on glass slides for examination for acid fast bacilli. The specimens were fixed in formal saline and processed and reported on by the staff of the Hong Kong University Pathology Department.

The first two biopsies taken in 1959 were processed and reported on in London.

E. ARTERIOGRAPHY

Arteriography was performed under analgesia and local anaesthesia where necessary. Cut downs were used to enable injections to be made directly into the anterior tibial artery at the junction of the middle and the lower third of the leg, and into the posterior tibial artery behind the medial malleolus.

Serial radiographs were taken by use of a rapid change system.

F. TREATMENT OF TARSAL BONE LESIONS

During the years the investigator has been at Hay Ling Chau a routine for the treatment of patients with tarsal bone lesions has been devised. This is described in the following pages.

Meanwhile it should be noted that patients may be admitted with lesions at any stage of development so that the whole routine may not be applicable to every patient.

Concurrently with the specific treatment of tarsal bone lesions, a careful watch must be kept on:

(a) The use of antileprotic drugs:- that suitable drugs are taken by all patients, irrespective of the duration of the disease.

(b) Lepra reaction:- if this occurs it may require special therapy.

(c) Haematology.

(d) The intake of vitamins and calcium, as many patients need supplements of these.

(e) Self-medication with self-purchased drugs such as corticosteroids and extra analgesics.

SPECIFIC TREATMENT

The specific treatment of tarsal bone lesions will depend on the radiological findings in conjunction with the history and the clinical findings.

1. If the radiographs show no evidence of an active tarsal bone lesion then: -

(a) The foot is supported in a crepe bandage.
(b) The patient is advised to reduce the amount of his walking.
(c) Diuretics are given if oedema is marked.
(d) The foot is checked each week or more frequently for changes of heat and swelling.
(e) The radiographs are repeated in four weeks, and again in a further four weeks if signs persist in the presence of negative radiographic findings.

If the foot becomes more swollen and/or more heat is present but sepsis is not present, the radiographs may be repeated earlier and/or the foot completely rested by the use of crutches or bed rest. Some type of splint is used to prevent such a patient from walking until the radiographs are rechecked.

2. If radiographic signs of an active tarsal bone lesion are present the foot is immobilised. Ideally this is achieved by a plaster of Paris walking cast. If marked swelling is present the patient is hospitalised and the foot elevated for several days until the swelling subsides before the cast is applied.

The plaster is always applied under direct supervision of the medical officer in charge who personally maintains the desired position during the application. This is considered important as hypermobility of the foot may result from disintegration and it is possible to produce more deformity during plaster application. If the foot is carefully moulded during plaster application it is often possible to improve the functional position of the foot, so minimising future disability.

Consideration must also be given to:

(a) Anaesthesia of the skin of the patient. Special precautions are taken to protect the patient from plaster rubs.

(b) Muscular weakness. Paralysis or paresis of the anterior tibial muscles and a tendency to over-action of the posterior tibial muscles must be compensated for by correct placement of the ankle and foot, or else contracture of the tendo Achillis and inversion of the heel may result. Even with an avulsed tendo Achillis insertion it appears to the investigator to be advisable to immobilise with the ankle at 90° rather than in plantar flexion.

The next day a walking appliance is attached. This may be:

i. A Bohler type iron if the talus body, lower end of tibia, or body of calcaneum is involved. It is advisable that

weight be not transmitted through these bones if they are damaged, and the plaster may be adapted during the application to achieve this.

ii. A wooden or rubber rocker if the above bones are not affected.

iii. A flat plaster with a rubber and canvas sandal for patients who cannot manage a wooden rocker.

For details of methods of the application of the walking plaster and of the walking appliances as used at Hay Ling Chau, see Reprint, Appendix J.

The plaster cast is usually applied for about three months during which time the patient is encouraged to walk freely. After three months the patient is admitted to hospital and the plaster is removed, radiographs are taken, position is checked on the wet film, and the plaster reapplied at once. The patient is kept under supervision while the plaster is off to prevent walking without support, and after reapplication he is kept in hospital till the plaster is dry and he is walking again. This routine of plaster change is repeated about each three to six months until the radiographic and clinical signs suggest healing is complete.

The next plaster may be applied for six to eight weeks only and the patient is admitted to hospital before its removal. After removal of the plaster the radiographs are checked and if they are satisfactory and the foot is ulcer free the patient commences trial walking - see page 66 for details.

If clinical signs of incomplete healing occur, the plaster is reapplied for eight to twelve weeks and then radiographs and trial walking are repeated. This method of testing healing may need to be repeated on a number of occasions until the clinician becomes used to assessing healing. The duration of immobilisation needed for healing is included in the Results of Treatment, page 89.

3. Complicating osteomyelitis or sepsis of the foot is usually treated locally by dressings and soaks, with systemic antibiotics until the marked discharge has ceased. During this time the foot is maintained in a functional position by a supporting gutter splint made of plaster of Paris or Cramer Wire. As soon as the infection is controlled a plaster cast is applied. The first cast often requires changing in three to four weeks because of the discharge, and is usually not a walking plaster but is applied to maintain position.

After removal of this first plaster a repeat radiograph will enable the clinician to assess bone condition. Frank sequestra may need removal, but as a routine it is better to be conservative as otherwise tissues may be removed that could be salvaged. Early radiographs often suggest multiple sequestra that do not need to be removed but seem to heal normally with the rest of the bone (see Patient No. 1996, page 194).

Paterson, 1959 - page 254, pointed out that all loose fragments of bone, associated with non-specific bone changes, were not sequestra. They may show a relative increased density compared with the adjacent porotic bone but do maintain a blood supply and will frequently recalcify and assist in reconstituting the foot.

The routine use of plaster casts for these cases once the infection is controlled is the same as for non-septic cases and the prognosis is good.

4. If marked deformity of bone is present but no sepsis, surgical intervention is desirable. By multiple wedge osteotomies it is usually possible to reshape the foot to produce a functional one without dangerous pressure points. It is desirable at surgery to over-correct the deformity. Bone grafts may be desirable in the reconstruction of a foot that is still undergoing disintegration and in which bone collapse has resulted in deformity. Bone grafts may also be used in the reconstruction of the healed deformed foot but the investigator usually prefers not to use them on account of the scar tissue and the possibility of a poor blood supply and resultant delayed healing. Any hard sclerosed bone must be removed until cancellous bleeding bone is exposed and then internal fixation is used to ensure adequate approximation of the raw surfaces.

Various methods of fixation have been tried by the investigator.

(a) Charnley's Clamps are recommended by some surgeons and provide an efficient means of achieving a functional position in some wedge osteotomies. In some patients, however, it may be difficult to maintain the optimum position by using only one set of clamps.

In the presence of diminished pain perception and normal muscle power it is undesirable that fixation be removed early or non-union may result, (see pages 93 and 128.) Long continued use of the clamps increases the risk of sepsis, and in at least one patient sepsis was the cause of final failure with the rejection of a bone graft.

(b) Blount's Staples, available in many sizes, have proved invaluable in the investigator's hands. Wedge osteotomies in various planes can individually be closed under vision, ensuring bone apposition. Primary skin closure is possible,

minimising the risk of sepsis. Provided the staples are set deeply, there is little need of removal, though this is usually done before final discharge if the staples can be easily palpated, to prevent the risk of sepsis if trauma occurs later, producing an ulcer over the staple.

(c) Kirschner Wires - 1.5 mm diameter - are sometimes used to assist in maintaining the arch of the foot, especially when a bone graft is inserted and it is not possible or practical to bridge the graft with a large staple.

The investigator routinely uses Blount's Staples and/or Kirschner Wires which can then be left in situ for six to nine months, ensuring maintenance of position without fear of secondary infection.

At surgery a padded cast is applied. This is removed after three weeks and radiographic studies are made, then a well fitted walking plaster cast is applied. Special care is taken to mould the foot if possible to the desired shape so that healing in a functional position will be encouraged.

After a wedge osteotomy of the mid-tarsal area it is necessary that a plaster cast be worn for at least nine months before trial walking is attempted for the first time.

5. If the foot is badly deformed and complicated by sepsis the initial treatment is aimed at controlling the sepsis as described in Methods F3, page 60. Once the ulcer is healed the leg and foot are placed in a walking plaster for a further three months before surgical reconstruction is attempted as in Methods F4, page 61. Thereafter the management will be the same as in any other foot requiring surgical intervention.

C. TECHNIQUES USED AT HAY LING CHAU

1. Foot care

A large number of leprosy patients have anaesthesia of part or all of a foot when first diagnosed. Although recovery of sensation may occur in a small percentage of patients who report early, many others will eventually develop more anaesthesia.

An attempt is made to teach all patients to look after their feet and counter lack of sensory perception with routine watchfulness and personal attention. To achieve this, lectures are given and teaching posters are used to explain the problem of anaesthesia.

Every patient has his feet examined regularly by a medical officer and appropriate treatment ordered. This is basically:

(a) Soaking in water to correct skin dehydration.
(b) Paring of callosities to prevent ulceration from pressure.
(c) Oiling to counteract for the sebum deficit and to keep the skin supple.
(d) Suitable dressings for ulcers, and institution of rest in the case of ulceration on weight bearing surfaces.

The ideal can often be achieved even in the presence of marked deformity and much scar tissue. The patient can, if he is careful, keep his feet ulcer free and, in suitable socially acceptable footwear, can continue his daily work (see page 155 for Footwear in Leprosy).

2. Physiotherapy for foot lesions at Hay Ling Chau was only commenced in 1962. Since that time patients with any weakness of leg muscles have been taught to do their own physiotherapy, so that they can continue it by themselves. The aim is re-education of remaining muscle fibres and prevention of deformity caused by the over-action of unopposed normal muscles.

Any person with any weakness of the anterior tibial muscles is provided with a toe-raising spring. These are made by the patients to a simple design which has proved quite effective and is described in a Reprint included in Appendix J.

As a result of the use of toe-raising springs and physiotherapy, many patients have recovered near normal function who might otherwise have required corrective surgery or who may have developed marked deformity.

3. Trial walking

It is impossible to assess, on radiological evidence alone, if healing after disintegration is complete. After a number of disappointments the following routine was formulated. It does reveal most cases of incomplete healing and at the same time provides a gradual return to activity that assists in recalcification of the osteoporotic bones and helps reduce the possibilities of stress fractures and further episodes of disintegration. Trial walking is also used at Hay Ling Chau, with modifications, on all occasions when normal ambulation is being resumed after prolonged non-use of a foot due to ulceration, reaction, surgery, or complete bed rest.

(a) The affected foot is firmly bandaged.

(b) Suitable shoes with resilient insoles are laced onto the foot.

(c) Walking around the bed is allowed for three minutes on three occasions on the first day.

(d) After walking the foot is checked for heat and swelling.

This procedure is repeated daily if no heat or swelling develops but the time is increased gradually to five minutes on the second day and ten minutes on the third day, so that at the end of a week the patient is walking about, in the hospital, for thirty minutes three times a day.

If swelling of the foot occurs, as in Patient No. 1318, page 182, or there are other indications of impending trouble, the increases are taken more slowly and diuretics may be given, but if the swelling subsides before the next day it is usually only "travel oedema" and the use of the crepe bandage support helps prevent this.

If heat over the damaged bones occurs and does not subside over night the bones should be regarded as not fully healed and complete immobilisation resumed for a further six to twelve weeks.

Trial walking also helps to prevent breakdown of the skin of the sole such as frequently occurs when a patient with an anaesthetic foot resumes walking after a prolonged period of complete immobilisation or bed rest.

Chapter 5
RESULTS

Examination of the Materials described in Chapter 3 has enabled the investigator to make the following observations.

A. CLINICAL FEATURES

1. Early lesions

The earliest indication of tarsal bone disintegration is usually a slight degree of swelling, with or without heat, around the ankle or on the dorsum of the foot (Fig. 2). There is rarely any erythema and the swelling is usually not enough to cause pitting oedema. Pain or localised tenderness are only occasionally present. Crepitus cannot be elicited. The patient rarely complains of any disability unless the swelling is gross.

A history of noticed trauma is rarely obtained, but even in a normal foot prolonged walking itself may result in a stress fracture, a bone lesion or a peripheral neuritis. Ulceration or other infection is rarely seen in association with an early lesion (see page 99 for Prevalence Rates of associated sepsis).

Some neurological deficit is clinically detectable in the affected leg of 83% of patients who have tarsal bone lesions. This may be the only other sign elicited on examination (see page 97 for Prevalence Rates of the associated neurological deficit).

These vague signs may persist for several months, fluctuating in severity with the activity of the patient. If the patient is sedentary, healing may occur without any obvious deformity, but it is more usual for the signs to persist and some degree of deformity to develop (see Patient No. 780, right foot, page 171).

Fig. 2a Swelling of the foot which may occur with an early disintegrating lesion of the cuneiform-navicular area.

Fig. 2b Early disintegration of cuneiform-navicular joint area (Patient No. 847). This type of lesion may be asymptomatic.

With a mild lesion, by the end of six months the slight swelling becomes a firm, almost brawny induration which does not subside over night or during periods of bed rest. There may be little or no associated heat. With a rapidly advancing lesion, marked deformity may occur within a few weeks of the onset.

2. Advanced lesions

The clinical features of advanced lesions vary with their duration and severity. There is rarely marked heat and swelling unless there has been a recent reactivation of the bone lesion or there is associated sepsis. Mild degrees of heat and swelling may occur after periods of increased activity or there may be a degree of chronic swelling similar to a brawny induration (Fig. 3).

Deformity is usually present, but its severity and the resultant disability vary. The most common deformities are:

a) Minimal bony deformity that causes no obvious external deformity or disability (Fig. 4; and history of Patient No. 1754, page 190).

b) Marked varus of the heel - usually due to deformity of the talus in association with a muscular palsy (Fig. 5).

c) Collapse of the longitudinal arch of the foot (Fig. 6; and history of Patient No. 1318, page 181).

d) Reversal of the concavity of the sole of the foot ("boat shaped" foot) so that the cuboid bone is the most prominent point of the sole and this site becomes ulcer prone (Fig. 7). This is often due to a compression of the talo-navicular area which results from continued activity after talo-navicular softening has occurred (see Patient No. 1611, page 185).

Fig. 3 a & b External appearance and radiographic appearance in 1970 of the ankle of a patient who had continued to walk on a damaged talus for five years before agreeing to treatment (Patient No. 1699 - see Table 16, page 221).

Fig. 4 Minimal bony deformity of navicular. No external deformity or disability. Healed lesion of Patient No. 1657.

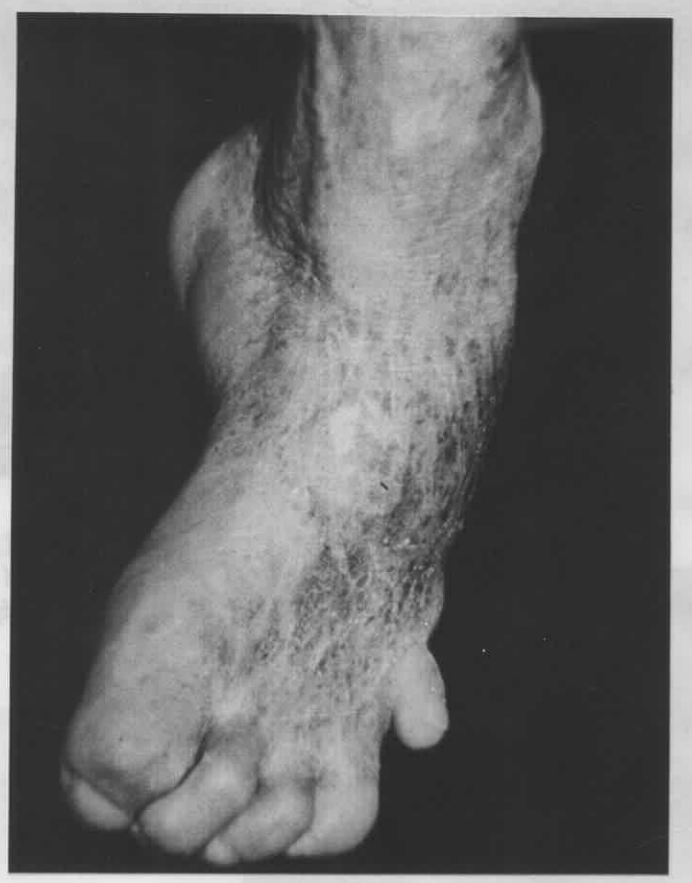


Fig. 5 Marked varus of heel was associated with paralysis of tibialis anterior muscle and over-action of tibialis posterior muscle. It resulted in marked ulceration of the lateral border of the foot and marked deformity of the fifth toe with loss of most of the fifth metatarsal.

Fig. 6a Collapse of the arch of the right foot

Fig. 6b Radiographic appearance of the above foot (Indian patient at Karigiri).

Fig. 7a Mildly "boat shaped" foot with chronic ulcer over cuboid prominence.

Fig. 7b Radiographic appearance of the above foot (Patient No. 1324). Impaction of talo-navicular area and collapse of arch has produced a prominence sufficient to cause chronic ulceration.

e) Hypermobility of the foot, usually through disorganisation of the mid-tarsal area (Fig. 8). Marked destruction of the talus and/or lower end of tibia and fibula may cause hypermobility of the ankle (Fig. 9).

f) Gross swelling of the whole foot in association with a rapidly advancing lesion such as that of Patient No. 1126, page 177, or in association with sepsis as in Patients No. 1154, page 179; and No. 1996, page 194.

The patient may present because of the disability or resultant ulceration. He usually can give a history of previous heat and swelling, though rarely of trauma. He may remember that the swelling commenced soon after the use of a plaster cast, a period of bed rest or some other period of limited ambulation.

It may be possible to palpate loose bone fragments in the foot or hypermobility may be present. In severe cases, the foot may appear as an appendage on the side of the tibia.

Fig. 8 Hypermobility of mid-tarsal area.

Fig. 9a Hypermobility of ankle.

Fig. 9b Radiographic appearance of the above.
(Elderly Chinese female in Singapore).

B. RADIOLOGICAL FINDINGS

1. Appearance of lesions

Radiological examination at the onset of clinical signs sometimes showed no evidence of a bone lesion. If the radiographs were repeated, lesions could usually be detected four to six weeks later. The lesions when first detected appeared as minimal fractures, either cracks or chips, osteoporosis, a vague haziness of one or more bones, or as frank disintegration.

Different patterns of lesions were observed: -

(a) Fractures of tarsal bones were usually associated with osteoporosis and minimal callus formation; the bone around the fracture tended to soften and then fragmentation and distortion of the remaining bone occurred with resultant deformity if the patient continued to use the unprotected foot (Fig. 10).

(b) In some patients fragmentation was the first abnormality detected (Fig. 11; and see Patient No. 1017, page 173); the fragments sometimes became displaced and were eventually absorbed or recalcified in an abnormal position, resulting in further deformity. On occasions, absorption of the fragmented bone proceeded to a marked degree until the radiograph suggested that there was complete destruction of the tarsal bones, as in Patient No. 1043, page 175; and Pattern 6, page 87.

The most common picture in an advanced unhealed lesion was a large area of disorganised bone showing irregular calcification and a tendency to fragmentation and loss of normal anatomical relations.

Fig. 10a Diagonal fracture of calcaneum (Chinese female in Malaysia) which was treated by bandage support and walking allowed.

Fig. 10b Marked deformity occurred within six months.

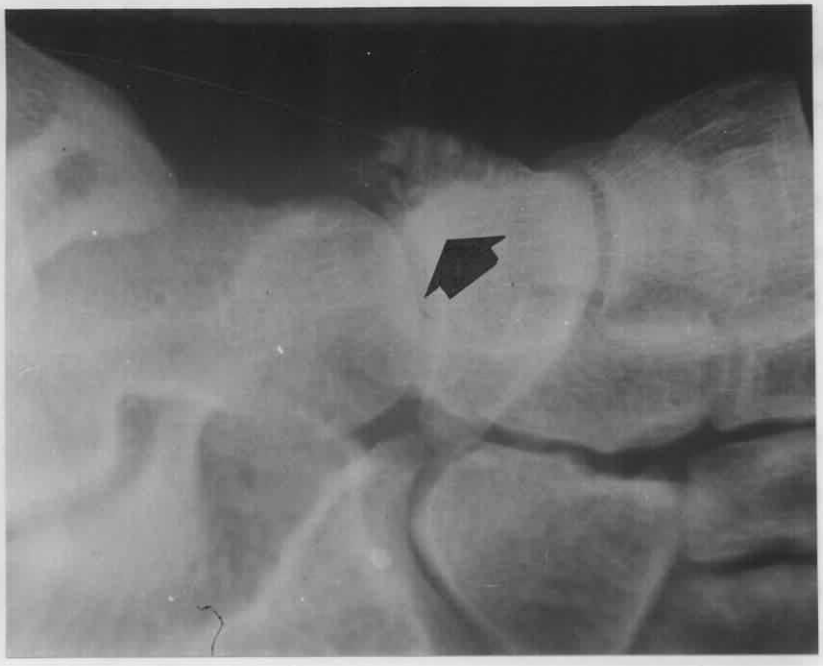


Fig. 11 Disintegration of the navicular without clinical signs. Patient No. 1646, see Table 15, page 220.

Fig. 12 Fracture of head of talus with extrusion of bone (Patient No. 1008).

(c) Fracture of one of the bones could be accompanied by what appeared to be an extrusion of portion of the bone through the break. This extrusion sometimes recalcified in the abnormal position or became detached and fragmented or was completely absorbed. This was most frequently seen to occur in the head of the talus and in the navicular (Fig. 12).

(d) In some patients a gradual change in the shape of a bone was observed over a period of time, in the absence of an obvious break in the cortex. The final shape of the talus depended on any associated muscular imbalance, the body of the talus becoming flattened, or the neck markedly angulated. The final picture was one of a deformed sclerosed bone presumably the result of the healing of a lesion within the body of the talus (see Figs. 13 and 14; and see Patient No. 1318, page 181).

(e) In some patients a vague haziness of the navicular and the head of the talus was the initial lesion observed, but several months later the patient developed a marked angulation of the neck of talus and collapse of the longitudinal arch of the foot, producing incipient ulceration at the pressure point so produced (see Patient No. 1318, page 181).

(f) In some patients osteomyelitis complicated the picture and it was impossible to determine, initially, which bones were affected by the infection and which by disintegration (see Patient No. 1154, page 179; and Patient No. 1996, page 194).

Lesions that have healed show increased calcification with or without deformity and this appearance persists for many years (see Patient No. 780, page 170).

Fig. 13a

Fig. 13b

Fig. 13 Comparison of the right (a) apparently normal foot and the left (b) affected foot, to show the distortion of the talus and the talo-navicular joint that resulted from slow activity during a period of two years. Male Chinese patient aged about 8-10 years (in Malaysia).

Fig. 14a
18 OCT 61

Fig. 14b
3 JUN 62

Fig. 14c
31 DEC 62

Fig. 14 a, b & c — Gradual change in shape of talus. Patient originally treated for fractured navicular by plaster of Paris walking cast. Talus damage was not suspected and changes continued after plaster was removed (Patient No. 1136).

The radiological findings are very variable but it would appear that any lesion of the foot bones in a leprosy patient may develop into disintegration.

The most common radiological findings are: -

a. A fracture of the head of the talus, not marked but with minimal displacement, that may be combined with extrusion of part of the substance of the talus. It may progress to complete disintegration of the head of the talus with collapse of the arch of the foot (see Figs. 6, 7 and 13).

b. Angulation of the neck of the talus with or without distortion of the body of the talus (see Patient No. 1318, page 181).

c. Disorganisation of the talo-navicular joint that may result in fusion of the joint or in an altered talo-navicular relationship, so that the dorsal surfaces are no longer approximately level and there is a tendency for the longitudinal arch of the foot to collapse (see Patient No. 1650, page 188; and Patient No. 1921, page 191).

d. Haziness, disintegration, or fracture of the upper portion of the navicular and/or talus. This frequently results in impaction and loss of the longitudinal arch of the foot (see Patient No. 1318, page 181; and Patient No. 1650, page 188).

e. Fracture of the calcaneum - this may be an avulsion of the tendo Achillis (Fig. 15), or a diagonal fracture similar to that caused by jumping from a height (Fig. 10; and Patient No. 1921, page 191).

Fig. 15 Avulsion of tendo Achillis (Patient No. 1650).

Fig. 16 Generalised disorganisation of several tarsal bones.

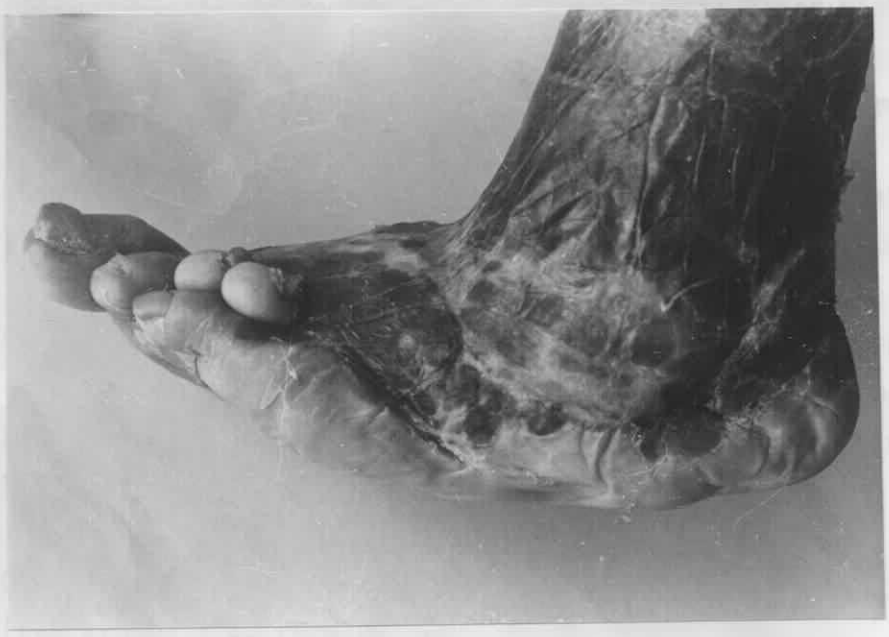


Fig. 17a

Fig. 17b

Fig. 17 a & b Deformity resultant from the involvement of several tarsal bones complicated by sepsis.

f. Generalised disorganisation of several tarsal bones with collapse of the longitudinal arch or other deformities (Figs. 16 and 17; and Patient No. 1126, page 171). Sepsis frequently develops as a secondary complication due to ulceration over a pressure point, as in Patient No. 1611, page 185.

g. Periosteal proliferation of the lower end of the tibia and fibula. This may appear as a single exostosis or as sheets of new bone and may involve the talus as well (see Patient No. 264, page 161).

h. Calcification of the interosseous membrane of the leg (see Appendix I, page 222).

i. Involvement of all the tarsal bones so that few anatomical land marks remain; one solid block of bone results if healing occurs (Fig. 18).

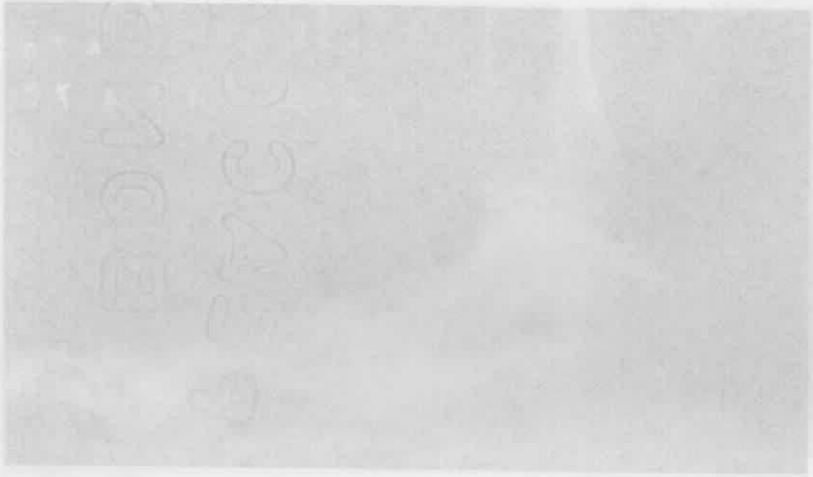


Fig. 18 Involvement of many tarsal bones in 1964-5 has resulted in one solid block of bone (Patient No. 1493).

Fig. 18a

JUN 64

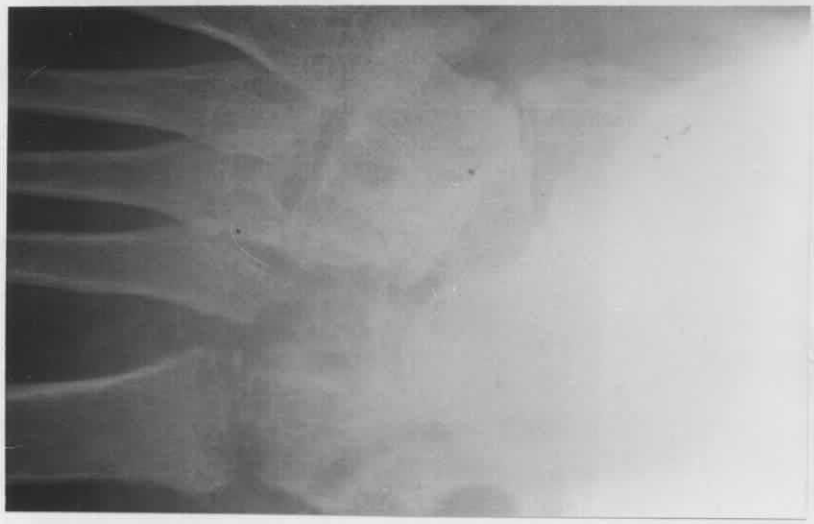


Fig. 18b

JAN 65

Fig. 18c

APR 70

Fig. 18 a, b & c Involvement of many tarsal bones in 1964-5 has resulted in one solid block of bone (Patient No. 1493).

2. Distribution of lesions

The distribution of tarsal bone lesions seen in the selected group of 400 patients has been analysed and is listed: -

Distribution of sites of lesions

Talus alone	8
Navicular alone	21
Navicular and talus	21
Navicular and cuneiforms	11
Talus, navicular and cuneiforms	16
Cuneiforms alone	1
Cuneiforms, with metatarsals and/or cuboid	13
Calcaneum alone	13
Talus, navicular and calcaneum	3
Cuboid, with an adjacent bone	4
Tibia, fibula and calcaneum	2
All tarsal bones	10
	117

(a) Fifty of these lesions involved the talus and the navicular, either alone or together. A further twenty-four lesions involved at least one of these two bones together with at least one other tarsal bone. In another ten feet all the tarsal bones were involved.

Thus the talus or navicular, either alone or in combination with one or more other tarsal bones, were involved in eighty-four feet - 72% of the lesions seen. No other tarsal bones were so commonly involved.

(b) The calcaneum either alone or in combination was involved in twenty-eight feet (24%). Avulsion of the tendo Achillis was the commonest lesion of the calcaneum.

(c) Cuneiforms were involved in forty-three feet (37%). Only one patient in the series showed isolated cuneiform involvement.

(d) If the lesion was located in one bone only it was most commonly seen in the navicular, but without treatment this type of localised lesion has been seen to spread rapidly to involve the talus and/or the cuneiforms.

Of the twenty-two patients who developed tarsal bone disintegration whilst under observation, four had the primary lesion in the talus, four in the navicular, one in the medial cuneiform, four in the medial cuneiform-metatarsal joint, and nine had a primary lesion of the calcaneum. One patient was not radiographed until all the tarsal bones were involved (Patient No. 1913). Hence it would appear from this investigation that the medial arch (Harris and Brand, 1966) and the posterior pillar of the foot are the two main sites for the primary lesion. Of the acute lesions of the calcaneum, six were avulsion of the insertion of the tendo Achillis and three of these followed surgery to the same foot, (see also page 112 and Appendix H, Table 14, page 219; and Appendix H, Table 16, page 221).

3. Arteriography

The five patients examined had stocking anaesthesia below the knee and paralysis of the anterior tibial muscles on the affected side.

Four patients without marked bony deformity showed no obvious abnormality of the large blood vessels (see Fig. 19 and Patient No. 771, page 168).

The fifth patient had marked collapse of the body of the talus with angulation of the neck of the talus and disorganisation of the talo-navicular joint. His arteriogram demonstrated an almost complete blockage of the dorsalis pedis artery adjacent to the site of the collapse of the neck of the talus (Fig. 20, and see history of Patient No. 1650, page 188).

(Details of these five patients are tabulated in Appendix F, page 206).

Fig. 19 Arteriogram showing good filling of the vessels of a relatively undeformed affected foot of the patient (No. 25) with disintegration of the navicular bone.

Fig. 20 Arteriogram of the deformed foot of Patient No. 1650 showing complete blockage of dorsalis pedis artery adjacent to the lesion.

C. DIFFERENTIAL DIAGNOSIS AND CORRELATION OF CLINICAL FINDINGS

In the early undeformed stage when there is minimal heat and little swelling, tarsal bone disintegration is most commonly misdiagnosed as a "sprain". Any "sprain" in the foot of a leprosy patient is to be suspected and should be well supported and reassessed at regular intervals. If the swelling persists for more than two to three weeks it should be assumed that a stress fracture has occurred and complete immobilisation instituted if radiographic studies are not possible (see Patient No. 1699, Table 16, page 221; and history of Patient No. 771, page 168).

In cases of minor fracture the first radiographs may not show the bone lesion. All patients with suspected stress fracture, sprains or other minimal trauma in which swelling persists longer than two to three weeks should be radiographically examined and the radiographs repeated after four to six weeks if signs persist.

Localised areas of swelling, but usually without heat, may be diagnosed as acute neuritis (see page 13). In neuritis the affected nerve is usually tender. Acute nerve lesions usually occur only in patients in very active or reactive phases of leprosy when they may also have large or tender nerves elsewhere. Neuritis may of course occur in association with or even be caused by the irritation of a tarsal bone disintegration, especially if it is the posterior tibial nerve that is involved.

Lepra reaction (see page 11) may be accompanied by neuritis or be associated with marked inflammatory lesions. These, if localised in the feet, may be confused with tarsal bone disintegration but are usually more widespread and associated with other generalised signs of reaction. The chronic hyperaemia of lepra reaction may lead to osteoporosis so that the foot of a patient with reaction is more prone to microtrauma in the course of the normal daily activities,

and the swelling of reaction may merge into that of tarsal bone disintegration. Thus the patient with chronic lepra reaction may therefore have tarsal bone disintegration as well, and so is in urgent need of regular careful radiographic studies to ascertain the condition of his tarsal bones from time to time (see Table 13, page 218).

Marked degrees of heat and swelling are frequently regarded as symptomatic of cellulitis, abscesses or other inflammatory conditions. Patients, suffering from tarsal bone disintegration, in whom incision and drainage had been attempted in the absence of lymphadenitis or a diagnostic leucocytosis, have been referred to the investigator for further treatment (see Patient No. 1043, page 175).

Antibiotics will do nothing to settle the swelling caused by tarsal bone disintegration. Complete rest of the injured foot will result in reduction of oedema and heat without the use of antibiotics, but they will return on the resumption of activity.

The presence of an ulcer or a sinus track should make one suspect sepsis and possibly osteomyelitis rather than primary disintegration, though osteomyelitis may progress to disintegration if ambulation is continued. The treatment of ulceration by plaster of Paris immobilisation for four to six weeks will assist in the healing of a tarsal bone lesion if it is present together with the sepsis. It is desirable, if possible, to make a correct diagnosis initially, as immobilisation for bone disintegration will need to be continued longer than the immobilisation used for the healing of an ulcer. If the diagnosis is still in doubt when the plaster is applied, then it is advisable to repeat the radiograph after the removal of the plaster and again four to six weeks later to make sure the bones are not showing signs of disintegration.

Traumatic fractures of the tarsal bones may occur and need to be considered as they will be painless in the presence of a generalised neural deficit. However, adequate radiographs should show a fracture, though a minimal fracture may not be seen until some osteoporosis has occurred around the fracture lines.

In all cases where any doubt exists it is advisable to treat for bone disintegration, as the lesions have been known to progress very rapidly and marked collapse and deformity have occurred within a few weeks of the first complaints of heat and swelling (see Patient No. 1582, page 183).

D. HISTOPATHOLOGY

The biopsies examined were taken from patients with different stages of disintegration and healing. Each biopsy showed individual features but the common findings may be summarised as follows: -

1. In some lesions the vascularity was increased and in others absorbed bone was replaced by vascular tissue. The lesions did not show the features of avascular necrosis.

2. Osteoclastic and osteoblastic activity proceeded side by side in the lesions. Healing sometimes results in normal bone and sometimes in callus-like tissue, fibro-cartilage, or fibrous tissue. Varying degrees of breakdown and healing were present in some lesions at the same time.

3. Articular surfaces were sometimes destroyed and replaced by fibrous tissue or fibro-cartilage. In some patients, patchy vascularisation of the articular cartilage from below was seen.

4. Focal areas of collapse were seen in some bones, especially in the sub-chondral tissues at the sites of stress.

5. No specifically leprous type of tissue reaction was seen even in lesions from patients with a high Bacillary Index (see page 157).

6. A low grade chronic osteomyelitis was present in some lesions.

7. At the time of biopsy, smears were taken from the bone lesions of some patients and were later examined for acid fast bacilli. None were found.

Details of biopsy reports are given in Appendix E, pages 196-204, and details of the patients examined are given in Appendix E, Table 1, page 205.

E. NATURAL PATTERNS OF THE DISEASE

Tarsal bone disintegration is a progressive lesion that usually leads to increasing deformity and disability, but spontaneous healing may occur. This progress can be divided into six patterns which are usually recognisable at radiographic examination.

Pattern 1 Minimal disintegration and healing without obvious external deformity

There is a stage of bone disintegration which results in minor irregularities of the bone contour but not in obvious deformity of the foot.

At the active phase, the foot or ankle will show heat and swelling but there will be no deformity. The patient will not complain of pain or disability. Radiographic examination may be negative for the first four to six weeks and after that time may show osteoporosis, early fragmentation or evidence of a fracture.

Many of these lesions are first seen when healing is occurring or complete (see history of Patient No. 1754, page 190). There is usually no clinical evidence of the lesion and the patient is unable to give any relevant history and is unaware of its presence. The radiographic examination may reveal a slight deformity of one or more bones. Serial radiography may show increasing sclerosis or stability of these lesions, indicating that healing occurs over a period of time.

Pattern 2 Marked disintegration with minimal deformity

A more marked disintegration of the tarsal bones occurs and proceeds, usually in association with attempts at healing until there is a definite deformity of one or more tarsal bones (see Patient No. 1921 - third lesion - page 192). The deformity, however, remains compatible with foot function. Frequently there is some loss of the longitudinal arch when the lesion involves the talo-navicular or cuneiform area, but not enough collapse to cause abnormal stresses sufficient to lead to ulceration and further deformity (see Patient No. 152, page 160; and Patient No. 780, right foot, page 171).

In active lesions there is usually heat and swelling though if the patient leads a sedentary life this may not be marked. Some increased mobility may also be present. Radiographically, a marked area of bone disintegration or collapse will be present.

Andersen, 1961, stated that a moderately flat foot may be an advantage as it tends to spread the stress over a larger weight bearing surface and eliminates normal take-off thrust. This could represent the healed Pattern 2 which may present as a non-swollen foot with a slight deformity, but no real disability. On careful questioning the patient will usually acknowledge that he previously had swelling, with or without heat, of the affected foot.

Lesions may remain slightly active for many years with minimal clinical signs and in these feet it is very difficult to ascertain if healing is complete. Several patients have been seen in whom activity was only recognised in retrospect though it had apparently been present for over ten years.

Pattern 3 Marked disintegration tending to healing with deformity

Disintegration may progress to severe bony involvement so that collapse of the arch of the foot (Fig. 6) and/or other marked deformity results. The general architecture of the foot is destroyed to a degree where it is impossible for the patient to walk without further resultant deformity and ulceration (Fig. 7).

The clinical picture is one of marked deformity usually of the "boat foot" variety (Price, 1964C - page 519) in which the plantar surface of the foot is convex with the cuboid as the most prominent point. Walking may cause ulceration at the most prominent point and secondary infection is common. This deformity is incompatible with normal footwear. The history of Patient No. 1611, page 185, illustrates how Pattern 3 type of deformity may develop.

Radiographic examination will show marked distortion of the normal anatomy, often with collapse of one or more bones. In the active lesions there will be a picture of disintegration and healing side by side and it may be difficult to state which bone was first involved or when healing is complete.

There may be hypermobility at the mid-tarsal area and this may heal as a pseudoarthrosis (Fig. 8). The resultant mobile foot is less prone to traumatic ulceration than is the rigid "boat foot", but will usually become ulcerated sooner or later.

Solid bony ankylosis of the ankle may occur as in Patient No. 454, page 166, in whom the resultant degree of varus predisposed to chronic ulceration of the lateral ray.

Pattern 3 is probably the commonest one encountered.

Pattern 4 Disintegration in association with sepsis

In this thesis sepsis is used to indicate secondary infection with organisms other than M. leprae. The association of sepsis and disintegration produces a varied clinical picture depending on the order of involvement and the severity of each. Sepsis may be superimposed on the patterns already described.

Clinically the patient may present with a hot swollen deformed foot that usually shows evidence of sinus formation or recent ulceration (see Patient No. 1154, page 179).

It is usually possible to divide these cases into one of two groups.

Firstly, there will be evidence of healed bone lesions with resultant obvious deformity which has predisposed to ulceration. Secondary osteomyelitis may be seen in association with the ulceration or sinus. In these patients the sepsis is secondary to the ulceration that has resulted from the abnormal pressures caused by walking on a deformed foot, but in some patients the history suggests that the primary lesion was a disintegration that showed heat and swelling. Incision and drainage was attempted with resultant secondary infection.

Secondly, gross osteomyelitis will be present without any sclerosis to indicate a healed lesion (Patient No. 1996, page 194). Unless the history is specific, it will be difficult to state if sepsis or disintegration was the primary lesion, but in some patients neglected primary trauma may be implicated as the initiating factor, as in Patient No. 2011, page 195.

Pattern 5 Spontaneously healed lesions with gross deformity

The previous patterns of tarsal bone involvement may progress to a stage when it is impossible for the patient to walk on the foot. He must resort to crutches or some other expedient that puts the damaged foot at rest and allows healing to occur. The remaining bones may reossify and the resultant foot, unless its position is controlled, may assume a very grotesque shape and become a useless appendage (Figs. 9 and 21).

If the patient has continued to walk he may do so on the distal end of the tibia with the foot as an appendage beside it, or with the forefoot flapping in front of a still functional heel.

Fig. 21 Gross deformity, no longer active (Patient No. 1598).

Fig. 22 Active disintegration without healing (Patient No. 1229).

Pattern 6 Progressive disintegration without healing

Progressive disintegration of the tarsal bones may continue without any attempts at healing until there is a marked destruction of some or all of the tarsal bones and it is impossible for the patient to walk (see Fig. 22). This may occur in spite of partial rest and minimal walking.

Clinically there is marked abnormality of shape and an affected foot has been described as "a bag of bones". There is rarely any heat unless secondary infection has supervened. The patient may, however, give a history of previous trauma, heat or swelling.

Radiographically there may be an apparently complete loss of some or all of the tarsal bones but recalcification of these will occur if the foot is adequately immobilised (see history of Patient No. 1043, page 175).

Any of these six patterns may result in healing or may progress to another pattern. It is unlikely for a lesion that shows healing to eventually develop into Pattern 6, so a progress from Pattern 5 to Pattern 6 is unlikely. Thus Patterns 1 to 5 may be considered as a continuing process and healing may occur at any stage in this process. It is possible by therapeutic means to encourage this healing.

The natural history of the disease left untreated is one of increasing deformity and instability in a foot that shows minimal clinical signs though heat and swelling are frequently present at some stage of the process. As bone deformity occurs the foot becomes more prone to ulceration and secondary infection is more likely to occur.

F. TREATMENT

1. Simple immobilisation using plaster of Paris

The treatment of patients with positive radiographic signs is described in Chapter 4, Section F, pages 58-63.

(a) The treatment of minimal lesions uncomplicated by deformity or sepsis.

Most early lesions showed definite signs of healing by the third month. Some patients with minimal lesions were able to commence trial walking (see page 66) at five months and proceed to full ambulation by the sixth month (see Patient No. 1646, page 186). If trial walking was commenced before the fifth month, heat and swelling usually occurred and the plaster had to be replaced.

Except in very marked lesions, healing was usually completed by the seventh or eighth month of immobilisation (see Patient No. 1017, page 173) and the affected bone appeared sclerotic on radiographic examination and remained so. This sclerosis was exaggerated by the relative osteoporosis of the unaffected bones (see case history of Patient No. 780, page 170). In no patient was a second lesion found in the same bone once healing was complete and walking was resumed.

(b) The treatment of lesions showing a marked degree of disintegration as yet unhealed, and uncomplicated by sepsis.

During the application of the plaster a number of these lesions were moulded into a satisfactory position and the resultant healed foot was stable and functional (see Patient No. 1582, page 183). Healing usually required eight to nine

months of immobilisation as these lesions usually involved the longitudinal arch. Early attempts at trial walking rapidly resulted in heat and swelling, and in some patients in further collapse of the arch.

In no patient did the tendency to disintegration continue while there was complete immobilisation.

(c) The treatment of lesions complicated by sepsis.

The length of time required to control sepsis varied from a few weeks to eighteen months but bone healing was occurring at the same time. The final results were satisfactory in those feet which were maintained in a functional position or were later surgically corrected. Bone healing did occur in the presence of sepsis but took a longer period of time, up to eighteen months in one foot with gross osteomyelitis on admission (see Patient No. 1996, page 194).

2. Surgical intervention at the acute stage of disintegration may be necessary

Bone grafts with adequate fixation will heal well and have enabled the investigator to restore feet to basically normal contours. Because it was usually the talo-navicular area that needed replacing, the foot was immobilised for a minimum of nine months before commencing trial walking. The bone grafts were usually well incorporated into the healing bone (see Patient No. 1126, page 177).

3. Surgical realignment after deformity had developed and bone healing had commenced or been completed

Most of these feet showed evidence of scar tissue and previous sepsis. Bone grafts were usually not attempted; wedge osteotomies with removal of sclerosed bone, internal fixation and long immobilisa-

tion resulted in sound healing (see Patient No. 1318, page 181).

Attempted use of bone grafts or a failure to remove all sclerosed bone sometimes resulted in non-union. The removal of adequate amounts of sclerosed bone appears to be an essential factor and must be accompanied by complete immobilisation (see page 61 for internal fixation, and page 58 and Appendix J for application of plaster of Paris casts).

C. COMPLICATIONS OF TREATMENT

1. Non-surgical

(a) Reactivation of disintegration occurred in a number of patients in whom normal walking was allowed at three months, as soon as the radiographic picture suggested healing. In some patients this disintegration progressed far more rapidly than it had before the period of immobilisation.

Healing again occurred when immobilisation was reinstituted. In the earlier years this did occur on a number of occasions and the case histories of Patient No. 402, page 163, and Patient No. 546, page 167, illustrate the progressive deterioration that may occur when immobilisation is not adequate.

(b) New lesions have developed in other tarsal bones on resumption of walking after immobilisation to achieve healing of the first tarsal bone disintegration. This usually occurred in patients who were allowed free walking at once, or who would not co-operate with trial walking (see Patient No. 1921, page 191; and Patient No. 1646, page 186). It was assumed that the new lesions were stress fractures of bones rendered osteoporotic during the long periods in walking plaster casts. The period of trial walking was instituted to encourage more normal bone healing and to teach the patient to be careful.

2. Surgical

(a) Sepsis. About a quarter of the patients treated surgically developed local sloughing and slow healing of surgical incisions. No patient treated surgically at Hay Ling Chau developed a degree of sepsis sufficient to significantly delay final bone healing. But at other centres sepsis was sometimes a problem when adequate supervision and facilities were not available.

(b) Non-union of the arthrodesis was seen in two patients who had relatively normal leg musculature, and in whom internal fixation was not used or in whom immobilisation was discarded early. It was also seen (at other centres) in association with sepsis and the early removal of Charnley Clamps which had been used for fixation. It was presumably due to incomplete immobilisation (see page 61 for Methods of Immobilisation).

(c) Poor positioning after wedge osteotomy caused unsatisfactory results in about ten of the early attempts at surgical correction, because of the failure to appreciate the need to over-correct the deformity and to remove the deforming force. In two patients, at another centre, with a fixed heel varus, associated with foot-drop, corrected by a modified triple arthrodesis, the deformity recurred within twelve months because the foot-drop was not also corrected and no type of toe-raising spring or supportive splint was used. Satisfactory results were achieved by further surgery.

(d) Recurrence of the deformity occurred in a few patients when free walking was allowed without a trial walking period three months after osteotomy of the mid-tarsal bones to correct collapse of the longitudinal arch. In one patient there was fracture of the metal staple used for internal fixation (Fig. 22) and in another there was bending of a Kirschner Wire which was used to maintain position.

Fig. 23 Fracture of Blount's Staple used as internal fixation for a mid-tarsal wedge osteotomy. Patient allowed free walking twelve weeks after surgery (Singapore Chinese female).

H. PREVALENCE RATES OF TARSAL BONE LESIONS

Four hundred patients (799 feet and legs) were examined to determine common factors in tarsal bone disintegration (Materials, Chapter 3, page 40; Methodology, Chapter 4, page 49 . Tables of Results in Appendix G, commencing page 207).

1. General statistics

94 patients (24%) had evidence of definite tarsal bone lesions.

18 patients (4.5%) had lesions in both feet.

112 feet (14%) were affected.

117 tarsal bone lesions were detected.

95 lesions were present on admission.

76 lesions were healed on admission.

19 lesions showed active disintegration on admission.

22 disintegration lesions developed while under treatment - i.e. after initial radiography.

34 patients (8.5%) showed a total of forty-one active disintegrating lesions.

4 patients developed one or more new disintegrating lesions in the same foot after healing of the initial lesion had commenced or even been completed.

11 patients admitted with healed lesions were admitted for correction of the deformity resultant from the lesions.

8 patients admitted with active lesions did so for treatment of the deformity and/or associated sepsis.

No patient who developed an active bony lesion whilst under observation developed severe deformity.

All patients included were under observation for periods between twelve months and six years (see also Appendix G, Table 3, page 207).

2. Side affected

There was no significant difference between the number of lesions affecting the right foot and the number affecting the left foot.

3. Sex

In 1969 the patient population at Hay Ling Chau was approximately 3.5 females to ten males, which proportion is similar to the sex distribution of the affected patients, as given in Appendix G, Table 3, page 207. Sex differentiation is not given in other tables.

4. Age

Six patients who developed lesions whilst under treatment were over fifty years of age and two were under twenty years of age. Patients in the older age groups had frequently suffered from leprosy for many years without adequate therapy and already had marked anaesthesia on admission. Table 4 shows the distribution of lesions by age on admission and it is obvious that a higher proportion of bone lesions were found in the older age groups. There is, however, no way of ascertaining the duration of a healed lesion or the age at which the lesion first occurred.

These statistics are tabulated in Appendix G, Table 4, page 208.

5. Duration of disease

An attempt was made to ascertain the duration of the disease but many patients could not give a reasonable estimate. The estimates varied from a few months to over thirty years, and on many occasions the clinical evidence did not support the patient's statement. This is in keeping with local custom of trying to hide leprosy as long as possible.

6. Classification of type of leprosy

(See page 9 for abbr viations of classifications).

The distribution of the selected patients according to the type of the disease suffered is listed in Appendix G, Table 5. It will be noted that 25% of the selected group of 400 patients had BB leprosy, of whom twenty-nine had bone lesions. These twenty-nine patients represent 31% of the affected patients.

The patients with LL leprosy constituted 39% of the selected group, but only 20% of the LL type patients showed tarsal bone lesions, representing 34% of the affected patients. The patients with TT leprosy are mostly treated as out-patients in Hong Kong, and admitted patients represented only 4.5% of the selected group. There was only one TT type patient with a tarsal bone lesion which was already healed on admission.

Patients with BL leprosy constituted 21% of the total group and 16% of the affected patients, while patients with BT leprosy constituted 10.5% of the group, and 18% of the affected patients. Five of the patients with BT leprosy had gross deformity of the affected foot and were admitted primarily for treatment of the foot lesion.

The patients with polar type leprosy, LL or TT, represented 43% of the admissions but only 35% of the affected feet, while the 57% of patients between the two extremes accounted for 65% of the affected feet (see Appendix G, Table 5, page 209).

7. Bacillary Index

The bacillary index (B.I.) is sometimes taken as a guide to the severity of the infection, and in LL leprosy, in conjunction with the clinical appearance, may assist in deciding how long the patient has had leprosy (see pages/57-8 for details of B.I. estimation).

The "on admission" bacillary index distribution of the selected patients is shown in Appendix G, Table 6, page 210, and accentuates the fact that a large proportion of new admissions have multibacillary forms of leprosy, which usually require prolonged therapy.

An attempt to correlate the B.I. level with the activity of the bone lesions was made. The thirty-four patients listed in Appendix G, Table 7, page 211, are those with active lesions. The B.I. reading used is the one taken nearest the time the lesion was first diagnosed. Of those with active lesions, 40% were already B.I. negative, and the remaining 60% are distributed fairly evenly over the whole B.I. range. No correlation is practical for patients admitted with healed lesions (see Table 3, page 207) in whom the lesion could have commenced a long time previously.

8. Associated clinical findings

a) A <u>neurological deficit</u> is often present in an affected foot.

(i) <u>Prevalence rate studies indicate that</u>:

1. 14% of all feet examined showed a tarsal bone lesion.
2. 33% of all legs examined showed normal sensation to touch below the knee.
3. 27% of affected feet had normal sensation to touch on the sole of the foot.

4. 22% of affected feet had normal sensation to touch on the dorsum of the foot.
5. 17% of affected feet had normal sensation to touch below the knee of the involved lower limb.
6. 38% of the affected feet showed stocking type anaesthesia below the knee of the involved lower limb.
7. 23% (181) of all legs examined showed stocking type anaesthesia below the knee.
8. 24% of the legs with stocking type anaesthesia showed a tarsal bone lesion of that foot.
9. Many patients showed a patchy superficial sensory loss, but assessment of deep sensation of the tarsal bones and associated areas was not attempted. Some loss of deep sensation may have been present in some of these patients.

See Appendix G, Table 8 and Graph, page 212.

(ii) Muscular paralysis or paresis

Voluntary muscle testing revealed that:

1. 13.5% of all legs examined showed complete loss of dorsiflexion of the ankle.
2. 6% of the feet examined showed tarsal bone lesions in association with a muscular paralysis or paresis.
3. 8% of the feet examined showed a tarsal bone lesion not associated with a detectable muscular paresis or paralysis.
4. 40% of the legs with a complete or partial loss of dorsiflexion showed a tarsal bone lesion.
5. 9% of the legs with normal dorsiflexion showed a tarsal bone lesion.
6. 43% of the affected feet showed absent or weak dorsiflexion of the ankle.

7. 57% of the affected legs appeared to have a normal muscular function.
8. 16% of the affected feet (eighteen patients) showed no obvious sensory impairment or muscular involvement in the affected lower limb.
9. In 2% of patients (seven) the side having a tarsal bone lesion showed less obvious neural involvement than the unaffected lower limb.

See Appendix G, Table 9 and Graph, page 213.

b) Association of ulceration and frank sepsis

The results of the study into the prevalence of ulceration in association with a tarsal bone lesion are tabulated in Appendix G, Table 10, page 214.

It showed that: -

1. Ulceration was present in twenty-five patients (22% of the affected feet) at the time when the tarsal bone lesion was first diagnosed. But in some of these patients the bone lesion was healed and the sepsis was of recent origin.

2. One patient developed a tarsal bone disintegration after removal of a walking plaster that had been applied as treatment for osteomyelitis. Disintegration after the use of walking plaster casts occurred in about 10% of patients who, prior to 1965, were treated for ulceration by this method or by prolonged bed rest (see also page 25; case history of Patient No. 1017, page 173; and Patient No. 1582, page 183).

3. In five patients (six feet) a bone disintegration developed in the operated foot within six months after foot-drop correction was performed. This represented 7.5% of the total of seventy-four foot-drop corrections performed on the selected patients. In each of these six cases there was some other complicating factor, usually prolonged immobilisation, as well as possible sepsis. In only one patient (No. 1882) was there definite sepsis on the affected foot (see Appendix H, Table 14, page 219).

4. In the selected patients twenty-six tarsal bone lesions developed whilst they were under observation. Of these, six were post-operative (see 3. above). Seventeen were free from sepsis, giving a prevalence of 85% of non-post-operative lesions as being free of sepsis.

5. Of the patients admitted with active bone lesions, ten had no obvious associated sepsis and nine had marked sepsis and marked deformity. The appearance in eight of these latter patients suggested that the sepsis was resultant from the deformity (see also pages 103-104). The ninth patient (No. 2011) gave a history suggesting that the sepsis preceded the tarsal bone lesion, and that the tarsal bone lesion was the result of continued activity in the presence of osteomyelitis (see also history of Patient No. 2011, page 195).

TABULATED RESUME OF PREVALENCE RATES

1. 24% of the selected Chinese leprosy patients showed definite evidence of tarsal bone lesions - either healed or active.

2. 14% of the total number of feet examined showed lesions of one or more tarsal bones.

3. There was no significant distribution difference between the lesions of the right and left foot.

4. There was no significant distribution difference between the sexes.

5. There was some indication that borderline type leprosy patients were more susceptible to tarsal bone lesions than patients who developed polar type leprosy. Only 35% of lesions occurred in patients with polar type leprosy, although these constituted 43% of the selected patients.

6. There was no obvious relationship between the bacillary index and the prevalence of lesions. Patients who developed new tarsal bone lesions were from all B.I. levels.

7. The incidence of lesions was relatively higher in the older age groups but many of these lesions were already healed. There is no indication that any age group is specifically at risk.

8. A large proportion of patients who develop tarsal bone disintegration have anaesthesia and/or paralysis of the affected leg. However, 17% of affected feet showed normal superficial sensation and 57% of affected feet appeared to have normal muscular function.

9. All the tarsal bones may be affected, but the talus, navicular and calcaneum are the most frequently involved.

10. Infection may be present in association with a tarsal bone lesion. Tarsal bone disintegration may occur which is apparently secondary to an infective process. However, some well documented cases show that sepsis is not a necessary predisposing factor and that tarsal bone disintegration can occur in its absence.

11. Gross deformity resultant from continued usage of the affected limb is often associated with sepsis. This deformity can itself be the cause of ulceration and hence of the secondary sepsis and osteomyelitis. With grossly deformed feet it is impossible to decide which bone was affected initially or whether sepsis was a primary or secondary factor.

12. Routine clinical examination cannot eliminate the presence of tarsal bone disintegration and every leprosy patient with warmth or swelling of the ankle or foot should be considered as suspect until proved otherwise.

I. AETIOLOGICAL FACTORS

To assist in determining the causative factors of tarsal bone disintegration, a more detailed study of the patients with active lesions was undertaken. These were divided into two groups: -

1. Active lesions present on admission. There were seventeen patients in this group with nineteen lesions, which were subdivided according to the presence or absence of sepsis of the affected area. Sepsis is here taken to imply a secondary infection with an organism other than M. leprae.

   (a) Non-septic

   The lesions were present in nine patients (ten feet), each of whom presented with virtually untreated highly active leprosy of LL, BL or BB type.
   Eight patients showed evidence of neuropathy.
   Six affected legs showed stocking anaesthesia below the knee.
   Five affected feet showed a complete, and one a partial, foot-drop.
   Four patients gave a definite history of at least one episode of swollen feet. Two of these were definitely due to lepra reaction (one with bilateral lesions), and it is likely that others were also due to lepra reaction.

   Only one patient gave a history of a foot injury. He fractured his ankle three months prior to admission (Patient No. 1680).

   Details are given in Appendix H, Table 11, pages 215-216.

   (b) Marked sepsis

   There were nine lesions in eight patients, complicated by sepsis and ulceration.

All were of BB or BT type leprosy and seven already had a negative or near negative bacillary index. All had received treatment, usually intermittently, for many years.
Seven patients showed involvement of all tarsal bones.
All affected feet had stocking anaesthesia below the knee and seven showed loss of deep pain sensation as well.
Seven feet showed complete foot-drop, one other foot showed clawed toes but no drop.
Only one patient gave a definite history of trauma and sepsis preceding the deformity. He also retained deep pain sensation and normal muscle function (Patient No. 2011, page 195).
The other eight feet showed such complicated patterns of disintegration and repair, associated with sepsis, that it was usually impossible to decide which was the initial lesion, the deformity, or the sepsis (see page 85).

Details are given in Appendix H, Table 12, page 217.

2. Active lesions developing whilst under treatment. There were seventeen patients in this group who developed twenty-two tarsal bone lesions and these have been divided into four groups. Only two of the non-post-operative patients had sepsis present immediately preceding the diagnosis of the lesion.

(a) Patients with lepra reaction

Lepra reaction is described on page 11 and its treatment on page 153. It is often associated with swelling and ulceration of the legs and feet, and also with general malaise, so that patients are disinclined to exercise or even continue their routine activities.

Six patients, as listed in Appendix H, Table 13, page 218, developed disintegrating lesions which were diagnosed during or soon after periods of lepra reaction.

Three patients had chronic swelling and hyperaemia of the feet for many months but all had swelling prior to the diagnosis of bone disintegration.
Two of these patients with reaction (three lesions) are known to have taken Prednisolone in the few months preceding the finding of a disintegrating tarsal bone. One of these patients (Patient No. 1640) was self-medicated with Prednisolone and developed chronic infection after surgery to his toes at that time, which immediately preceded the diagnosis of tarsal bone disintegration. A year later he was again self-medicated with Prednisolone and on that occasion fractured several vertebral bodies.
In only one other non-post-operative patient was sepsis present at or immediately preceding the bone lesion (Patient No. 1921, page 191).
Only one patient had normal muscle power in the affected foot; all patients showed areas of anaesthesia on the affected leg.

(b) Five patients are listed in Appendix H, Table 14, page 219, in whom six lesions were first seen soon after surgery to that foot.
In each case the operation was foot-drop correction, using tibialis posterior as a tendino-muscular transfer.
Some degree of anaesthesia was also present as well as the paralysis.
Three of these lesions were avulsion of the tendo Achillis in patients who had been hospitalised for long periods before surgery.
Two lesions were of the mid-tarsal bones in a teenaged boy (Patient No. 1656) resulting in loss of the longitudinal arch of the foot - but developing slowly for several months after surgery. This was probably not directly attributable to trauma at the operation or in the post-operation re-education period.

One patient had obvious sepsis of the affected foot immediately following surgery (Patient No. 1882).

Of a total of seventy-four foot-drop corrections performed for the 400 patients reviewed, only six (7.5%) of them developed post-operative bone lesions in the operated foot, and in each of these patients there was some factor that caused an abnormally prolonged period of non-ambulation, either before or after surgery.

(c) Patients who developed tarsal bone lesions after the use of a walking plaster

Three patients are listed in Appendix H, Table 15, page 220, who developed five bone lesions, each of which was diagnosed soon after the removal of a walking plaster cast. Each of these patients had some degree of anaesthesia and muscle weakness of the affected leg.

A total of forty-four patients out of the selected 400 had walking plaster casts applied for treatment of foot ulceration. Only one of the patients in this series, treated in this way, developed a tarsal bone disintegration after the removal of the plaster cast.

The patients included in this series were admitted for treatment between 1963 and 1968.

It was during 1963-4 that we realised that bone disintegration occurred in over 10% of those patients who were treated for foot ulceration or bone disintegration by the use of a walking plaster cast and were allowed to resume normal ambulation at once on the removal of the cast. The routine of trial walking was instituted in 1964-5 and since then

the incidence has progressively dropped until now it is almost zero. The duration of immobilisation is a determining factor in the prevalence rate as disintegration rarely is seen in patients immobilised for less than six weeks if they were normally active before the plaster was applied.

The third patient, No. 1921, in Table 15, page 220, was advised re trial walking, but refused to co-operate and his case history, given in full on page 191, is similar to several others that were treated in 1960-64.

(d) Four patients who developed tarsal bone lesions while under observation but in whom no common factors can be elicited are listed in Appendix H, Table 16, page 221.

Two of these were elderly women whose normal activities were very limited. One patient (No. 1766) had marked osteoporosis. The other patient (No. 1831) was confined to bed for many months in an attempt to heal an ulcer on the right foot and when walking was resumed she avulsed the tendo Achillis insertion in the totally anaesthetic left foot on which a foot-drop correction had previously been performed. Osteoporosis is assumed to have been a major causative factor in both these women.

One patient (No. 1722) had no relevant history that could suggest a specific causative factor. He had stocking anaesthesia and complete drop of the affected foot and the lesion, already healed, was detected on routine radiographic examination.

One patient (No. 1699) complained of a twisted ankle. He had stocking anaesthesia and a complete drop of the affected

foot. The radiograph showed a fractured calcaneum and abnormal talus. The calcaneum was healing well when the patient absconded after six months treatment in a walking plaster cast. He eventually returned four years later, having had no further treatment. The calcaneum was stable but activity of the talus had continued until destruction of the bones of the ankle had occurred and this had resulted in instability and deformity of the ankle, so that it was no longer possible for him to weight bear on the foot (see Fig. 3 for illustrations of the condition of the ankle on readmission in 1970).

J. THE RESULTS OF THE APPLICATION OF MEASURES FOR THE PREVENTION OF TARSAL BONE DISINTEGRATION

In the years prior to 1964 only obviously involved feet were radiographed in the lateral position. A number of disintegrating lesions were presumably not diagnosed, and some probably progressed to spontaneous healing. However, during 1963 the staff were beginning to suspect these lesions and more patients were radiographed and some early lesions were detected.

In 1964-65 trial walking (page 66) was instituted as a routine and since that time the incidence of lesions has steadily fallen, although every patient now has routine lateral and DP radiographs.

The number of lesions suspected or actually disintegrating that were detected in patients under treatment is: -

1956 - 1959	26 lesions (mostly advanced)
1960 + 1961	13
1962 + 1963	14
1964 + 1965	23
1966 + 1967	11
1968 + 1969	8
1970	3

Of the lesions detected in 1970 two were avulsion of the tendo Achillis in patients who had been relatively inactive for a long period of time, and neither really developed disintegration. The other was Patient No. 1582 whose history is given on page 183, in whom possible disintegration was predicted, but precautions were not taken and free walking was resumed rapidly.

It is felt that with alert medical staff it should be possible to prevent most lesions in co-operative patients with adequate medical treatment, physiotherapy and foot care, though there will always be a small group of patients who develop these lesions because they do not accept the treatment of choice.

Chapter 6
DISCUSSION

Tarsal bone lesions in leprosy patients have long been recognised because of the resultant disabilities but until the last decade little has been written about the development of these lesions and their treatment. In 1961, Andersen wrote of the lack of understanding of the pathogenesis of tarsal instability and deformities. He stated that inconsistant results had followed attempts at surgical correction. This present investigation has produced results that permit the correlation of the findings of other workers and presents a picture of the condition as a whole.

The early lesions of tarsal bone disintegration as seen in this series were described by Paterson and Job (1964 - page 441). They also mentioned the problem of collapse of weakened bones and of superimposed secondary infection.

Serial radiographs have been studied by the investigator; they show the progress of tarsal bone disintegration from minimal lesions to gross deformities in patients who have had no treatment at all, or who have received insufficient treatment. In some cases, patients resumed unrestricted activity before healing was complete, and further disintegration occurred. A study of this series has provided the basis for description of the natural history of disintegration, thus providing an understanding of the nature of the disease, its progress, the effects of treatment, and the final outcome.

The progressive nature of tarsal bone lesions as described under Patterns of Disease in this dissertation (see pages 80-86) was stressed by Price (1960A), who also warned of the final deformities and disabilities that could result. Later, in 1964C - page 519, he described the "boat shaped" foot (Pattern 3 - page 82 in this dissertation) and the problem of keeping it free from ulcers. In 1964-O he suggested that

the collapse of the foot which may follow osteoporosis and disuse atrophy, may provide a good "rocker" foot so that the foot may remain free from ulcers, despite walking. In the investigator's experience, any foot in which the deformity is sufficient to produce a "rocker" foot is usually prone to develop an ulcer on the point of the rocker. Milder degrees of collapse in which the arch has flattened (Pattern 2, page 81) but no rocker has formed, may in fact remain ulcer free if the patient wears suitable shoes and does not abuse his foot. This type of foot is described by Lennox (1964, 1965) as occurring by a gradual descent of the head of the talus until there is an almost complete collapse of the longitudinal arch.

Johnson (1967) refers to neurotrophic bones and joints in general, but the lesions described are very similar to those seen by the investigator. He speaks of the alternating patterns of destruction and repair that can produce very bizarre results and stresses that healing can occur. Further destruction can also occur, however, if activity is continued or resumed before healing is complete. This sequence has been seen by the investigator on several occasions, especially in non-co-operative patients. He also mentions that very few text books describe the natural course of the changes which occur in neurotrophic joints and that most writers neglect the importance of fractures in initiating neurotrophic joint changes.

The various stages of healing and repair have been recognised during operations on the foot when healed bones are found to be sclerotic, whilst other bones in the same foot may be soft and osteoporotic. Karat et.al. (1968) mention the hardness of healed metatarsals. It has been observed by the investigator that healed tarsals are as hard as healed metatarsals and that sclerotic bones do not seem to become soft even after prolonged immobilisation.

Most references in the literature are to the tarsal bones as a whole with no differentiation regarding lesions of individual bones.

In 1966 Harris and Brand divided the lesions into patterns of destruction but their patients were selected for radiography so that there are no comparative figures available. Their impression was that the calcaneum was one of the bones most frequently injured. In our series 24% of affected feet showed calcaneum involvement.

Harris and Brand also stated that the talus was rarely the site of the primary lesion. In our series, the talus was involved alone in four feet - 19% of the patients who developed lesions while under observation, and the talus was involved in 44% of all affected feet.

The Harris and Brand classification of "anterior pillar - medial arch" includes the most common and most deforming lesions that occurred in our series. They stated that collapse in this pattern was common and consistant; the present investigator found 72% of lesions came into this category.

Harris and Brand found that lesions of the cuneiform-metatarsal base were not common, and nearly all appeared after definite violence. In this series, the investigator found thirty-five lesions involving one or more cuneiform bones (30% of affected feet). A primary lesion of the cuneiforms was seen in only one patient in this series but has been occasionally seen in patients not in the present series (see case history of Patient No. 1126, page 177).

Paterson and Job (1964 - page 441) stated that probably 2% of patients in leprosaria develop tarsal bone disintegration. In our series 24% of such patients had tarsal bone lesions. Some lesions had already healed when the first radiograph was taken. In these patients it was not always possible to be certain that disintegration had occurred, though in some patients the resultant deformity suggested that the lesion was really a disintegration.

Out of 400 patients examined, thirty-four showed active disinte-

gration radiographically; this would suggest that disintegration affects at least 8.5% of patients in leprosaria. The figure may be higher as others of the 400 have developed new lesions in the past twelve months since the survey was done and these are not included in the figures given.

Hence the incidence of tarsal bone disintegration would probably be higher than 8.5% if the series of patients was followed up for a longer period.

In this series of 400 patients, a total of ninety-four patients showed 117 tarsal bone lesions in 112 feet, giving a prevalence rate of 24% of patients and 14.0% of feet examined. Both feet were affected in 4.5% of patients. This is a much higher figure than the figure of 2% which was quoted by Paterson and Job. The rate we quote of 24% does probably include some lesions that may not have been disintegration initially, as seventy-six of them were already healed on admission. But the nature of the deformities present on admission would indicate that a large proportion of these lesions were in fact tarsal bone disintegration. Twenty-two lesions of definite disintegration appeared during treatment and these occurred in nineteen patients, so that disintegration occurred whilst under treatment in 4.5% of the patients investigated. In addition, there were nineteen active lesions in seventeen patients, on admission; a prevalence rate of 4.25% of patients investigated. Note that two patients, having active lesions on admission, each developed a second lesion later, so they appear in both groups of figures. Hence the total number of active lesions seen is forty-one in thirty-four patients, giving a prevalence rate of 8.5% of investigated patients who showed one or more active lesions during treatment.

Most writers make no attempt to estimate the prevalence of this condition. Karat et. al. (1968) stated that the Charcot type of disintegration is rare in leprosy patients. Harris and Brand (1966) stated that while theirs was not a statistical study, they considered

that disintegration of the tarsus was not as common as erosion of the more peripheral bones (e.g. phalanges). The investigator considers that this statement may be correct, but wishes to point out that a large proportion of peripheral erosions produce no obvious deformity or disability and many are frequently only slowly progressive, whereas tarsal bone lesions frequently cause deformity and marked disability, and may progress rapidly.

In 1960A, Price commented on the fact that the frequency of neuropathic joints in leprosy is under-estimated in the absence of radiological facilities. It would also be correct to state that it is under-estimated when it is not positively suspected, as the average radiograph of the foot is not taken with the prime objective of showing the commonly affected bones (see page 51 for positioning).

These figures of the incidence of tarsal bone disintegration support the plea made by Price (1960A) that some pathological changes in the joint should be suspected in any leprosy patient with an unexplained, painless or moderately tender swelling of the foot or around the ankle.

Heat and swelling are accepted clinical signs (Lennox, 1964) and in the investigator's experience may be the only early ones.

The differential diagnosis may be difficult. Price (1960A) commented on the difficulty of clinically differentiating between "a spreading infection" and an "early neuropathy". When sepsis is present, the heat and swelling will usually subside in a few days if the part is rested and the patient given suitable antibiotics. Price (1964C - pages 523-4) used a lack of this improvement as a criterion of diagnosis, but in the investigator's experience the swelling accompanying tarsal bone disintegration will also subside if complete rest is enforced for a week or so, but it will return as soon as walking is resumed. Rapid resumption of walking after recovery from a septic foot may also result

in return of oedema and swelling; observation for longer than a week may be needed to make a definite diagnosis.

Harris and Brand (1966) in describing the physical signs mention changes of gait as important and Lennox (1964) adds mobility of sub-talar or mid-tarsal joints with crepitus. These are, however, usually late signs due to the deformity and not due to the disintegration process. By the time this deformity has developed there is frequently little or no heat and swelling of the foot.

It has been stated by Cochrane (1947 - pages 52-53; 1959 - pages 138-9; and 1964 - page 275; and Karat et al. 1968) that tarsal bone lesions are usually associated with ulceration. Harris and Brand (1966) acknowledged the presence of sepsis in many patients but concluded that its role was not obvious. Price (1960A) pointed out that neurotrophic lesions occur in the absence of sepsis but may often be associated with plantar ulceration. The biopsies in the present investigation do not support the theory that sepsis plays a role in the causation of these lesions.

Specific leprous granuloma of bone has been described by Job (1963) and has been observed to heal during medication with antileprotic drugs (Erickson and Johanson, 1948; Paterson and Job, 1964 - pages 426-7) being replaced by fibrous tissue.

However, this specific granuloma was not found in the biopsies examined in this series. The state of healing seen represented a wide range, and is probably to be correlated with the duration and the rate of progression of the disintegration at the time of the removal of the biopsy.

Johnson (1967) pointed out that many orthopaedic text books describe the well developed neuropathic joint of the Charcot type, but not the early stages of the condition. Some of the biopsies in this

investigation were of the earlier stages and this may explain the observed differences.

The biopsies did, however, show evidence of increased vascularity, which may be associated with either bone destruction or bone formation (Johnson, 1963 - page 636). This by itself may lead to softening of the bone and hence render fracture from minor trauma more likely (Johnson, 1967). This increased vascularity does not support a suggestion that avascular necrosis is possibly the basic pathological lesion.

Price (1964C - page 523), in discussing the pathology of neurotrophic lesions, stated that the articular cartilage collapses as its blood supply fails.

Browne (1969) stated that in leprosy vascular lesions may cause degenerative changes or aseptic necrosis but does not specifically mention tarsal bone disintegration.

Watson Jones (1952 - page 67) pointed out that avascular necrosis may be diagnosed early by the relatively increased density of the affected bones as compared with the porosis of neighbouring bones. It has been shown by Bobechko and Harris (1960) that in experimental avascular necrosis of the head of the femur in rabbits the new bone is laid down over the dead bone, and that the increased density is due, partly at least, to the increase in the total bulk of bone. In these rabbits the increased density was detectable within three to eight weeks. In man the rate of reossification will vary with the size of the avascular bone lesion. This early increased density is in contrast to the findings in tarsal bone disintegration in leprosy when an early decrease in density in the affected bone is relatively common and sclerosis is a late finding. In early tarsal bone disintegration the neighbouring bones are of normal density when compared with the relatively porotic disintegrating bone. Biopsy findings

in the present investigation do not support the suggestion that avascular necrosis is the basic pathology (see page 79). Some of those examined did, however, show aseptic necrosis.

The increased vascularity found in some of these biopsies is in agreement with the basically normal vascular patterns of major blood vessels shown in the arteriograms of the undeformed patients.

Bang and Tiep (1958), Lechat and Chardome (1958), Paterson (1961), and Lechat (1962) each reported various abnormalities detected by arteriography in the feet and hands showing "mutilation", deformities, or bone absorption. An abnormality of major blood vessels found in one patient in this series who had a marked bony deformity is in agreement with these findings.

However, no reference could be found to arteriography being carried out on patients without obvious deformities, or disabilities, as were the first four investigated in this series. In view of the absence of abnormalities in these patients it is surmised that marked vascular abnormalities are secondary to the bone changes and not a causation.

Paterson (1961) also mentioned the presence of excessive hyperaemia with local inflammation. Paterson and Job (1964 - page 443) stated that they could find no evidence to support a theory that vascular defects were the prime cause of bone changes in leprosy.

Most disintegrating lesions will eventually become obvious during serial radiography, but in the absence of radiographical examination, diagnosis may be difficult. "Bunching of tarsal periosteum" has been described by Andersen (1969) as an incipient form of disintegration, but this cannot be recognised by the investigator unless it corresponds to "lipping".

The latter has been seen to remain constant for many years and not to proceed to disintegration, though it could be the healed result of a mild disintegration resulting in little deformity and no disability (Patterns 1 and 2, pages 80–81 of this dissertation).

Radiographic descriptions of the lesions vary according to the writers and the stage at which the lesion was seen and treated. Osteoporosis is not included as a tarsal bone lesion in this dissertation, as it does not automatically progress to disintegration. However, osteoporosis may soften and weaken bone so that it is more vulnerable to trauma in the course of normal activities. Decalcification may be detected in the neighbourhood of a fracture, as the primary response of the neurotrophic limb to trauma (Paterson and Job, 1964 - page 443). Its presence should therefore alert the patient and the clinician to the impending danger. The investigator has been able to predict correctly that tarsal bone disintegration would occur in a number of patients if necessary precautions were not taken (see Patient No. 1582, page 183).

It is well known (Paterson, 1961) that disuse itself may lead to osteoporosis, as may the use of high doses of corticosteroids for prolonged periods (Karat et. al., 1968). Hyperaemia (Johnson, 1963 - page 636) may also induce changes in the calcification patterns. Hence any of these factors, by inducing osteoporosis, may presumably predispose to stress fractures which in turn may progress to tarsal bone disintegration.

Sclerosis of bone has also been found in radiographic examination (Lennox, 1964). In the investigator's experience sclerosis is never present in the early lesion, but it does occur late and is then a sign of healing. A lesion that has healed with minimum deformity may only be recognised by the residual sclerosis. In patients with alternating periods of progression and regression, sclerosis has been seen side by side with disintegrating bone.

Price (1964C- page 519) and Harris and Brand (1966) emphasised the importance of collapsed bone, and pointed out that this collapse was responsible for many of the resultant deformities. Collapse is very likely to follow osteoporosis, but it does not typically occur in sclerotic bone. The healed and sclerosed bone seems rarely to be subject to further damage, and at surgery it has been observed by the investigator, that they maintain their hardness when other bones have softened from disuse.

It has been stated by Paterson and Job (1964 - page 441) that disintegration of the tarsal bones occurs in the presence of stocking anaesthesia and muscular palsy. This study reveals that 16% of affected feet showed no clinically detectable neurological deficit, and that in some patients the deficit on the unaffected foot was greater than that on the affected foot (see page 99). Only 38% of affected feet occurred in association with stocking anaesthesia of the affected lower limb. However, since in this study only clinical methods of testing were used, minor abnormalities may well have been missed.

Abnormal walking mechanisms were implicated by Harris and Brand (1966) as a causative factor. In the absence of marked bone deformity, the most common cause of an abnormal walking mechanism, in a leprosy patient, is a muscular palsy. In this series only 43% of the tarsal bone lesions occurred in feet that showed a definite muscular palsy. But of all the patients with some loss of foot dorsiflexion, 44% showed tarsal bone lesions, whereas only 9% of feet with normal dorsiflexion showed these lesions. Hence the incidence of tarsal bone lesions is higher in patients with loss of dorsiflexion but is not confined to these patients.

It has been stated (Paterson and Job, 1964 - page 441; Brand, 1963 and 1966; Price, 1964C - page 523; and others) that tarsal bone disintegration may follow fractures and microtraumata which because

of diminished pain perseption are neglected. Johnson (1967) concluded that fractures were of major importance in the initiation of neurotrophic joint changes, but that other factors which disrupted the stability of joints could also precipitate neurotrophic changes.

Microtraumata and fractures may not lead to disintegration if the site is one that is not subject to stress during activity, or if pain perception is normal and the patient spares his foot while the bone lesions are healing.

Many of these lesions are found in the major weight bearing areas of the foot, especially the posterior pillar and the medial longitudinal arch. Here the dynamics of walking cause continued stresses as described by Harris and Brand (1966). The pull of the remaining muscles may distract or impact the fragments, resulting in a slowly increasing deformity that may not be noticed by the patient till an ulcer occurs on a newly developed pressure point.

Lennox (1964, 1965) described the progressive collapse of the arch of the foot and credited it to softening of, or damage to, the plantar ligaments. Karat et. al. (1968) credited the same process directly to secondary infection, implying that chronic heel ulcers frequently progress to involve the calcaneo-navicular joints. The investigator has treated at least forty chronic heel ulcers and sinuses, some extending well into the calcaneum, but has rarely seen the calcaneo joints involved in these patients. There was no patient in this series in whom disintegration of the calcaneum was associated with ulceration of the heel.

Many patients present with bony disintegration of the tarsus, together with deformity and ulceration. This led to the assumption that bone necrosis and disintegration was the result of sepsis (Cochrane, 1947 - pages 52-53; and 1964 - page 275).

Price (1960A) gave a more correct appraisal of the situation by observing that sepsis often accompanies a late lesion, but that the lesion may be present in the absence of sepsis. In our series of twelve non-post-operative lesions occurring while the patients were under observation, only one was associated with sepsis at the time of, or immediately preceding the detection of the lesion. However, some workers (Karat et. al., 1968) maintain that sepsis is an important precipitating factor. It is recognised that patients with sepsis are more likely to present for treatment than patients with minimal deformities; hence some series may not present a true picture of the lesions that occur because of the method of selecting feet for radiographic study.

For many years it has been accepted treatment of ulceration of the feet to apply a walking plaster for six weeks or longer (Price, 1959; 1960J; Brand, 1963, 1966). Little is stated in the literature about the risk to the underlying bones when the patient is allowed to resume walking after removal of the plaster cast. Lennox (1965) threw some doubt on this practice, and suggested that it may be a factor in the development of neuropathic bone lesions. Saikawa (1965) records one patient in whom neuropathic bone changes developed after the use of a walking plaster as the treatment for a plantar ulcer.

Srinivasan and Mukherjee (1964) carefully described the application of a walking plaster They recommended that it remain on for eight weeks, during which time proper shoes are made for the patient to wear as soon as the plaster is removed. They suggested that a bandage be worn around the ulcer area for a few weeks after removal of the plaster, but made no other suggestions regarding post-plaster care or possible complications. Generally no limitation of activity is placed upon the patient and in some centres the patient may walk several miles on the day the plaster is removed, to reach home.

If the patient whose bones are osteoporotic is advised not to use the limb at all, further decalcification will occur, but carefully supervised activity and supportive therapy has been shown to encourage recalcification with minimal risk of the occurrence of disintegration.

There is no record of the number of patients who have developed bone lesions after wearing plasters, and since it is not possible, in many countries, routinely to radiograph every patient, it is impossible to discover this figure. Harris and Brand (1966) and Karat et. al. (1968) quote their examples from selected patients, and many of these show severe grades of damage. A large proportion of the patients referred to in these two articles were first diagnosed as having a tarsal bone lesion after surgery had been performed, or after treatment of chronic ulceration by immobilisation in a plaster cast.

Morris (1968) pointed out that a fracture might occur in an otherwise normal foot after a period of bed rest or immobilisation. He stated that it resembled both a fatigue fracture and a pathological fracture in a bone that had become osteoporotic through disuse.

Johnson (1963) referred to the effects of disuse and immobilisation on normal feet, and Harris and Brand (1966) mentioned the increased vulnerability of patients with leprosy to minor traumata.

These observations help to explain the findings in this series.

Of the patients in whom ulceration of the foot was treated by a walking plaster, 10% developed tarsal bone disintegration after removal of the plaster cast (see page 99).

Of patients in this series who had surgical correction of foot-

drop performed, 7.5% developed post-operative bone disintegration. In each case the activities of the patient had been restricted for an unusually long period, either before or after surgery, because of complications. Ross and Maclean (1964) mention one patient in whom metatarsal disintegration followed foot-drop correction. Saikawa (1965) records a neuropathic bone lesion that occurred after foot surgery. Paterson and Job (1964 - page 445) stated that sudden unusual trauma, after surgery to an osteoporotic foot, may cause a fracture. It is assumed that the increased duration of restricted activity predisposes to an osteoporosis sufficient to allow a stress fracture to occur when muscular activity is resumed.

Six patients (39% of those who developed lesions while under observation) were suffering from chronic lepra reaction, necessitating prolonged bed rest and inactivity.

The writings of Harris and Brand (1966) and Karat et.al. (1968) suggest that our findings may be present in other countries if more radiological examinations of suspicious lesions were made. Lennox (1965) also observed similar post-immobilisation neurotrophic lesions.

Johnson (1967) pointed out that hyperaemia is the body's natural response to injury, a piece of damaged bone, or the presence of an irritant. It has been shown by Johnson (1963 - page 636) that passive hyperaemia encourages osteoblastic activity, but that active hyperaemia may sometimes induce osteoclastic activity. He also stated that either type of hyperaemia may be activated by neurological disturbances, and that active hyperaemia may merge into passive hyperaemia and eventually a normal circulation is restored. He commented on the neural stimulus to hyperaemia and osteoclastic activity in Sudeck's atrophy, and stated that a lepromatous inflammation of a peripheral nerve was responsible for a similar situation in leprosy. As nerve inflammation is a frequent feature of lepra reaction this may well

explain the incidence of osteoporosis in the feet of many leprosy patients (Cochrane, 1957 - page 52 ; Paterson, 1961; Paterson and Job, 1964 - page 445; and Karat et. al., 1968).

Some of these workers have mentioned that increased osteoclastic activity has been observed during corticosteroid administration. Johnson (1963 - page 637) also mentions that adrenal cortical hormones increase osteoclastic activity. Since these drugs are sometimes given in the treatment of lepra reaction their action may increase the osteoporosis that occurs in these patients.

Disuse also results in osteoporosis when a normal bone is immobilised. It has been shown in young goats that reduction of the quantity of cancellous bone is complete within three weeks, though this may not be radiographically detectable.(Johnson, 1963 - page 607). Bone that has in this manner undergone porosis will be more prone to fracture under minimal trauma than would a normal bone. The same process occurs in man, though the time realtionships have not been determined.

This could well explain the incidence of fractures in the immediate post-operative period when there has been complete immobilisation of the affected leg for six weeks or more, and then sudden return to free walking. In these patients osteoporosis may play a leading role in the initiation of tarsal disintegration, as it apparently does in patients who have been immobilised in walking plasters.

Trueta (1963) showed that immobilisation of the calcaneum in rabbits by severing the tendo Achillis but allowing the rabbit free movement, resulted in marked osteoporosis accompanied by hyperaemia by the fifteenth day, but once the immobilisation was discontinued there was rapid recovery. Further investigations suggested that the

inhibition of muscle contraction caused passive hyperaemia and osteoporosis; this may explain why oedema of the foot may accompany acute paralysis in some patients with reactional borderline leprosy. Johnson (1967) stated that hyperaemia weakened bones and predisposed them to further injury so that even a minor fracture may result in a marked disability.

Paterson and Job (1964 - page 443) pointed out that nerve lesions alone do not produce bone changes, but that when nerves are damaged there is a change in the mechanism of repair. In long bones excessive callus is frequently laid down on the medullary side of the shaft, and in tarsal bones decalcification rather than callus formation tends to occur. Barnetson (1951) suggested that a failure of reflex vasomotor responses in vessels supplied by damaged nerves was a causative factor in bone atrophy in leprosy.

Johnson (1967) pointed out that fractures were of major importance in the initiation of neuropathic lesions. If this is correlated with the above observations it would appear that lepra reaction may lead to osteoporosis by causing hyperaemia and interrupting the normal neural control of the blood supply. Osteoporosis can also be caused by the use of corticosteroids, by bed rest and by immobilisation which may all be part of the treatment of lepra reaction. The body's response to a fracture in the presence of an interruption of the normal nerve supply is one of osteoclastic activity and hyperaemia (Johnson, 1963 - page 605). All these factors may weaken the bones, predisposing them to fractures under relatively minor trauma or to stress fractures. Because of the lack of pain associated with the neurological deficit, the fracture tends to be neglected and disintegration occurs from the movement taking place at the fracture site during usage.

Paterson and Job (1964 - page 441); Brand (1963); Price (1964C - page 523) and others, have accepted that microtraumata and fractures predispose to tarsal bone disintegration when diminution of pain perception results in the neglect of the traumatised limb. Tarsal bone disintegration could thus be considered as the result of repeated or neglected stress fractures in which healing is delayed.

Harris and Brand (1966) describe the grinding together of two fractured surfaces until the bone fragments. This process would be facilitated by the presence of osteoporosis acquired as described previously. It could be prevented by efficient immobilisation which prevents the fractured surfaces from grinding together.

The vicious cycle that may develop in the neurotrophic joint after trauma was described by Johnson in 1967. He also emphasised that repair will occur at any stage of the cycle if proper protection is provided to prevent further trauma. Repair results in dense sclerotic bone but if activity is resumed too early or too vigorously further bone breakdown may occur.

In tarsal bone lesions, nature's attempts at normal healing fail because of repeated further trauma. Without immobilisation and in the absence of pain perception, even limited activity may cause enough movement of the fractured fragments to disorganise any haematoma and callus that forms, so that complete healing cannot occur. This is supported by the biopsy findings which show osteoblastic and osteoclastic activity proceeding side by side.

It has been pointed out by Paterson and Job (1964 - page 443) that there may be minimal new bone formation and osteoporosis around a fracture of the tarsal bones in a neurotrophic limb. The present investigator has observed minimal callus response around a fracture in a neurotrophic limb. The reparative function is apparently unaf-

fected and may even be hyperactive and continue for a prolonged period. The result in the untreated patient is usually non-union, which eventually progresses to further absorption of the damaged bone, near the fracture, and may result in gross loss of bone. In many patients, all that is needed is complete immobilisation for an adequate period to allow complete healing to occur (see pages 89-91).

Johnson (1967) pointed out that neuropathic bone lesions will heal if treated correctly. The principles laid down by him apply to all leprosy patients with tarsal bone lesions. In the investigator's experience treatment by simple immobilisation in a plaster cast has proved effective in all patients who co-operated and persisted until healing was achieved.

Paterson and Job (1964 - page 441) suggested that healing may occur during immobilisation in a plaster cast, but gave no indication of the time required. Price (1964c - page 524 in the same edition of "Leprosy in Theory and Practice") stated that the application of a plaster cast would accelerate the destruction of the foot. He recommended the use of weight bearing callipers for twelve to eighteen months. This was attempted in several of our patients but it was very difficult to ensure that the muscles of the lower leg and foot were rendered non-functional, as recommended by Price, and partial use of the foot led to increasing deformity; it was therefore discontinued.

Bed rest, without mention of immobilisation, is recommended by Ross and Maclean (1964). Having observed some patients treated in this way and in whom healing was accompanied by increasing deformity (see Patient No. 1611, page 185), the investigator concluded that control of the position during healing was advisable and could reduce resultant disability.

The ability to heal may be unimpaired but attempts at healing are repeatedly foiled by renewed activity on the part of the patient. The adjacent osteoporotic bone is also damaged during these bouts of activity, resulting in an extension of the area of disintegration.

Price (1964C - page 524) recommended arthrodesis as the quickest method of achieving healing. Lennox (1965) also suggested that arthrodesis should not be delayed once a diagnosis was made. Both stated that a period of about three months was adequate to give sound healing. In the present investigator's experience, three months' healing is adequate for a calcaneo-talus wedge, but not for arthrodesis of the talo-navicular area in which weight bearing tends to impact the bones of the dorsal surface of the foot and to distract those of the plantar surface. Following a talo-navicular wedge or a bone graft in this area, some nine to twelve months should be allowed to elapse in order to achieve clinical healing. More than one patient has imperilled a good immediate post-operative result by walking after three months, which caused a collapse of the medial arch, though final bone healing was sound (see pages 93 and 93A).

Ikeda (1965) stated that it may require up to two and a half years to achieve complete healing, but as yet no patient treated by the investigator has required longer than twelve to fifteen months to obtain clinical healing. However, radiological changes may continue for many months more.

Harris and Brand (1966) suggested surgical fusion if conservative measures fail, and stated that the post-operative plaster immobilisation should be retained for half as long again as for the same operation in a patient who retains sensation in the foot. However, they stated that in the early stages treatment with special footwear was possible when the posterior pillar and medial arch were involved, but that once definite bone damage had occurred full immobilisation was necessary.

Johnson (1967) mentioned the use of internal fixation with a supplementary strong plaster cast because of the incidence of broken nails and plates that occur in anaesthetic limbs after surgery. He also mentioned that moulded shoes and arch supports may be needed. Lennox (1965) was of the opinion that an arthrodesis would be successful only of a special surgical shoe was worn afterwards. He acknowledged that in many instances the results of correcting a badly deformed foot was "an unglamorous appendage".

Because of the difficulty of achieving an ulcer-free acceptable foot, Price (1964C - page 521) suggested that amputation may be the solution for the ulcer prone neurotrophic rigid foot. Lennox (1965) mentioned the difficulties of limb fitting in the presence of an anaesthetic stump and stated that a salvage procedure should be attempted if possible. Johnson (1967) stated that amputation should rarely be needed for the bone problems in the neurotrophic limb, but may be necessary for the resultant ulceration and osteomyelitis.

In the patients under treatment at Hay Ling Chau, one of the aims of surgery is to avoid the necessity of providing surgical shoes (see page 155). These are not socially acceptable in this area, they are very expensive, and it is extremely difficult to get a suitable pair in a reasonable length of time. Between 1964 and 1970 about thirty patients at Hay Ling Chau had surgical procedures to correct or reduce deformities caused by tarsal bone lesions. Most of these patients have left hospital walking in normal footwear. In this area, crepe rubber soled shoes with soft fabric uppers are easily obtainable and socially acceptable. These are recommended with the addition of soft insoles where necessary. For most patients a walking plaster was used for about nine months when surgery to the arch of the foot had been performed, but a follow-up three to five years later, in the earlier patients, has shown that healing was stable and the position has been maintained.

Chapter 7
CONCLUSIONS

These studies have led the investigator to the following conclusions.

1. In leprosy, tarsal bone disintegration is often progressive. It may begin in any one of the tarsal bones and spread to one or more adjacent bones.

   Microtraumata and stress fractures play an important causative role and their presence should be treated seriously in order to prevent progress to major disability. Diminished pain perception means that the patient has no compelling warning to rest an injured foot and his continued activity results in an increase in the area of disintegration. The lesion may spread slowly or rapidly until all the tarsal bones are disorganised, but spontaneous healing may occur at any stage leaving some measure of residual deformity that may not constitute a real disability.

2. The early clinical signs of tarsal bone disintegration are heat and swelling, usually painless, of the affected foot or ankle. In any leprosy patient these symptoms should arouse suspicion af bone trauma. It is desirable that radiographic examination should be undertaken.

3. The radiological appearances vary from the slightly hazy to the grossly osteoporotic bone; from multiple disintegrating fragments to a dense block of sclerotic bone in the healed lesions; from a basically normal anatomical picture to one of gross deformity. There is, however, no difference in the fundamental lesion, and all active lesionw will respond to treatment.

The lesions may be detectable radiographically only after four to eight weeks. Meanwhile osteoporosis occurs around the lesion and this makes it radiographically detectable. This softening of the bone also makes it more vulnerable to further trauma.

4. Many lesions are only detected in retrospect, by comparison with a later radiograph, but the findings in this investigation suggest that the incidence of tarsal bone lesions in Chinese leprosy patients is at least 8% and may be as high as 24%.

5. Investigations suggest that these lesions are not due to avascular necrosis or specific lepromatous bone involvement. Biopsy findings support the theory that repeated microtraumata perpetuate the destruction of bone by hindering nature's attempts at healing.

6. The patients who are most likely to develop tarsal bone disintegration are: -

   (a) Those with atypical forms of leprosy, borderline(BB) and the transitional types (BL and BT).

   (b) Those with osteoporosis due to inactivity, immobilisation, surgery, the use of plaster casts, corticosteroids, or old age.

   (c) Those with a neurological deficit. This may result in: -

      i. An interruption of the superficial nerves, which may interfere with the normal control of circulation and thus predispose to hyperaemia.

ii. Anaesthesia, which may be either: -

1) Deep, allowing the continued use of a traumatised limb without pain; or

2) Superficial, allowing trauma of the skin and the entry of infection.

iii. Muscular weakness or paralysis, allowing the development of abnormal stresses during activity, is of special importance if the bones are weakened. Acute paralysis may reflexly cause oedema and hyperaemia which may further weaken the bones.

(d) Those who traumatise their feet in daily activities. Minor traumata and stress fractures may occur without obvious accident and may be neglected because of the diminution of pain perception.

(e) Those with hyperaemia of the feet, such as may accompany oedema and may occur during reaction, intercurrent diseases and infections.

This hyperaemia may soften bones and also loosen ligaments, allowing them to stretch so that abnormal stresses fall on the underlying bones.

(f) Those with septic foci of the feet. The resultant osteomyelitis may weaken the adjacent bones and the resultant hyperaemia may allow osteoporosis, predisposing the patient to bone damage during trauma.

7. The patients at risk are therefore: -

(a) Patients with multiple nerve lesions affecting the feet, especially if intercurrent disease or complications of leprosy have resulted in limitation of mobility for a period that is long enough to produce osteoporosis.

(b) Patients whose activity is reduced or whose limbs are immobilised for any reason; either foot may be affected at or soon after the time of immobilisation.

(c) Patients who wear a walking plaster for periods of time long enough for osteoporosis to occur, rendering the foot more vulnerable to trauma when walking is resumed. In the previously normal foot six weeks or more immobilisation appears to predispose to stress fractures.

(d) Patients with lepra reaction who:-

i. Tend to pursue a sedentary life or complete bed rest.

ii. Have swollen, hyperaemic feet, with generalised or localised oedema.

iii. Are treated with corticosteroids.

iv. Develop acute paralysis of the leg muscles.

8. The basic principle of treatment is immobilisation in the optimum functional position until healing is complete. Consideration must be given to the final desired position from the initiation of treatment, even if sepsis needs to

be controlled before full immobilisation can be instituted. Gross deformity may require surgical correction but it is possible in the majority of co-operative patients to achieve sound healing in a position that is compatible with function and the wearing of a socially acceptable shoe.

9. Tarsal bone disintegration, when neglected, can be a very disabling condition but with early diagnosis and treatment, complete healing can occur leaving no significant residual deformity or disability.

Chapter 8
APPLICATION

The findings recorded in this thesis reveal that it should be possible to reduce the prevalence of deformity and disability resultant from tarsal bone lesions in leprosy patients. To achieve this the following action is advocated:

1. Every leprosy worker should be alerted to the existence of this condition, its symptoms and signs, its dangers and treatment. Much of the field work in endemic areas is done by paramedical workers and technicians who have only limited training and minimal supervision by trained medical officers. These workers are relied upon for early diagnosis and treatment, and should be trained to detect early bone lesions.

2. Public education should be intensified to encourage persons affected with leprosy to come early for treatment. Early diagnosis and correct treatment of leprosy will reduce the incidence and severity of lepra reaction and nerve involvement. Thus the complications of leprosy which predispose to osteoporosis and hyperaemia and to tarsal bone disintegration will be reduced.

3. Patients under treatment should be instructed in rules of daily living and general foot care, and especially to look for heat and swelling and to report abnormalities as soon as possible. In some endemic areas there may be several weeks or even months between clinics, so patients should be taught simple first aid, including the application of a supportive bandage for use if there is any doubt regarding an affected foot.

4. Physiotherapy should be taught to all patients with muscular

paralysis or paresis, and a toe-raising spring provided when necessary. Instruction should also be given regarding suitable footwear. Patients with anaesthetic feet should always wear shoes.

5. The leprosy worker should regularly examine the feet of all patients under his care. This will help in early diagnosis and will accentuate to the patient the need of foot care.

6. In the treatment of sepsis it should be remembered that there may be an underlying or complicating bone lesion. The foot should be protected during healing to prevent possible deformity. Sepsis of long duration may cause osteoporosis and hence predispose to tarsal bone disintegration.

7. Where radiological facilities are available every patient should have dorsiplantar and lateral radiographs taken when the diagnosis of leprosy is first confirmed. If this is not practical, because of expense or availability of film, then radiographs should be taken of every suspected foot. If the initial radiograph, in such a situation, shows no lesion it should be repeated in four to six weeks time and in the meanwhile the patient should use a supportive bandage.

8. Where no radiological facilities are available the leprosy worker must rely on clinical features. It is most important that he be well trained in this aspect. Any obviously fractured foot should have a plaster cast applied at once. If reasonable doubt exists regarding the nature of the lesion a supportive bandage can be applied for a few weeks whilst the patient reduces his activity. If heat and swelling do not subside then a walking plaster cast should be applied, unless some other obvious cause has manifested itself. If there is any indication that the heat

and swelling are the result of tarsal bone disintegration a walking plaster cast should be applied for a minimum of three months and followed by a period of trial walking. If there is recurrence of the heat and swelling the plaster should be replaced as this indicates that a lesion was present and it is not yet completely healed.

9. All lesions which are radiologically confirmed should be treated at once by immobilisation until there is clinical and radiological evidence of healing, and no heat and swelling occur during the period of trial walking. This immobilisation is usually achieved by the use of a plaster of Paris walking cast fitted with a rocker or a walking iron. These plasters can usually be applied for two to three months and on removal a radiographic check is made. The patient should not be allowed to commence trial walking when the plaster is removed until it is decided that healing is radiographically complete.

Rarely is a true lesion healed within three months, so at the first plaster change one can reapply the plaster as soon as the radiographs are satisfactory. It is advisable to replace the plaster on the day of removal in every case where healing is incomplete. Early walking will delay final healing.

10. The plaster cast should be applied by the Medical Officer concerned or by some well trained technician who understands the fact that deformity can be caused by a poorly applied cast. If the foot is hypermobile it is often possible to reshape it so that healing will occur in a more functional position. In this way it will be possible to prevent or at least minimise the reconstructive surgery needed at a later date. In lesions of bones directly involved in weight bearing, it is desirable that the weight be

borne from the tibia and not through the foot.

11. Patients should be kept mobile and weight bearing if possible. Healing will proceed with immobilisation even during complete bed rest, but if the patient is completely immobilised there is danger of osteoporosis developing in the other foot which may predispose to a further lesion when walking is resumed.

12. The presence of osteoporosis from any cause, in a person with a neurotrophic foot, should alert the medical staff to extra care when ambulation is resumed after long periods of rest, inactivity or immobilisation. If these persons rapidly resume their normal activity they are prone to develop stress fractures which may well be neglected because of the diminution of pain perception, and disintegration may commence.

13. Marked deformity should be surgically corrected if possible. If this is not practical, the patient should be instructed in the use of suitable footwear and in any special precautions needed to prevent ulceration and the development of further deformity and disability.

14. Personnel, at all levels, should be recruited to take treatment to millions, of as yet untreated, leprosy patients. These sufferers have no chance of preventing deformity at the present time, though the means of controlling the disease and preventing much of the accompanying deformity are known.

15. Research should be extended to widen the understanding of this disease and its complications. Further studies in the histo-pathology of affected bones may increase our understanding of tarsal bone disintegration and so reduce the numbers disabled by this disorder. New drugs for the treatment of leprosy are needed

to control the disease and to reduce the complications that predispose to deformity and disability.

Gross deformity of the foot following tarsal bone disintegration can be prevented by early adequate treatment. The disabilities resulting from gross deformities can be reduced by reconstructive surgery. These facts are not yet generally known, and even though the lesions are sometimes recognised, suitable treatment is not always given.

Our present knowledge is such that once the lesion is diagnosed, further deformity should not occur, and the patient's prognosis should be satisfactory, providing he is adequately treated, along the lines laid down in this thesis.

APPENDIXES

Appendix A

HONG KONG LEPROSARIUM - HAY LING CHAU

1. BACKGROUND

The Colony of Hong Kong is on the south-east coast of China and comprises a peninsula of the mainland and some 250 adjacent islands. The capital, Victoria, is commonly spoken of as Hong Kong, and it is on the Island of Hong Kong (second largest of the group) across the harbour from Kowloon, which is on the mainland. Hong Kong means "Fragrant Harbour". A map of the Colony is found on page 144.

At the end of the Japanese occupation (1945) the population of the Colony was about half a million, but this was increased during the next few years by immigration from neighbouring parts of South China and then more rapidly by the influx of refugees which followed political upheaval in China. This population change must be remembered in any statistical study of disease patterns in the Colony and particularly in regard to leprosy. A continuing - though lessening - flow of refugees still comes into Hong Kong, but of recent years the population increase, soon up to four millions, has been largely due to natural increase. It is estimated that half the population is aged below twenty.

Prior to the Communist take-over in China (1949) there was no major problem of leprosy in Hong Kong. Leprosy patients found were sent back to their ancestral villages or to leprosy colonies run by missionaries on the China side of the border. The influx of refugees changed this situation. Among them were many sufferers, including long-term cases with multiple deformities. It soon became obvious that these could not be deported back to China and the Hong Kong Government requested the assistance of The Leprosy Mission, then called The Mission to Lepers, to aid in the setting up of a treatment programme.

A group of leprosy patients had congregated together on Hong Kong Island and regular effective drug treatment was given to them while routine out-patient clinics were established elsewhere in the Colony. The next step was to seek a site for a residential institution to provide segregation for contagious patients, and intensive care and hospital facilities for those in need of them. After much searching and opposition, the Government in 1951 offered the island, now known as Hay Ling Chau. This island was situated nine miles west of the main harbour and was undeveloped.

At the time Hay Ling Chau began it was still generally accepted that patients with bacillary remnants in their skin were able to transmit the disease and these patients were required to remain at Hay Ling Chau until their skin smear (see page 157) became negative and remained so for six months.

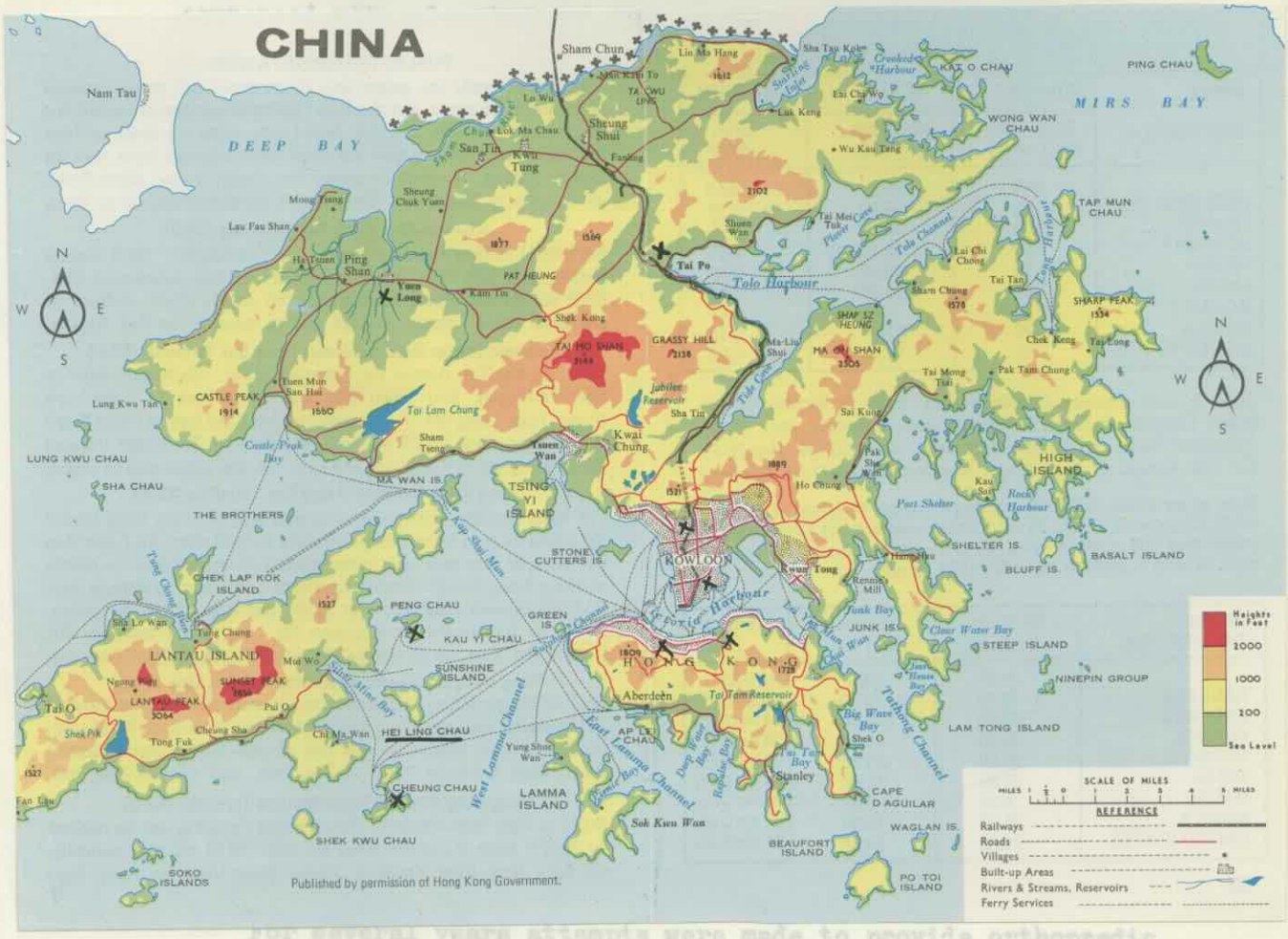
At that time little was understood about the development in leprosy of deformity and disability and patients were not taught how to protect themselves from injury. Because of this, a number of patients developed increasing deformity and disability while under treatment. It was not until 1958 that an active rehabilitation programme, including physiotherapy and reconstructive surgery, was commenced, and prior to that date, as far as possible, patients with negative skin smears were discharged to Hong Kong, in spite of deformities and disabilities, to make room for others who were considered contagious and in need of segregation.

Many of the patients who were admitted for treatment during the 1950s had had leprosy for many years without any effective treatment. At the commencement of the leprosarium at Hay Ling Chau the facilities were very simple as the object of the institution was mainly segregation of contagious cases. The main criterion for selection for admission was infectivity, though a few grossly disabled patients were admitted for care, as it was obvious that they could not look

after themselves and they had no known relatives who could undertake the responsibility.

This picture has changed over the past nineteen years, as the disease has come under control in the Colony and fewer active cases are entering the Colony as refugees. In Hong Kong the incidence of newly diagnosed leprosy patients is dropping each year and many of these new patients are diagnosed before any deformity has developed. Many of them are non-contagious at diagnosis and, under new legislation in accordance with modern scientific knowledge, are not segregated. This means that an increasing proportion of new patients are being treated in the out-patient clinics and that Hay Ling Chau is no longer filled to capacity as it was until 1962.

Since 1962 selected non-contagious but disabled patients have been admitted to Hay Ling Chau for reconstructive surgery and rehabilitation. This number was increased after 1964 when the number of patients at the leprosarium decreased sufficiently to make beds available for this purpose.



Map of Hong Kong

Government Out-patient Clinics indicated by x

3. DEVELOPMENT OF FACILITIES

In 1951 the first patients to come to Hay Ling Chau were selected because of their ability to undertake manual work, and they commenced site clearing and the erection of buildings on a virtually barren island.

For the first few years the emphasis was on providing residential accommodation for those needing segregation. Medical treatment was provided from a simple dispensary by a doctor and his assistants until the hospital block was constructed.

The Maxwell Memorial Medical Centre was opened by the Governor of Hong Kong in 1954, providing wards, treatment rooms, laboratory, operating theatre and other facilities. In 1956 the radiographic facilities were installed. By 1958 the wards of the Medical Centre had been increased to provide bed care for about sixty patients. Simple physiotherapy was started in 1958 at about the same time as the reconstructive surgery programme was inaugurated. A full physiotherapy programme was not undertaken until a full time physiotherapist was available in 1961 and soon after that occupational therapy was also organised to assist in the re-education of the severely deformed.

For several years attempts were made to provide orthopaedic footwear for patients with deformed feet; the standards achieved were not generally high and patients did not readily accept "different shoes", so since 1964 this has lapsed in favour of surgical realignment of the feet so that the patient can wear normal shoes.

Hay Ling Chau today provides residential, recreational and educational facilities as well as medical facilities for patients recommended for admission. Accommodation is available for 540 patients, but in 1970 the number did not exceed 275 at any time.

Duration of stay is gradually shortening over the years. Once the average length of stay was five years, but with more modern methods of treatment and a more scientific approach to the disease this is shortening considerably. However, there are still many patients resident for long periods who have provided the basis of this study.

4. STATISTICAL SUMMARY OF PATIENTS WHO HAVE BEEN TREATED AT HAY LING CHAU IN THE LAST TEN YEARS

	Patients at Hay Ling Chau on 1st January	New admissions	Readmissions	Discharged certified non-contagious	Other discharges and deaths *
1960	540	119	5	69	52
1961	543	114	11	89	58
1962	521	122	9	71	56
1963	525	122	11	94	53
1964	511	98	20	53	67
1965	509	90	22	69	75
1966	477	71	21	74	66
1967	429	57	13	100	55
1968	344	58	10	77	54
1969	281	61	28	55	40
1970	275	50	20	52	52
1971	241				

* Discharges include those who are near negative but are not entitled to a certificate, and absconders. There have only been about five deaths annually.

Appendix B

TREATMENT OF LEPROSY

1. DRUGS

In the 1940s it was realised that Dapsone (Diamino-Diphenyl Sulphone) and some of its derivatives were effective drugs in the treatment of leprosy. Dapsone became generally available about 1948 at a cheap price and is still the basic drug of choice in doses from 200 to 400 mg weekly for maintenance. Smaller doses may be used in acutely active and reactive forms of the disease. It may be given orally or parenterally, and is usually well tolerated. Toxic effects may occur at higher dose levels.

In the 1950s Thiambutasone was also found to be effective. Initially given orally, the absorption of it was unreliable and in Chinese patients it tended to produce gastric symptoms. The development of an injectable form in the 1960s has increased its usefulness. However, drug resistance may occur within two years so it is not advisable for long term treatment.

Lamprene (Clofazamine) was first tried in leprosy in 1960 and has shown itself to be an extremely useful drug. The only undesirable effect is a darkening of the skin. This colour, however, slowly fades on cessation of treatment. Lamprene minimises lepra reaction, neuritis and other complications which lead to many of the deformities and disabilities of leprosy patients.

Long-acting sulphonamides, Isonicotinic Acid Hydrazide (INAH) and Streptomycin are all effective in the treatment of leprosy, but less so than the three drugs previously listed. There are also other drugs that have been tried and may have usefulness in situations where lepra reaction is a real problem and Lamprene is not available.

Drug resistance of M. leprae to Dapsone and Thiambutasone has been proved, but so far not to Lamprene. However, for mass treatment Dapsone remains the drug of choice, and for many patients it is the only drug they require.

2. PHYSIOTHERAPY

Formally, deformity was accepted as an inevitable part of leprosy itself. Nowadays, it is known that much deformity is the result of neglect and ignorance on the part of the patient.

Lepra reaction (page 11) may cause oedema, induration and ulceration of the hands and feet. There may be an associated malaise and a tendency to neglect even the necessities of daily living. This may result in stiffening of the affected limbs, and contractures of skin and tendons may develop as a result. Physiotherapy to maintain a full range of joint movement every day does much to maintain suppleness and joint mobility.

Acute neuritis (page 13) may cause paralysis or paresis. Unless the limbs are kept mobile and the patient is taught to use any remaining muscle fibres, contractures and secondary disuse atrophy may follow.

Physiotherapy to teach isolated movements of a muscle before tendon-transfer surgery and to re-educate the muscle after surgery, is an essential part of the rehabilitation programme, to ensure optimum usefulness of the limb.

Even after reconstructive surgery patients need to continue exercises for many years, to maintain function.

Patients at Hay Ling Chau are taught, as far as possible, to do their own physiotherapy, as most need to continue it for many years, to avoid later development of deformity.

3. SURGERY

In the 1940s it was realised that leprosy produced typical patterns of nerve involvement and resultant deformity. Also, that a relatively small number of reconstructive surgical procedures would restore function to a large proportion of the patients with paralytic deformities.

With regard to the foot, the commonest reconstructive surgical procedures are: -

i. Provision of an active dorsiflexor of the foot. The usual procedure is the transfer of the tibialis posterior muscle to provide an active dorsiflexor (Gunn and Molesworth, 1957; Selvapandian and Brand, 1959). Before performing the operation, consideration must be given to the whole pattern of paralysis in the foot, or further deformity at the talo-navicular joint may result from the removal of the tendinous support of the joint provided by the tibialis posterior tendon (Warren, 1968). Care must also be taken to ensure adequate eversion of the foot after surgery, especially if the ligaments are lax or there has been a definite pre-operative inversion. Elongation of tendo Achillis is frequently advisable, at the same operation, to ensure that adequate dorsiflexion is achieved.

ii. Claw toe correction to increase the weight bearing surface of the sole and help protect the metatarsal heads. The Girdlestone operation, using the flexor digitorum longus tendon to provide a lumbrical for its own toe, is ideal. The removal of the flexor digitorum longus from the terminal phalanx reduces the marked degree of flexion of the inter-phalangeal joints that may occur due to over-action of this muscle. Its removal therefore assists in straightening the toes so that they become more functional and weight bearing again.

iii. Osteotomy to correct basic bone deformity which cannot be corrected passively. The commonest problem is the varus calcaneum. Over-correction is desirable, as the deformity tends to recur even after muscle transfers. Correction by wedge osteotomy in several different planes may be needed to realign a badly deformed foot. Inversion may be accentuated by deformity of either the talus or the calcaneum.

All reconstructive surgery must be followed by a period of physiotherapy and instruction so that the patient learns to care for himself and to use the limb correctly. Reconstructive surgery plays an important role in the rehabilitation of leprosy patients, enabling them to return to the community with the obvious stigmata of leprosy removed and their disability reduced so that many of them are able to support themselves and their families again.

A list is given of the surgical procedures performed by the investigator, on the feet, for the benefit of leprosy patients during the five year period 1965-1969. This shows the variety of the procedures utilised and indicates the frequency of these complications. The number of patients under treatment at Hay Ling Chau during these years is listed on page 147.

LIST OF OPERATIONS PERFORMED ON THE FEET OF LEPROSY PATIENTS BY THE INVESTIGATOR, 1965-1969

	At Hay Ling Chau	At other centres
Tendon transfer for foot-drop correction	123	89
Arthrodesis of ankle or foot	39	26
Tenodesis for dropped foot	7	2
Tendon transfer for claw toes	46	15
Other tendon surgery	6	-
Arthrectomy for claw toes	18	5
Arthrodesis of toes	7	7
Below-knee amputation	1	-
Symes amputation	3	-
Trimming of abnormal or rough bone to prevent further trauma or ulceration (usually on the sole)	56	10
Sequestrectomy and/or curettage of sinus track	42	6
Other minor surgery to the foot such as skin grafts and the removal of scars, deformed toes and tumours	32	9
	380	169

4. THE TREATMENT OF LEPRA REACTION

As stated on page 11, "Reaction" is used to cover a number of different medical entities. The treatments that have been tried are multitudinous.

Basically, most patients with "lepra reaction" manifest an acute exacerbation that is usually associated with some form of immunological response.

The essentials in treating these patients are: -

1. Rest of body and mind. In many patients emotional factors appear to trigger a lepra reaction. Sedatives and tranquilisers shorten the severity of the reaction.

2. Analgesics where necessary, remembering this is a chronic disease and analgesics may be needed for long periods.

3. Attention to basic medical needs; dehydration is very common in Chinese patients who are unwell from any cause; secondary infections, anaemia, and metabolic disturbances, will aggravate lepra reaction.

4. The use of specific drugs whose usefulness has been found empirically and is not always understood. Sodium antimony tartate injections, antimalarial compounds, anti-inflammatory compounds, antihistamines and diuretics, all seem to help in selected patients.

The treatment of lepra reaction is time consuming and requires close observation of the patient. It may necessitate months or years of hospitalisation and careful medication.

Many workers rely on the administration of corticosteroids to control lepra reaction.

In patients suffering from BB-BT-TT leprosy who develop acute neuritis, the use of corticosteroids may save nerve function. A three to four week course is usually adequate. As these patients have a high natural immunity, they respond rapidly to adequate drug treatment and rarely require a longer course. Up to 70% recovery of nerve function can be expected in this group of patients if seen within two months of an acute paralysis.

In patients suffering from LL and BL leprosy the problem is different, because they have a lower level of immunity and lepra reaction may become a very chronic problem. Corticosteroids may produce an initial improvement but the patient rapidly becomes dependent on increasing doses to control the reaction. Moon facies and the complications of steroid dependency are not uncommon in lepromatous patients in whom lepra reaction is controlled by the use of corticosteroids.

In recent years the antileprotic Clofazamine (see page 148) has been shown to have an anti-reaction effect. Since it became available the incidence of severe reaction has steadily declined until chronic reaction in LL or BL leprosy is now a rarity amongst the patients cared for by the investigator.

5. FOOTWEAR IN LEPROSY

All patients showing any neurological deficit, because of the possibility of areas of anaesthesia on the sole of the foot, ought to wear protective footwear. This footwear should provide adequate resilience to buffer the intense pressures which occur during the strike and take off phases of walking (Gleave, 1962; Bauman, Girling and Brand, 1963; Brand, 1966). At the same time the sole must provide adequate protection to prevent the penetration of sharp objects which might damage the skin.

Many patients defer treatment until deformity has already occurred to a degree which prevents the use of normal shoes. Orthopaedic footwear has been recommended (Gleave, 1962; Bauman, Girling and Brand, 1963) and will, when well fitted and constantly worn, protect many of these feet from further damage. However, in the developing countries, it is frequently impossible to obtain the services of an efficient prosthetist. Even if the patient can afford the price of the shoes he will often refuse to wear them because they label him as different from the rest of his community. There is also the fact that these special shoes need to be constantly repaired and replaced. Orthopaedic footwear does not, therefore, provide a practical method of controlling the ulceration of thousands of patients in the developing countries, though it can be effective for those who can be provided for and who will co-operate.

Each geographical area must assess its own social needs, and devise footwear which is suitable and socially acceptable for those who already have deformity. For the newer patients, it should be possible to prevent marked deformity and the need for special footwear. To do this the patients must be taught the essentials of foot care at an early stage of the disease, and to purchase, and always wear, suitable shoes that will protect their feet from damage. In many of the countries of South East Asia these are readily and cheaply available but the patient needs instruction as to which ones to purchase.

6. RULES FOR DAILY LIVING

Patients need to be taught the dangers of anaesthesia and paralysis.

Because of the loss of pain perception, leprosy patients are constantly in danger of damaging their hands and feet in the course of daily activities. They should learn: -

(i) To examine their hands and feet every day, in order to detect any ulceration, haematoma, blister or other evidence of trauma which requires treatment. This is best done at the end of the day so that treatment can be given before retiring.

(ii) To soak their feet and hands in water daily to hydrate the dry skin and so help prevent splits and cracks.

(iii) To oil their hands and feet at least daily to compensate for the sebum deficiency, to prevent dehydration and to keep the skin supple.

(iv) To treat any ulcer or break in the skin and to apply a suitable dressing to keep it clean. This may need to include a splint if the ulcer is over a moving surface.

(v) Always to wear suitable shoes which will give adequate protection, and if there is muscular weakness of dorsiflexion, to use a toe-raising spring to avoid further deformities.

(vi) To spare their feet whenever possible. "Don't stand if you can sit, don't walk if you can ride".

Appendix C

LABORATORY PROCEDURES

A. SKIN SMEAR EXAMINATION

M. leprae may be found in the skin of some leprosy patients and their presence may assist in the diagnosis, in the classification, and in the assessment of the patient's progress.

Their presence is detected in skin smears taken according to Cochrane, (1964 - page 613) and read according to the method of Ridley, (1964 - page 620). At Hay Ling Chau these smears are taken from six sites and after staining are examined microscopically. Each smear is allotted a number that indicates the number of acid fast bacilli that can be seen in that smear. The grading is a logarithmic one.

6 = more than 1,000 bacilli per oil immersion field
5 = 100 - 999 bacilli per " " "
4 = 10 - 99 bacilli per " " "
3 = 1 - 9 bacilli per " " "
2 = 1 - 9 bacilli per 10 oil immersion fields
1 = 1 - 9 bacilli per 100 " " "
0 = No bacilli can be seen in the whole slide

The average of the six sites is then taken and the result is recognised as the Bacillary Index (B.I.).

On the same smears an estimation is made of the proportion of bacilli that appear as normal or solid staining rods. The average from the six fields is termed the Morphological Index (M.I.) and is expressed as the percentage of solid rods seen amongst the disintegrating bacilli.

In these skin smears the acid fast bacilli may appear singly or

in small groups. In highly active lepromatous leprosy they lie in large agglomerations or globi. These globi are reported as containing thirty or forty bacilli in African patients, but in East Asia there may be 300 to 400 bacilli in each globus. These globi each represent a group of bacilli previously contained in one reticulo-endothelial cell.

In Chinese patients at the commencement of treatment, the B.I. may be 5.0 or 6.0 in a patient with florid lepromatous leprosy with a history of about ten years' duration, but a B.I. of 4.0 - 5.0 is more common in patients with either borderline or lepromatous leprosy at the beginning of therapy.

Patients with a high B.I. may be said to have multi-bacillary leprosy and those with a low B.I. may be said to have pauci-bacillary leprosy. Patients in whom the B.I. is 0.0 are spoken of as being B.I. negative.

Patients with tuberculoid leprosy usually have a B.I. of under 1.0 and frequently of 0.0. An active lesion in a reactive phase may, however, yield a reading of 3.0 or 4.0 for a short period.

In Chinese patients in Hong Kong the M.I. is rarely above 25% "solid rods" at the beginning of treatment, though occasionally readings as high as 60% have been recorded in relapsed lepromatous leprosy. Patients with borderline and tuberculoid leprosy rarely have a high M.I.

B. CULTURE OF MYCOBACTERIUM LEPRAE

Successful culture of *M. leprae* on laboratory medium has not yet been achieved, though in the last decade it has become possible to culture the organism on the foot pads of mice (Shepard, 1962). More recently, innoculation of the organisms into thymectomised irradiated mice has resulted in the development of leprous lesions and the multiplication of the organism (Rees, 1970). These methods of investigation are only available in specialised centres and the average worker has no means of definitely identifying *M. leprae* isolated from a patient under his care.

Appendix D

SELECTED CASE HISTORIES

Arranged according to admission number

PATIENT NO. 152

Female, aged twenty-four years on admission in DEC 50, with BT leprosy of twelve years duration. Her B.I. was negative. She had not previously received antileprotic drugs. On 10 JAN 51 it was noted that her left leg was normal but that the left foot was swollen. The right foot had a large open ulcer on the heel and on the lateral side of the foot, partly due to her walking on an anaesthetic foot with fixed talipes-equinovarus.

The first radiographic examination was 1 AUG 57 when the right foot was grossly deformed and the left foot showed compression of the distal tarsal bones (Fig. 24) but this fact was not noted in the report at that time. No lateral view was taken then and the first satisfactory lateral radiograph was taken in DEC 63 and this showed an obvious deformity resulting from compression of the cuneiforms of the left foot.

COMMENT

It is presumed, because of the swelling, that the left foot showed cuneiform disintegration at the time of her admission, DEC 50. No specific treatment was given then but the patient's walking was limited because of ulceration and sepsis of the other foot. Apparently healing occurred with deformity of a degree that was compatible with function and this foot has not caused the patient much trouble (Pattern 2, page 83). She had the right foot amputated in 1963.

Fig. 24 Spontaneous healing of disintegration of the cuneiforms.

PATIENT NO. 264

Male, aged forty-seven on admission 6 JUL 66 for treatment of a swollen ankle. He had been treated for lepromatous leprosy since 1951 and his B.I. was negative.

A history was given of a fall while at work in SEP 65 and he was told he had a fractured ankle. He was hospitalised for fifteen days and put into a walking plaster for four months. After this the swelling persisted and there was marked limitation of ankle movement. He was told that his ankle would not heal and no further treatment was advised. After six months of discomfort when walking, he requested readmission to Hay Ling Chau.

On 8 JUL 66 a radiographic study (Fig. 25) showed a fracture of the lower end of the fibula with some displacement and complete collapse of the body of the talus and irregularity and haziness of the other bones around the ankle joint. After three days' elevation to reduce the swelling, a walking plaster was applied for three months and radiographs on 5 OCT 66 showed some improvement and a walking plaster was reapplied. By 16 JAN 67 the radiographs showed further improvement, though osteoporosis was present. He was allowed to resume walking which he did without further swelling.

16 MAR 67 a radiograph showed better calcification and the patient had achieved a relatively normal range of ankle movement. He was able to resume his previous occupation and has had no further complaints relating to his foot. New radiographic studies in NOV 69 showed a stable foot with reasonable ankle function, although some residual deformity was present.

COMMENT

In this patient there is a definite history of trauma and treatment for fractured fibula but after four months the immobilisation

was discontinued. It is assumed that the talus may have been involved at that time, or was traumatised on resumption of walking while it was still osteoporotic after the initial four months in a walking plaster. Unrestricted walking for six months allowed further development of the lesion which healed during six months immobilisation of the foot and leg in a walking plaster.

Fig. 25a

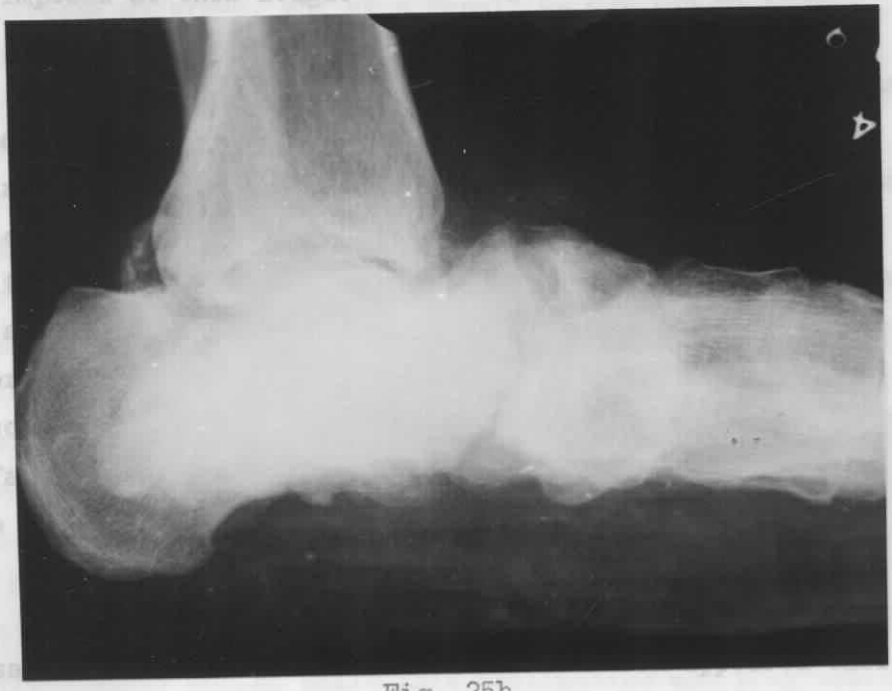


Fig. 25b

Fig. 25 a & b Collapse of the talus and periosteal proliferation of all the surrounding bones, occurring in the 4-10 months period following a fractured ankle.

PATIENT NO. 402

Male, aged thirty-eight on admission in JAN 53, with untreated, severe lepromatous leprosy and a B.I. of 6.0.

He had a history of at least twelve years leprosy on admission and already had stocking anaesthesia of both legs below the knee and of the right thigh. He had bilateral foot-drop.

His case notes are rather deficient until AUG 54 when it was reported that his right foot was swollen but that he had no ulcers on his foot. However, in JAN 55 his right third toe became septic, and from that date onward he seems to have had a series of ulcers on the right foot, which was intermittently reported as being swollen. In AUG 55 his right ankle was queried as being a "Charcot joint" but no radiograph was taken as no machine was available at that time. His left foot was noted as being normal. No limitation of activity was imposed at this stage.

In NOV 55 he had painless inflammation of the right ankle and oedema of the leg and was advised to rest in bed. This was obviously an infection as a sequestrum was removed two weeks later and the patient returned to work in JAN 56 walking, although he was advised not to do any heavy work. In MAY 56 a radiograph machine was installed, and on the 22 MAY 56 radiographs were taken (Fig. 26a) and reported on as showing gross involvement of the metatarsals and the tarso-metatarsal joint area. Infection and destruction of the joint surfaces with some erosion of the cuneiforms and the cuboid bone were also present.

In retrospect there was obviously marked destruction of all the tarsal bones in front of the talus head which appeared basically normal. The navicular and the cuboid were grossly involved. There was no lateral view available on this date. A walking plaster was applied 11 JUN 56 with a walking iron and renewed as necessary, until

19 FEB 57 when a radiograph was reported as "showing good reconstruction of the tarsal bones" (Fig. 26b). The plaster was reapplied until 20 AUG 57 when it was removed and the patient was told to walk using crutches for partial weight bearing.

In SEP 57 a radiograph was reported on as being "about the same" but in retrospect there is definitely new deformity of the body of the talus but a lateral view only was taken (Fig. 26c). The patient was allowed to continue walking until 27 SEP 57 when a walking plaster was reapplied as his leg was swollen. This plaster only remained on for four weeks and then it is presumed that the patient walked again, as he was not in hospital and the investigator knows from later experience he could not manage crutches because of the deformity of his hands.

It was noted on 28 NOV 57 that the right ankle joint was becoming completely deformed, and as a result he was put in hospital and told to limit walking and to use crutches. The next radiograph, taken 21 JAN 58, showed collapse of most of the body of the talus but the head and neck were still intact. No specific treatment was given at that time except the patient remained in hospital, theoretically using crutches. On 30 APR 58 the left foot was operated on for correction of foot-drop by tibialis posterior transfer. This presumably led to more stress on the right foot, which was unprotected at the time; a radiograph taken on 29 APR 58 was reported on as showing no change but in retrospect it is obvious that by then the calcaneum was also involved. A radiograph taken eight weeks later on 1 JUL 58 showed that the lower end of the tibia was involved and the foot was in a typical dropped position (Fig. 26d). It is noted that the patient had not had any immobilisation of this foot for the previous eight months while this destruction was progressing, and on 11 JUL 58 a below-knee amputation was performed of the right foot.

COMMENT

This patient gives a fairly good picture of the slow, relentless progress of tarsal bone disintegration that will occur in a foot that is not immobilised and which the patient continues to use, even if only partially. In this case the sepsis of 1955 obviously had something to do with the initial destruction, but the improvement during his fourteen months in a plaster cast (11 JUN 56 to 20 AUG 57) was very good and it is assumed that repair would have proceeded and the final disorganisation of the ankle might not have occurred if plaster immobilisation had been continued.

In retrospect, it may be observed that each time a walking plaster was applied there was improvement in the bone condition to such a degree that led to the belief that healing was satisfactory and immobilisation could be abandoned. However, in this patient and in a number of others, it has been found that under similar circumstances further breakdown occurred and immobilisation was necessary for a prolonged period of time.

In this patient the lesion was allowed to progress to complete loss of the talus. Today even that loss could be compensated for by surgery, but the patient was subjected to amputation, and that led to chronic problems due to ulceration of the anaesthetic stump (see page 129).

Fig. 26a MAY 56. Radiograph showing disintegration of tarsals and metatarsals associated with osteomyelitis.

Fig. 26b FEB 57. Some healing of all lesions occurred in nine months.

Fig. 26c SEP 57. Deformity developing in the tarsal bones.

Fig. 26d JUL 58. Marked loss of the body of the talus with involvement of the tibia and calcaneum.

PATIENT NO. 454

Female, aged sixty-one on readmission in SEP 62, with BB leprosy of many years duration. She had received treatment for some years and was B.I. negative. Stocking anaesthesia of the legs and multiple deformities of hands and feet were present.

She was readmitted for treatment of ulceration of both feet and bilateral foot-drop; right for ten years and left for three years.

On admission a radiograph showed ankylosis of the right ankle with some plantarflexion (Fig. 27).

She had a history of chronic ulceration of the feet but could not give any history relevant to the ankylosis.

COMMENT

The ankylosis had presumably followed a bone lesion of the talus and/or tibia, and the position of the ankle was such that, in walking, pressure was localised to the lateral border of the foot and chronic ulceration had occurred. An arthrodesis in a more satisfactory position, has allowed her to remain ulcer free for five years.

Fig. 27a Solid bony ankylosis of the right ankle in equinovarus position.

Fig. 27b Ulceration on soles of feet on readmission.

PATIENT NO. 546

Male, aged twenty-five on admission in MAR 56, with BT leprosy of fifteen years duration. His B.I. was negative.

No record is available of his anaesthesia and paralysis. He had infected trophic ulcers on both feet.

On 8 JUN 56 his right ankle was reported as being swollen with a discharging sinus which was opened and drained on 29 JUN 56 and eventually closed. Radiographs on 7 AUG 56 showed marked involvement of talus and less severe involvement of other bones (Fig. 28a).

By 23 OCT the bone destruction had progressed considerably in spite of bed rest and antibiotics. But immobilisation was not persisted with, though he did have walking plasters for a few weeks on each of a number of occasions in the next nine months. By 4 JUN 57 there was gross deformity of the ankle (Fig. 28b) and a below knee amputation was performed on 27 AUG 57.

COMMENT

This is a typical history of ten to twenty years ago when many of these lesions progressed to the stage when amputation was considered essential. In view of the presence of the sinus, this lesion may have commenced as an osteomyelitis, but the later appearance is not conclusive.

Fig. 28a AUG 56. First available radiograph shows active lesion, with bone destruction almost confined to the talus.

Fig. 28b JUN 57. The lesion has progressed considerably during a period of ten months and there is now marked deformity of the calcaneum and tibia.

PATIENT NO. 771

Female, aged fifteen on admission in NOV 56, with atypical lepromatous leprosy and multiple ulcers on both her legs. Her B.I. was 0.3.

The first record of anaesthesia was taken in 1960 when both legs were reported as showing patchy anaesthesia. She had a left foot-drop on admission.

Due to the ulceration of her legs she was hospitalised from NOV 56 to APR 57. On 30 APR 57 after discharge from hospital she complained of swelling and tenderness of the left ankle. The records state "from injury", but no indication of what injury is given. No active treatment was given and on 15 JUL 57 she still complained of a swollen left ankle.

On 1 AUG 57 a lateral radiograph (Fig. 29) showed a chip off the talus head and narrowing of the talo-navicular joint. A fitted plaster with a walking iron was applied to the left foot until 3 JAN 58 when it was removed and she was allowed to walk with crutches in the hospital. On 1 FEB 58 a radiograph was taken and reported on as "no change". However, in retrospect it can be seen that there was some healing of the talo-navicular joint area but there was also distortion of the naviculo-cuneiform and the cuneiform-metatarsal joints which was not seen earlier. It should be noted that she had been walking with crutches for one month after removal of the walking plaster prior to this radiograph, without specific support to her foot.

On 9 FEB 58 she was discharged from hospital. On 2 MAY 58 a left tibialis posterior transfer was performed to correct her foot-drop. The tendon was inserted into the surface of the navicular bone using a wire suture. She was in a non-weight bearing plaster cast for six weeks. On the removal of the plaster she resumed walking at once and was discharged from hospital a week later.

The foot became swollen and remained so. On 5 SEP 58 an oblique radiograph showed deformity of the talus head and the distal lateral portion of the calcaneum. No specific therapy was ordered. On 18 MAR 59 radiographs showed the talus head very compressed and the arch of the foot had collapsed (Fig. 29b). A walking plaster cast was applied on 23 MAR 59. Further films show osteoporosis but progressive healing and no further collapse. On 10 DEC 59 an arteriogram showed a normal dorsalis pedis artery and its branches.

The final walking plaster was removed on 18 JAN 60 and she was allowed up on crutches, but those were soon discarded. Radiographs on 9 JUN 61 showed that the tarsal bones had healed but there was disorganisation of the tarsal joints, and deformity of the foot generally (Fig. 29c).

COMMENT

It would appear that the initial trauma was relatively minor and that the treatment with a plaster cast and a walking iron had encouraged healing but support had not been continued long enough to allow full healing. The surgical interference and six weeks immobilisation without walking, followed at once by unlimited walking, probably reactivated the bone breakdown. This was allowed to continue for more than six months; even so, the foot healed in a reasonable position, with little disability though an obviously disorganised tarsal area remained. It is presumed that a longer period in the walking plaster initially would have achieved better healing and that it would have been desirable to either delay the surgery till full healing was achieved, or else to institute a much slower return to full walking to allow recalcification of the obviously osteoporotic bones, as has since been done with other patients.

A verbal communication from the patient in 1969 indicated that her foot caused no trouble, but she was not available to come for radiographic examination.

Fig. 29a AUG 57. Chip off head of talus. The swelling was present for the previous three months.

Fig. 29b MAR 59. Compression of the head of talus and collapse of the longitudinal arch.
The wire was used for attachment of the tibialis posterior tendon.

Fig. 29c JUN 61 healing has occurred in deformed but functional position.

PATIENT NO. 780

Male, aged thirty-two on admission in DEC 56, with lepromatous leprosy. His B.I. was 4.0. He had received therapy for two years from 1951-3 and said his first lesions appeared in 1950.

On admission he had stocking anaesthesia of both legs below the knees and there were ulcers under the first and fifth metatarsal heads of both feet. He also had claw hands. In JAN 57 a lateral radiograph of the right ankle and foot was normal but the left foot was not examined.

On 16 APR 59 bilateral amputation of the fifth toe and metatarsal head was performed for chronic ulceration, and on 28 MAY 59 he was permitted partial weight bearing on crutches for several weeks before resuming normal walking. He complained of a left swollen foot which was ignored until 19 SEP 59 when it was very swollen and he was ordered to use crutches again and a crepe bandage support, but there was little improvement in the degree of swelling.

On 16 OCT 59 a lateral radiograph (Fig. 30a) showed definite disintegration of the left navicular bone and a fracture of the left fourth metataraal shaft for which he was admitted to hospital for strict bed rest. On 12 NOV 59 he had an arteriogram and a biopsy of the navicular. The arteriogram showed normal main vessels around the disintegrating navicular (see page 206).

The biopsy showed disorganisation of the articular surfaces with the collapse of sub-chondral bone and formation of callus-like tissue in the marrow spaces (see page 197 for report).

On 13 NOV 59 a well-fitting plaster with a walking iron was applied which was changed on 22 JAN 60 when radiographs showed good healing of the navicular lesion.

On 20 OCT 60 a lateral radiograph showed apparent complete healing of the lesion with loss of talo-navicular joint space (Fig. 30b). He was allowed to resume walking with a crepe bandage support and left hospital on 7 NOV 60. There were no further complaints regarding his left foot.

Serial radiographs showed progressive change until 27 FEB 62 when increased sclerosis was present (Fig. 30c).

He was called for follow-up and a radiograph taken on 14 AUG 69 showed that there was no change in the left navicular, but some deformity of the left cuneiform-metatarsal area had occurred, so that the cuneiforms, the cuboid and the metatarsal heads now formed a solid block, yet the functional shape of the foot was still satisfactory.

He was readmitted in MAY 71 with an impacted fracture of the left calcaneum. The navicular appeared stable.

However, in MAY 71 radiographic examination of the right foot showed a healing lesion of the talo-navicular area (Fig. 30d). There had obviously been a marked disintegration which was healing with some deformity. Previous radiographs of the right tarsal bones taken in DEC 69 had appeared normal. At that time he was hospitalised for five months for an infected right fourth toe and other surgery. The toe was eventually amputated in MAR 70 and he resumed work as a sanitary cleaner on 1 APRIL 70. He stated that painless swelling of the right foot appeared soon after and persisted for nine months. The resultant foot has a collapsed arch (Pattern 2, page 83) but remains functional.

COMMENT

This history illustrates an early lesion in which healing continued between 16 OCT 59 and 27 FEB 62 with the aid of a walking plaster

for the first twelve months and remained stable until MAY 71 - a period of over nine years. It also shows spontaneous healing of a lesion of the metatarso-cuneiform area of the same foot which occurred at a later date.

The right foot illustrates the possibility of stress fracture and disintegration occurring on the sudden resumption of normal activity after prolonged bed rest and reduced activity, and of the spontaneous healing of such a lesion with a resultant functional foot.

Fig. 30a OCT 59. Disintegrating lesion of navicular bone.

Fig. 30b OCT 60. Healing of navicular bone.

Fig. 30c FEB 62. Increased calcification in healed bone (left foot).

Fig. 30d MAY 71. Healing lesion of the right talo-navicular area. Disintegration probably commenced in APR 70.

PATIENT NO. 1017

Male, aged fifty-two when admitted in NOV 58, with lepromatous leprosy. His B.I. was 3.5. His feet, except the left sole, were anaesthetic to touch on admission, but by 1962 a measure of sensation had returned to the left foot. The right foot remained anaesthetic; it also showed clawing of the toes.

The patient had many ulcers on the right foot in 1959-60. The first available lateral radiograph was taken on 23 APR 65 and showed no tarsal bone lesions. On 3 JUN 65 it was reported that he had a large ulcer on the right big toe and, because he refused to use crutches, a walking plaster was applied. This remained on for six weeks and was removed on 16 JUL 65 when the ulcer was reported as being fully healed. The patient was advised to limit his walking, but he did not, nor would he use crutches. Later he complained of a swollen foot and on 27 AUG 65 a lateral radiograph (Fig. 31a) showed definite activity in the first metatarsal base-cuneiform area, but no evidence of bone breakdown could be seen in the DP view.

A walking plaster was applied for four months and the radiograph of 14 DEC 65 (Fig. 31b) showed good healing of the metatarsal base-cuneiform area, but decalcification of the foot had occurred.

A walking plaster cast was reapplied for a further twelve weeks, during which time further healing of the tarsal bone lesion had occurred. On 6 JUL 66 a radiograph showed good healing of the meta-tarso-cuneiform area which remained healed until his discharge in 1968.

COMMENT

This patient was allowed to resume walking, which he did injudiciously immediately on the removal of a walking plaster cast which had been on for six weeks, and before a radiograph was taken. This

history is a good example of what can happen when a patient commences unrestricted walking immediately upon the removal of a walking plaster cast, even though he was normally active up until the time of application of the cast. It is suspected that the incidence of these lesions is under-estimated in out-patient clinic work, or where radiograms are not frequently taken.

Fig. 31a AUG 65. Early lesion of first metatarso-cuneiform joint area.

Fig. 31b JUL 66. Same lesion healed eleven months later.

PATIENT NO. 1043

Female, aged forty-nine when readmitted 25 APR 69, as a long standing lepromatous case. Her B.I. was negative. Both legs showed stocking anaesthesia below the mid-thighs and had been so since at least 1961. She had a left foot-drop correction performed on 23 SEP 60 and right claw toe correction on 25 NOV 60. She had a right foot-drop correction performed on 26 JAN 65 using the tibialis posterior tendon inserted into the tibialis anterior and the peroneus brevis tendons. She was walking well in early MAY 65 and discharged from hospital on 20 MAY 65. At that time radiographs showed some irregularity of left tarsal bones but the right tarsal bones appeared normal.

In APR 69 she was seen by the investigator at an out-patient clinic with a grossly swollen right foot and ankle. The history obtained indicated an initial mild swelling which appeared in NOV 68 but no treatment was given. By February the foot was painful when standing, and more swollen and less stable. She had been given antibiotics on three ocasions each for ten days duration and in March an incision for drainage was performed, but no pus was obtained. During this time she had remained at home with minimal walking, cared for by her family.

At that time, on palpation, it was obvious that there was gross destruction of the tarsal bones and possibly of the ankle bones. The patient's general physical condition was pitiable. She was anaemic and weak. The surgical wound had not healed but there was no frank sepsis. A plaster cast to protect the foot was applied in out-patients on 16 APR while admission was arranged.

This plaster was removed on 1 MAY 69 and radiographs were taken. The right foot showed gross destruction of all tarsal bones in front of the talus with involvement of the front of the calcaneum, the lower end of tibia and the metatarsal bases (Figs. 32a and 32b).

The incision wound had healed, and some swelling had subsided. A new fitted plaster was applied with a walking iron to allow weight bearing from the tibia only, and with a soft foot extension to maintain the foot in a functional position. She was encouraged to walk as much as possible.

The patient's general physical condition rapidly improved. The plaster was removed at three monthly intervals and radiographic examination done. These showed serial improvement in the bone condition with much reconstruction of bone, as seen in Figs. 32c and 32d of JAN 70. Free walking was allowed in DEC 70 and the patient was discharged home in MAR 71 to return for radiographic follow-up.

COMMENT

This case provides a good picture of the severity of destruction that can develop in six months of neglect. This patient is reliable and hence it is unlikely that the lesion was present for much longer than the stated period. It shows that even a gross bone deficit on radiographic examination can be compensated for in a few months of conservative therapy.

Fig. 32a

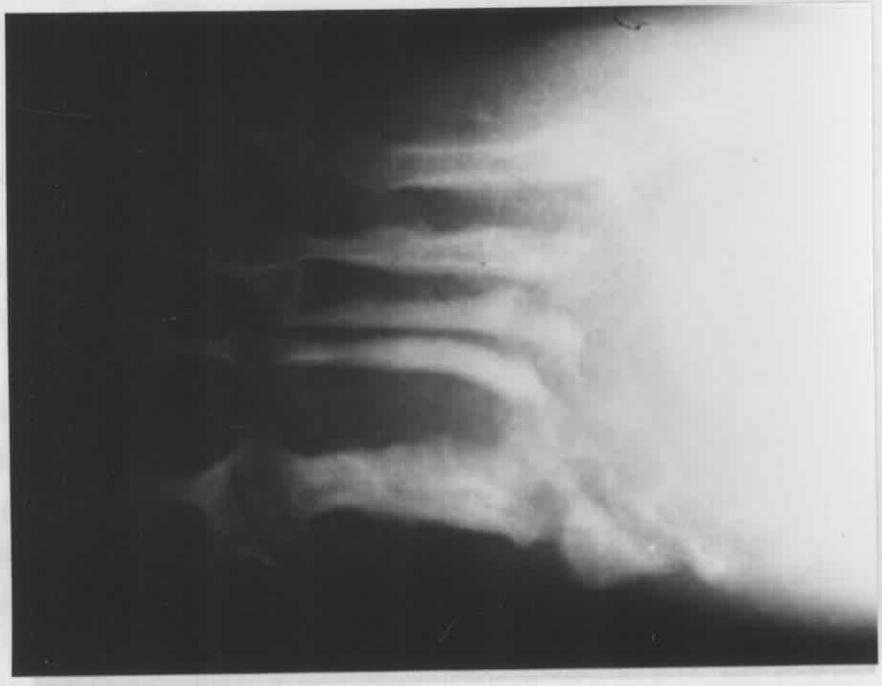


Fig. 32b

Fig. 32 a & b APR 69. Marked decalcification and disintegration present on readmission.

Fig. 32c

Fig. 32d

Fig. 32 c & d Increased calcification and reconstruction of the foot that has occurred during nine months of immobilisation in a walking plaster cast.

PATIENT NO. 1126

Male, aged thirty-six on admission in SEP 59; a grossly neglected atypical lepromatous case with a B.I. of 4.0, due partly to irregular therapy over many years. On admission he had stocking anaesthesia of both legs below the knee and of both arms and hands. He also had complete paralysis of all muscles of the right leg below the knee and triple palsy of the right hand, as well as a low ulnar, and a low median palsy of the left hand, and a partial facial paralysis.

Routine radiographs showed possible early breakdown of the lateral cuneiforms of the left foot (Fig. 33a) in OCT 64. No history of trauma could be elicitied. Previous radiographs did not show any tarsal bone lesion of the left foot.

He was advised to have a walking plaster but he refused. Serial radiographs showed further disintegration in spite of attempted conservative management, by the use of a plaster gutter splint and bed rest. He eventually agreed to a walking plaster cast in late FEB 65 when it was obvious that the lesion was not healing but getting worse. He removed this cast himself in early April when he absconded from our care. However, he returned of his own free will on 21 MAY 65 - six weeks later - as the affected foot was now grossly swollen and painful and he had developed severe generalised lepra reaction.

On 21 MAY 65 radiographs showed complete disorganisation of the navicular with extrusion of the fragments on the medial and dorsal aspects of the foot. The head of the talus was in contact with the cuneiforms (Fig. 33b) and there was flattening of the arch of the foot (Fig. 33c).

He was hospitalised with the swollen foot on a splint and elevated. On 1 JUN 65 his general condition had improved enough to operate. An iliac crest bone graft was used to replace the lost navicular and

to stabilise the arch. It was felt that haste was desirable and he would probably not agree to surgery once he felt he was improving. Biopsy was done at the same time, which showed regrowth of bone, and fibrous replacement of bone in areas of aseptic necrosis (report in full on page 198).

Well fitted walking plaster casts were continued for nine months, during which time the patient was not allowed to leave the institution. In MAR 66 limited walking was allowed. By 6 JUL 66 radiographs showed that healing was progressing satisfactorily (Figs. 33d and 33e). The metal pin was still in situ and the mass of bone on the dorsum of the foot that was originally navicular was still visible. The swelling had now subsided and function was good; there was no ulceration. On 4 DEC 66 he requested leave for business reasons and once again he did not return. He has been seen in the out-patient department, and the foot appears to be of reasonable shape without ulceration, but it was not possible to obtain new radiographs.

COMMENT

This is an excellent example of how bones can disintegrate very rapidly. The progress of the lesions during the use of protective splinting and bed rest shows that these do not provide adequate immobilisation to encourage healing. The very rapid destruction of the bones that occurred between FEB 65 and MAY 65 can presumably be credited to unrestricted walking, after his abscondment, on a foot that was grossly decalcified and weakened from almost six months of bed rest and limited activity.

This patient demonstrates that healing after surgery can be achieved within nine months in a walking plaster in spite of the poor state of the foot at the time of surgery. Although it was only possible to continue the radiographic follow-up for eight months after the removal of the final plaster and commencement of walking, this legnth of time appeared sufficient to indicate that the original lesions and the surgical scars had healed.

Fig. 33a NOV 64. Early lateral cuneiform lesion.

Fig. 33b

Fig. 33c

Fig. 33 b & c MAY 65. Marked collapse of the navicular at the time of readmission just prior to surgery.

Fig. 33d

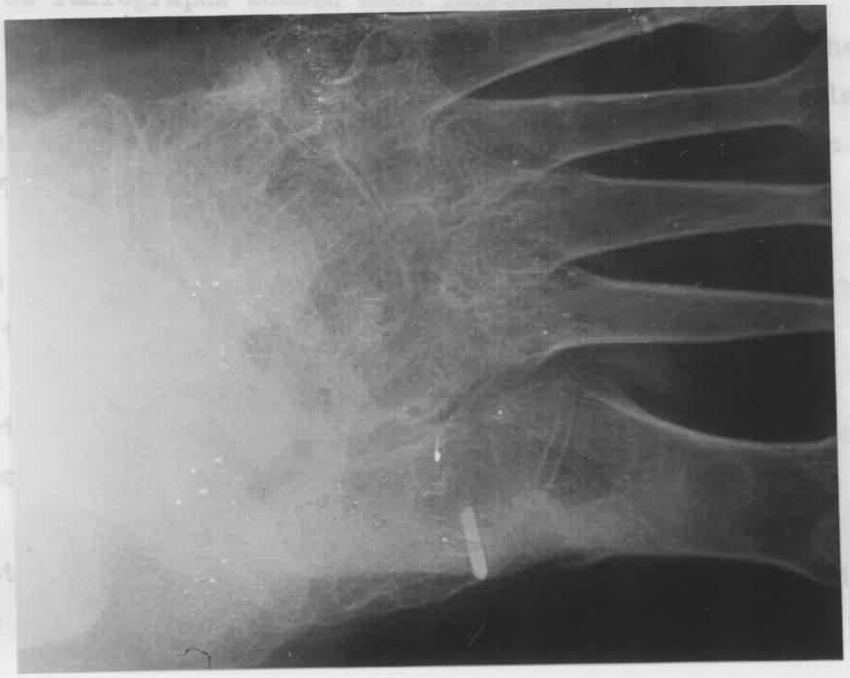


Fig. 33e

Fig. 33 d & e Healing in a satisfactory position has been achieved by nine months immobilisation after bone grafting to replace disintegrated navicular (one pin still in situ).

PATIENT NO. 1154

Male, aged thirty-three on admission in NOV 59, with BT leprosy of seven years duration. His skin smear was negative. He had not had any antileprotic drugs. He had stocking anaesthesia of both legs up to the groin and bilateral foot-drop. On admission the left foot was grossly swollen and discharged large amounts of foul pus from multiple sinuses (see Fig. 34).

Radiograph of 9 DEC 59 (Fig. 34b) showed marked decalcification of all foot bones with gross destruction of the metatarsals, phalanges, tarsal bones and the lower end of the tibia. He was put on strict bed rest with antibiotics and local treatment. Removal of sequestra was performed as they were identified but he was not provided with any form of splint.

On 2 JUN 60 radiographs showed much improvement in the bone condition and by 17 NOV 60 there was very good calcification of the remaining foot bones with smoothing off of the tibia to form a false joint with the tarsal bones. The foot was held in plantar flexion (Fig. 34d). The infection had cleared.

On 10 AUG 62 a left foot-drop correction was performed, together with Achillis tendon lengthening, and the use of extra strips of fascia lata to help provide stabilisation of the ankle area. This appeared effective and enough stability was achieved for the patient to walk in a boot that laced above the ankle.

It was noted that both legs showed marked interosseous calcification (see also Appendix I, page 222).

COMMENT

This patient shows that it is possible to salvage most of the bone that is still present even though it appears highly decalcified

on the radiographs. If a policy of conservative therapy with antibiotic cover and rest is maintained it is interesting to note how comparatively little sequestra is discharged or needs to be removed.

It is, however, important to keep the feet in a good position during the healing phase. In retrospect it would appear that if this patient had been provided with a splint to immobilise his leg with his foot at right angles to his tibia while the infection was being controlled, he may have developed a spontaneous ankle fusion which would have produced a much more stable and useful foot than was eventually obtained. This has been seen to occur in other patients with similar conditions.

Fig. 34a NOV 59. External appearance on admission. Pus discharge from ankle sinus.

Fig. 34b Radiographic appearance of the above.

Fig. 34c External appearance after twelve months treatment.

Fig. 34d JAN 61. Radiographic appearance.

PATIENT NO. 1318

Male, aged twenty-seven on admission in APR 61, with atypical tuberculoid leprosy. His skin smear was near negative but he had polyneuritis with a left claw hand and a left foot-drop. He had stocking anaesthesia of the left leg below the knee. On admission he had some lepra reaction which settled rapidly but due to the unusual pigmentation of his face he did not leave the institution as soon as he might have done. Hence there was opportunity to observe the changes in his left foot.

On 13 JUL 62 a left foot-drop correction using tibialis posterior transfer with a tendon bone attachment was performed under spinal anaesthesia. On 2 SEP 62 he commenced walking - i.e. after six weeks in a non-weight bearing plaster, and he made no complaints of discomfort in the left foot up to the time of his discharge from hospital on 28 SEP 62.

A radiograph on 27 DEC 62 (Fig. 35a) showed some osteoporosis and irregularity of the dorsal surface of the talus, navicular and cuneiforms. During the absence of the investigator there was partial limitation of activity for the next six months and then no further treatment was given, though radiographs taken in JUN 63 showed obvious progressive bone changes.

On the investigator's return in MAY 64 a radiograph showed that the talo-navicular-cuneiform area was one solid mass of well calcified bone but the arch of the foot had completely collapsed (Fig. 35b). Clinically the foot was obviously flattened, with the distal half rotated laterally at the mid-tarsal joint, so that the head of the talus appeared to rest on the ground as the patient walked and the overlying skin was already showing signs of pressure and incipient ulceration. There was no heat or swelling and it is presumed that bone healing was complete.

In AUG 64 a double wedge osteotomy was performed to reconstruct the arch of the foot and to correct the lateral deviation of the forefoot. A below-knee walking plaster was applied on 29 AUG 64, approximately three weeks after surgery, making sure that the plaster was well moulded to maintain the arch of the foot in the desired position.

In DEC 64 trial walking was allowed but heat and swelling rapidly developed.

On 5 JAN 65 a plaster shoe was applied to maintain the arch and to allow ankle movement. This was replaced as needed until the end of May when trial walking was again allowed. No further heat and swelling occurred.

Follow-up radiograph (Fig. 35c) on 25 APR 66 showed fusion of the talo-navicular-cuneiform bones with some correction of the arch but there were still residual bony irregularities on the dorsum of the foot over the first metatarsal base area. There had been no attempt to correct this deformity at surgery. He could wear normal shoes again and there was no abnormal pressure on the longitudinal arch of his foot. The patient left our care in JUN 67 with no further complaints regarding his foot.

COMMENT

When the osteoporosis was seen in DEC 62, the investigator warned that the foot should be rested and closely observed, but in her prolonged absence this was not done, which allowed the long follow-up to complete healing in a deformed position. This was the first patient in whom osteotomy through sclerosed bone was attempted, but healing was satisfactory. This was also the first patient in whom trial walking was used.

Fig. 35a Early hazy lesion of talus and navicular (DEC 62) was seen and the possibility of collapse of the talus was predicted.

Fig. 35b Healing of lesions had occurred but there was deformity of the talus and collapse of the arch and incipient ulceration was developing.

Fig. 35c After wedge osteotomy there was enough reconstruction of the arch (APR 66) to prevent ulceration of it.

PATIENT NO. 1582

Male, aged thirty-five on admission in JUL 63, with lepromatous leprosy. His B.I. was 4.7. He already had a left foot-drop and stocking anaesthesia of both legs below mid-thigh. There was also muscle weakness of the right leg which lessened over the next couple of years.

In 1969 he suffered from a badly infected left fourth toe and as a result a walking plaster was applied in early December. He was later hospitalised for removal of the plaster and a sequestrum. In FEB 1970 the radiograph showed marked decalcification of all the foot bones (Fig. 36a), and a warning was issued by the investigator that walking would need to be resumed extremely slowly to prevent tarsal bone disintegration from occurring. This was not done and in spite of swelling of the left foot and some pain in the foot, during the absence of the investigator, this patient was discharged from hospital on 23 MAR 70 having been walking for only a few days. On 11 MAY he complained of a swollen ankle. A crepe bandage was applied and he continued free walking but no radiograph was taken.

In JUN 70 the foot was still grossly swollen and a radiograph was taken on the 15th which showed an abnormality of the talus head. Although there was a query that it was disintegrating, the patient continued to walk. A further radiograph was taken on 23 JUN which showed definite disintegration and impaction of the head of the talus, with some collapse of the longitudinal arch and fracture of the fifth metatarsal base (Fig. 36b). Walking still continued until 29 JUN when he was admitted to hospital. A walking plaster was applied by a technician on 1 JUL without any attempt being made to correct the deformed position of the foot.

On 19 AUG the attention of the investigator was drawn to the condition of this patient's foot and he was immediately readmitted to hospital. The plaster cast was removed. Clinically there was

marked flattening of the arch of the foot. Radiographs on 20 AUG showed a marked compression of the talus head and the fracture of the fifth metatarsal base. A new plaster was applied by the investigator on 25 AUG and attempts at correcting the deformity resulted in some distraction of the impacted fragments of talus and reconstruction of the arch, with better approximation of the fragments of the metatarsal base.

Radiographs in DEC showed that reconstruction of the talus head was occurring in a much better position and the arch of the foot was improved. Bone healing appeared complete in APR 71 (Fig. 36d) and trial walking was duly instituted, after the foot-drop was corrected and the deformed fifth metatarsal head was surgically reshaped to prevent ulceration.

COMMENT

This case history illustrates how quickly deformity can develop. There was a marked change in the condition of the head of the talus between 15 JUN 70 and 23 JUN 70 and a corresponding collapse of the longitudinal arch of the foot. It also shows how relatively easy it is to reconstruct the arch of the foot during the phase when the bones are still only partially healed. It also illustrates that it is possible to predict some patients at risk.

Fig. 36a FEB 70. Marked decalcification of all foot bones following immobilisation for four months for an infected fourth toe.

Fig. 36b 15 JUN 70. Disintegration of talus has commenced and fracture of fifth metatarsal head has occurred.

Fig. 36c 23 JUN 70. Marked further collapse of the talus has occurred in eight days, with distraction of the fragments of the head of the fifth metatarsal.

Fig. 36d 28 APR 71. Healed lesion after distraction of fragments and six months walking plaster.

PATIENT NO. 1611

Female, aged fifty-two on admission in OCT 63, with gross deformity of hands and feet due to borderline leprosy of at least thirty years duration. Antileprotic drugs had been taken for seven years before admission. Her B.I. was negative. She had stocking anaesthesia below the knees. The right foot showed loss of toes, probably from sepsis, and a healed lesion of the talus and navicular which remained stable throughout her hospitalisation.

There was an ulcer over the cuboid area of the left foot and radiographic examination showed active bone lesion of the talus and navicular and complete loss of the normal architecture of the other tarsal bones (Fig. 37a). In the absence of the investigator she was treated by bed rest without local immobilisation. No active methods were employed to ensure rest and it is assumed that she continued limited walking. The deformity slowly increased until there was a marked prominence of the cuboid bone associated with collapse of the navicular, resulting in a "boat shaped" foot (Fig. 37b) with chronic ulceration over the cuboid prominance (Pattern 3, page 84).

COMMENT

A combined lesion, but impossible when first seen to say if ulceration followed or preceded the marked deformity, but deformity increased rapidly in this patient in spite of the local therapy, limited activity and control of infection. In retrospect it can be assumed that healing in a functional position could have been achieved without surgery if the foot had been immobilised in a carefully shaped walking plaster as soon as the ulceration would permit. It may have been possible to mould the foot to improve its shape when first seen, or at least to prevent further deformity, but this was not attempted.

Fig. 37a OCT 63. On admission, active bone disintegration in association with osteomyelitis was present.

Fig. 37b Increased deformity has occurred during twelve months, together with bone healing with resultant "boat shaped" foot deformity that is prone to ulceration over the cuboid.

PATIENT NO. 1646

Male, aged twenty-nine on admission in FEB 64, with borderline leprosy of fourteen years duration. He received Dapsone from 1958 to 1963 and his B.I. was negative but he had multiple deformities and was admitted for surgery.

On admission he had bilateral dropped feet, right for seven years and left for eleven years, and both legs showed stocking anaesthesia below mid-thigh. He also had ulceration of lateral side of his right foot and osteomyelitis of the right fourth and fifth metatarsals. On 26 JUN 64 a left foot-drop correction was performed with a good result and no immediate post-operative complication. However, due to slow healing of the osteomyelitis of the right foot he was not walking until NOV 64.

Routine radiographs on 26 JAN 65 showed early breakdown of left navicular (Fig. 38) - patient had no complaints about the foot and swelling was not obvious. On 16 FEB 65 a biopsy was taken of part of the navicular bone (see report on page 201), and then a walking plaster was applied to the left foot and changed as needed until 7 JUL 65 when he commenced to walk with firm bandage support. On 27 AUG 65 a lateral radiograph showed activity of the medial cuneiform and first metatarsal base. A walking plaster was applied for ten weeks and then trial walking was commenced.

In JAN 66 his foot felt hot and was erythematous, and radiographs suggested new activity of navicular and of the first metatarsal base area, but a supporting bandage was ordered and repeat radiographs in six weeks did not confirm any new activity.

Radiographs taken NOV 69 showed that the bones remained stable.

COMMENT

A case of acute breakdown occurring on resumption of full walking after six months of limited activity following surgery in an anaesthetic paralysed foot - detected early - biopsied and followed up for four full years after recovery without further tarsal involvement.

Fig. 38a Early disintegration of navicular. See also Fig. 11, page 71B.

Fig. 38b FEB 66. Lesion healed.

PATIENT NO. 1650

Male, aged twenty-nine on admission in MAR 64, with atypical lepromatous leprosy of five years duration. His B.I. was 4.0 on admission. He had not previously received antileprotic drugs.

He had a left foot-drop of four years duration with stocking anaesthesia of left leg below the knee, and obvious deformity of the left fifth toe associated with a chronic sinus under the fifth metatarsal head. There were no other ulcers and no other deformities.

An AP radiograph taken 26 MAR 64 showed loss of the fifth metatarsal head. In retrospect it can be seen that there was a slight abnormality of the left navicular, but this was not reported on at the time.

On 6 JUN 64 an oblique radiograph showed irregularities of the talus head and adjacent navicular which were not commented on. A walking plaster was used from 16 JUN 64 to 17 AUG 64 to ennourage healing of the sinus under the fifth metatarsal head.

On 28 SEP 64 the first lateral view radiograph showed increased lipping of the talus head and deformity of the navicular - at this time the patient stated that three years previously he had developed a swollen painful foot but had no memory of any accident. It was considered at the time that the lesion was stable and no therapy was given and no follow-up radiograph was ordered.

Further radiographs were taken in DEC 65 and the lateral view (Fig. 39a) showed further deformity of the talus head and the navicular, and it was realised that the lesion was active.

However, the patient absconded before further radiogfaphs could be taken or therapy instituted. In APR 69 he returned for more therapy. During his absence he had severe ulceration of the left

forefoot resulting in surgical amputation of the three middle toes through the metatarsal shafts.

On 28 APR 69 radiographs showed loss of the lateral part of the forefoot and marked tarsal bone activity associated with destruction of some bones, but this was partly obscured by swelling. The lateral radiograph showed compression of the body of the talus and marked distortion of the head and neck of the talus and of the navicular, with marked activity in the area of the talo-navicular joint and a prominence of the cuboid on the sole. An arteriogram was also done (see Fig. 19, page 77A and Table 2, page 206).

A walking plaster cast was applied and reapplied as necessary until bone healing appeared complete in JAN 70. It has remained stable since then.

COMMENT

This patient demonstrates the slowly progressive nature of some lesions. Radiographs showed activity for a period of five years.

In retrospect adequate therapy should have been given in 1964-5, but even after five years of active bone lesions, healing occurred within nine months once the foot was completely immobilised, and has remained stable for the next fifteen months.

Fig. 39a Lesion before patient absconded (DEC 65). Not obviously active but change in the shape of the talus has occurred in the previous six months.

Fig. 39b Lesion on return in 1969 showing deformity due to impaction of talus and navicular with collapse of the arch. Activity still present in the bone lesions.

PATIENT NO. 1754

Male, aged twenty-one on admission in MAR 65, with atypical lepromatous leprosy of five years duration. His B.I. was 4.2 and his M.I. 12%. He had had no previous antileprotic drugs. On admission he had patchy anaesthesia of feet and legs and some weakness of the left hand.

He had had an acute reactive episode in the middle of 1964 when he noticed some swelling of the right foot. After admission he had reaction with neuritis and swollen feet, for which he was ordered bandage support. Radiographs were taken on admission and the unusual appearance of the right talus was commented on but its significance was not appreciated. In JUL 65 the size of the lesion had increased slightly but it was better calcified and was obviously healing (Fig. 40). No specific therapy was given. Healing continued and has remained firm for five years in spite of recurrent neuritis and reaction.

COMMENT

This is a Pattern 1 type bone lesion (page 82) which was present and active on admission. Healing occurred spontaneously with slight deformity and no functional disability.

Fig. 40 Abnormal head of talus, asymptomless development and no residual disability.

PATIENT NO. 1921

Male, aged thirty-two on admission in MAY 67, with atypical lepromatous leprosy of twenty-five years duration. On admission his B.I. was 4.7. It would appear from his history that the initial lesion was a tuberculoid or near-tuberculoid lesion and no antileprotic drugs were taken. In the three years prior to admission many new lesions of different characteristics had appeared. Left foot-drop and claw toes were present for one year and weakness of dorsiflexion of the right foot for six months. Both hands became clawed in this time. Both legs showed stocking anaesthesia below the groins. He was in a highly active phase of the disease showing a tendency to lepra reaction.

In JUL 67 he developed an infected ulcer of the right foot and when the acute phase had subsided a walking plaster was applied on 5 AUG. On removal of the plaster on 27 SEP the ulcer was still present and a radiograph showed osteomyelitis of the fifth metatarsal head. A new walking plaster was applied and maintained until 20 FEB 68 when the ulcer was healed, and radiographs showed that the metatarsal head was also healed with some deformity. Walking was allowed under supervision commencing 24 FEB 68.

On 2 MAR he complained that the foot was hot and swollen and as this persisted radiographs were taken on 21 MAR 68 and again on 28 MAR 68. These showed early breakdown of the cuneiform bone and the first metatarsal base (Fig. 41a). A walking plaster was applied for three months. The patient continued to have a lot of lepra reaction at this stage and was hospitalised.

The plaster was removed on 12 JUN 68 and radiographs showed considerable improvement of the bone condition. As he had a number of plaster sores on the dorsum of the foot he was kept in a plaster back-slab and given physiotherapy but no walking was allowed until 27 AUG 68. For the next few months he had chronic lepra reaction

and remained in hospital on limited activity but walking was allowed, and his foot appeared satisfactory when he was discharged from hospital on 9 NOV 68.

On 12 NOV he again complained of a swollen foot and radiographs showed bone activity in the navicular (Fig. 41b), with some compression. A walking plaster was applied in NOV 68 and was reapplied in MAR 69 and used till JUL 69 when healing appeared firm radiographically. Then the patient was slowly allowed to resume normal walking under supervision. Radiograph of 22 JUL 69 lateral view showed healing of navicular and a normal calcaneum. The radiograph of 16 SEP 69, while he was still in hospital, showed compression of the calcaneum (Fig. 41c) that was apparently already healing. No further therapy was ordered for this lesion.

COMMENT

Treatment of ulceration of the feet by walking plasters is an accepted form of therapy for leprosy patients. In this patient the original ulcer was associated with osteomyelitis and so a period of immobilisation longer than usual was necessary to achieve healing. There were, however, recurrent episodes of bone breakdown each time walking was resumed after the use of a walking plaster.

There was some sepsis initially, also the use of walking plasters for four and a half months resulted in a considerable degree of osteoporosis. The patient's foot was totally anaesthetic and he had a complete foot-drop on admission, with indications of further increasing nerve deficit during his recurrent bouts of lepra reaction. He was a careless patient with little idea of looking after himself, and his resumption of walking had to be carefully supervised or he would walk without any restriction as soon as the plaster was removed. It is quite conceivable that he did in fact over-exert himself, and it is interesting to notice that while in hospital from AUG to NOV 68

there was no heat or swelling of the ankle although the navicular was damaged and he was walking on it. Within three days of leaving hospital his foot was hot and swollen and he complained of this, leading to a new radiographic examination and the diagnosis of a new lesion.

On healing of this lesion he was allowed to resume walking and within eight weeks showed a third lesion that was already healing but causing no symptoms. He had three distinct bone lesions, each affecting a different bone, and each occurring soon after walking was resumed on the removal of a walking plaster.

Fig. 41a Lesion of medial cuneiform and first metatarsal as diagnosed within four weeks of resumption of free walking after use of a walking plaster to encourage the healing of an ulcer associated with osteomyelitis of the fifth metatarsal head.

Fig. 41b Lesion of navicular which produced symptoms within seven days of discharge from hospital though it had apparently been present and asymptomatic before discharge.

Fig. 41c Lesion of calcaneum which was diagnosed by radiograph about eight weeks after the removal of plaster, when radiograph had shown a normal calcaneum. Sclerosing of navicular and altered talo-navicular relationship also seen in this film.

PATIENT NO. 1996

Male, aged forty-five on admission in AUG 68, with borderline leprosy of twelve years duration. He was self-treated with Promin. His B.I. was 2.7. Both legs were anaesthetic below the mid-thigh. The right foot showed marked deformity of the toes, apparently due to sepsis from ulceration associated with claw toes of five to six years duration. The left foot was grossly swollen and discharging foul pus from many deep sinuses (Fig. 42c).

Radiographs showed gross disorganisation of all bones of the foot (Figs. 42a and 42b). The tarsal bones and metatarsal bases were disintegrating, but the metatarsal shafts showed evidence of periosteal proliferation. A number of sequestra were later removed after a period of immobilisation and treatment along the lines described on page 60.

Once the sepsis was controlled walking plasters were utilised to enable him to weight bear from the tibia (see pages 58-59).

Recalcification of the fragments occurred rapidly. He is now walking satisfactorily, though he has some chronic scar tissue under the metatarsal heads that requires constant care.

COMMENT

This patient had already been informed that amputation was necessary. Recent radiographs show sclerosed bone and he is now able to walk in normal shoes.

Fig. 42a

Fig. 42b

Fig. 42 a & b AUG 68. Marked decalcification associated with osteomyelitis and disintegration on admission.

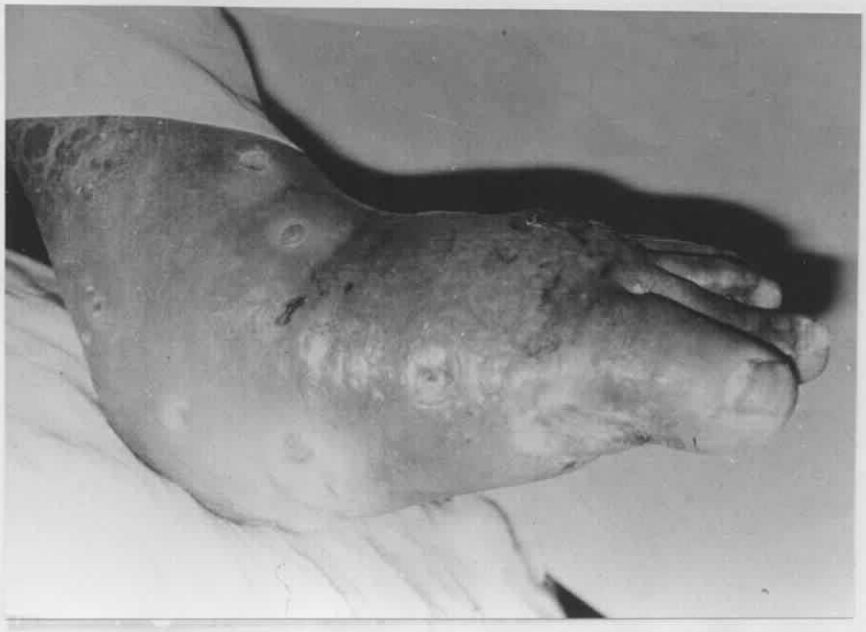


Fig. 42c AUG 68. External appearance of foot on admission.

Fig. 42d JUL 71. External appearance of foot.

Fig. 42e

Fig. 42f

Fig. 42 e & f Marked recalcification that occurred within twelve months (AUG 69).

PATIENT NO. 2011

Male, aged twenty-seven on admission in DEC 68, with atypical tuberculoid leprosy of fifteen years duration.

On admission his B.I. was negative. Both legs showed stocking anaesthesia below mid-calf and he had a large ulcer on the sole of his right foot of more than two years duration. The left toes were clawed. Neither foot showed weakness of dorsiflexion.

He gave a history of cutting his foot on coral and the resultant wound became infected and had not healed. The ulcer was about 8 cm x 6 cm in size, granulating but deep, with bone visible at the base. It was centred over the cuboid (Fig. 43a). Radiographs taken DEC 68, DP view, showed an apparently complete loss of the posterior portion of the cuboid and the anterior portion of the calcaneum (Fig. 43b). The lateral view confirmed the impression and showed complete collapse of talus and marked deformity ofthe calcaneum as well.

After initial therapy with antibiotics and local dressings, as described on page 60, a plaster cast with a walking iron to allow weight bearing from the tibia, was applied and changed as necessary until soft tissue healing was achieved. Then a wedge osteotomy was performed to provide a flat walking surface.

The patient is now ulcer free, wearing normal shoes (Fig. 43c).

COMMENT

Although some sensation remained, the patient continued to walk in spite of increasing deformity and ulceration. Infection is obviously the initiating factor in this patient.

Fig. 43a DEC 68. Chronic ulceration of the sole of the foot, present for five years following a coral cut.

Fig. 43b Marked loss of cuboid; probably due primarily to osteomyelitis, but now associated with collapse of other bones.

Fig. 43c DEC 68. Lateral radiograph shows marked deformity of the heel.

Fig. 43d MAR 71. Healed reconstructed foot.

Fig. 43e MAR 71. Sole of healed foot. Residual scars are present over the heel area, but the patient has learnt to keep his foot ulcer free. He is wearing normal soft-soled shoes.

Appendix F

EXTRACTS OF BIOPSY REPORTS

PATIENT NO. 309 M/28 10.3.60

SPECIMEN

Biopsy from (ankle) talo-navicular joint in patient with leprosy and neuropathic joint.

REPORT

Histopathology

The articular surface is largely destroyed and replaced by fibrous tissue and fibro-cartilage.

The marrow spaces immediately underlying the articular surface show slight fibrosis and some local increase in vascularity; the bone is otherwise normal.

(SIGNED) H. A. SISSONS
(LONDON)

PATIENT NO. 780 M/34 10.3.60

SPECIMEN

Biopsy from navicular in patient with leprosy and neuropathic joint.

REPORT

Histopathology

The specimen shows some, although not complete, disorganisation of the articular surface. The underlying bone shows replacement of its marrow spaces by vascular fibrous tissue with some newly formed bone. The histological appearances suggest that collapse of the sub-chondral bone has occurred, with the formation of callus-like tissue in the marrow spaces.

(SIGNED) H. A. SISSONS
(LONDON)

PATIENT NO. 1126 M/39 3.6.65

SPECIMEN

Bone from navicular, talus and cuneiforms in leprosy patient with neuropathic foot.

REPORT

Histopathology

All portions show regrowth of bone in areas where an aseptic necrosis has occurred.

1. This regrowth is irregular and fibrous in the navicular - a strip of fibrous tissue probably periosteum having been mistaken grossly as a piece of skin. In this fibrous and osteoblastic tissue, irregular osteoid is forming and together with irregular fragments of new bone is surrounding and incorporating fragments of old dead bone. It seems that as well as bone necrosis, fibrous replacement of bone has also occurred.

2. Under the cartilage in the section of talus there is also bone necrosis with rather poorly formed active regeneration by osteoid and fibrous tissue.

3. This fragment from cuneiform is similar to 2, but necrosis is much less marked and repair by osteoid is of much better quality. However, the cycle of breakdown and regrowth seems the same.

(SIGNED) JAMES B. GIBSON

PATIENT NO. 1493 F/30 30.7.64

SPECIMEN

Biopsy from disintegrating right navicular in the acute phase. Patient with active lepromatous leprosy.

REPORT

Histopathology

Acid fast bacilli have not been identified in any of the sections. Specimen 1. is a portion of chronically inflammed synovium which is oedematous and extensively infiltrated by plasma cells. The features are not specific.

Specimen 2. from lateral edge and 3. from deeper in the navicular bone show a sub-acute, partially suppurative osteomyelitis with bone destruction, and also marked osteoblastic activity with new bone formation. A few giant cells are present but there are no specific features about the material. The lesion is thought to be basically a trophic one, and may be compared with a Charcot's joint in this respect. I can find no evidence of an actual leprous infection of this area or of a primary vascular aetiology but perhaps vessels at a distance might be affected.

(SIGNED) JAMES B. GIBSON

PATIENT NO. 1493 F/30 7.3.65

SPECIMEN

Bone from healed deformed left foot. Patient with active lepromatous leprosy.

REPORT

Histopathology

The changes are generally less than those seen in previous cases.

I. In the pieces of navicular bone there appears to be some general osteoporosis but the main changes are cortical and articular with some fibrillation and cellularity of the articular cartilage and vascular and proliferative synovial changes with pigmentation, probably traumatic in origin.

The cortex is thin and osteoclasts and marrow fibrosis are rather prominent here with poor formation of bone close to cellular areas of cartilage.

Similar changes and pannus-like areas are seen in specimens III and IV, and also in II (head of talus).

The appearances may be summarised as a loss of well formed bone and replacement by softer vascular tissue at points of stress.

Vascular and nervous lesions are not seen.

(SIGNED) JAMES B. GIBSON

PATIENT NO. 1646 M/30 17.2.65

SPECIMEN

Fragments of disintegrating left navicular bone.

REPORT

Histopathology

Specimen 1 (submitted in gauze) is a fragment of bone with rather a thick periosteum overlying an irregular cortex of bone in which are interspersed areas of cartilage. Many trabeculae are thick and the marrow is notably vascular. There is considerable osteoblastic activity and some osteoclastic activity, but the main change appears to be patchy replacement of bone by irregular cartilage and fibro-cartilage.

Specimen 2 shows similar features with more osteoid. The excresences are made up largely of osteoid and irregular cartilage. In one small area there is a chronic inflammatory process.

There are no noteworthy changes in arteries and the few nerves present show nothing of note.

(SIGNED) JAMES B. GIBSON

PATIENT NO. 1656 M/15 15.3.66

SPECIMEN

Chips from right deformed talo-navicular joint, removed during reconstructive surgery.

REPORT

Histopathology

Microscopy shows some loss of mucopolysaccharide and apparent increase of fibrous tissue in the superficial cartilage. In places, there is some vascularisation of cartilage from below. The small amount of sub-chondral bone appears viable. There is no evidence of any active inflammatory process.

(SIGNED) DR. M. E. CATTO

PATIENT FROM KUALA LUMPUR M/35 2.4.66

SPECIMEN

Pieces of bone and cartilage removed during reconstructive surgery to reshape a deformed foot with active disintegration of the talus.

REPORT

Histopathology

Microscopy shows that the normal hyaline cartilage of the articular surface has been almost completely replaced by a mass of fibrocartilage and of collagen. Within this there are a few islands of hyaline cartilage and also numerous fragments of dead bone and cartilage. In places, remaining hyaline cartilage is being eroded from below by vascular granulation tissue and there is also new revascularisation of the middle layer of the cartilage with ossification, a thin rim of the original deeper cartilage being left surrounded by bone on both sides. The marrow spaces are a little fibrosed, more vascular than usual and with a sprinkling of chronic inflammatory cells. The bone trabeculae show evidence of very active remodelling, having fibre bone excrescences on their surfaces. In places the trabeculae are much broadened with irregular cement lines suggesting that active remodelling and resorption have continued for some time. There is no histological evidence in this material of aseptic bone necrosis. I think the small dead bone fragments embedded in the hyaline cartilage are probably the result of collapse and disintegration. There is no clear evidence in the broad remodelled trabeculae of central cores of necrotic bone as is seen many years after an ischaemic episode in bone from caison workers or from ischaemic femoral heads removed following fracture. It looks as though deep vascular erosion of the joint surface plays at least some part in its destruction.

(SIGNED) DR. M. E. CATTO

PATIENT'S NAME: ABDUL (KUALA LUMPUR) M/30 30.3.66

SPECIMEN

Bone from deformed foot - removed at reconstructive surgery - chronic history of three years of alternating breakdown and repair.

REPORT

Histopathology

The specimen consists partly of some slivers of fairly normal though thin lamellar trabeculae with a little oedema of the intervening fatty marrow. In addition, there are areas where the bone is more actively proliferating and there is some fibro-cartilage also in the fibrotic marrow spaces. A few foci of chronic inflammatory cells are seen.

Unfortunately these appearances are, I think, the result of collapse with its attendant bone remodelling. The cause of the collapse is not apparent.

(SIGNED) DR. M. E. CATTO

TABLE 1

DETAILS OF PATIENTS FROM WHOM BIOPSY WAS TAKEN

Patient's Number, Sex, age, Type, B.I. on admission	Neural involvement	Patient's complaint, Side affected, Significant history	Radiograph	Biopsy	Summary of Biopsy Report
No. 309 M/30 LL type B.I. 0.2	Bilateral foot-drop and stocking anaesthesia. Bilateral ulnar and median paralysis	23 APR 59 swollen right ankle. Tibialis posterior transfer for foot-drop performed on 18 DEC 58	Disintegrating navicular	12 NOV 59 with arteriogram - i.e. six months after lesion first seen	Articular surface destroyed, replaced by fibrous tissue and fibrocartilage. Fibrosis of marrow spaces and increased vascularity (See page 196)
No. 780 M/34 LL type B.I. 4.0	Left foot-drop, right claw toes. Bilateral stocking anaesthesia below knees. Bilateral ulnar and median paralysis	19 SEP 59 swollen left ankle. On 16 APR 59 had removal of left fifth toe and metatarsal head	Disintegrating navicular	31 OCT 59 with arteriogram	Collapse of subchondral bone. Formation of callus-like tissue in marrow spaces. Some disorganisation of articular surface (See page 197)
No. 1126 M/41 BL type B.I. 1.8	Bilateral stocking anaesthesia below knee. Right leg and right arm complete paralysis. Left claw hand. Bilateral facial weakness	Left foot No complaint	NOV 64 middle cuneiform haziness. Refused treatment. MAY 65 disorganised navicular and cuneiform	1 JUN 65 biopsy taken when graft inserted to replace navicular and to reconstruct the arch of the foot	Regrowth of bone and fibrous replacement of bone in areas of aseptic necrosis (see page 198)
No. 1493 F/30 BL type B.I. 3.5	Bilateral stocking anaesthesia. Bilateral foot-drop. Gross deformity of hands, feet and face	13 MAR 64 swollen right foot	Decalcification and gross disorganisation of talus, navicular and adjacent bones	28 JUL 64 biopsy taken when iliac crest graft inserted to stabilise foot	A sub-acute partially suppurative osteomyelitis, with bone destruction and marked osteoblastic activity. No evidence of leprous infection (See page 199)
		Left foot chronic deformity. Inversion of calcaneum and tendency to "boat shaped" foot with chronic ulceration	Deformity of talus and navicular present on admission	5 MAR 65 biopsy taken when wedge resection performed to realign left foot	Osteoporosis with loss of well formed bone and replacement by softer vascular tissue at areas of stress (See page 200)
No. 1646 M/29 BB type B.I. 0.0	Bilateral stocking anaesthesia below mid-thigh. Bilateral foot-drop. Bilateral ulnar and median paralysis	Left foot No complaint Tibialis posterior transfer for foot-drop performed on 26 JUN 64	JAN 65 disintegrating left navicular	FEB 65 as elective procedure	Patchy replacement of bone by irregular cartilage and fibro-cartilage. The marrow is very vascular. The periostium is thickened. (See page 201)
No. 1656 M/14 BB type B.I. 0.0	Bilateral foot-drop and claw toes. Left ulnar paralysis	Right arch flattening and forefoot deviation occurred three months after tibialis posterior transfer for foot-drop	Head of talus hazy and malalignment of talo-navicular joint	19 JUL 66 biopsy and arthrodesis of talo-navicular joint to stabilise foot	Increased fibrous tissue in the superficial cartilage and vascularisation from below (See page 202)

TABLE 2

DETAILS OF PATIENTS ON WHOM ARTERIOGRAPHY WAS PERFORMED

Patient's Number, Sex, Age, Type, B.I. on admission	Neural involvement	Side affected, Patient's complaint	Radiographic lesion, Date detected	Date of Arteriogram	Report	Comments
No. 25 M/38 BB type B.I. 0.0	Multiple nerve lesions, stocking anaesthesia of legs. Bilateral foot-drop	2 NOV 59 bruised left ankle. Swelling from then till DEC 59	DEC 59 navicular lesion	10 DEC 59	Good arterial filling (Fig. 19, page 77A)	Stable when last examined in 1969. No disability
No. 309 M/30 LL type B.I. 0.2	Bilateral stocking anaesthesia below knee and foot-drop. Multiple other nerve lesions, arms and face	23 APR 59 swollen right ankle. Tibialis posterior transfer, using bone insertion, to correct foot-drop was performed on 18 DEC 58	MAY 59 disintegration of navicular	a) 30 NOV 59 femoral arteriogram b) 12 NOV 59 posterior tibial arteriogram	a) Not successful b) Apparently normal filling	Healed with some flattening of the arch. No real disability due to lesion
No. 771 F/17 BL type B.I. 0.3	Both legs patchy anaesthesia. Left foot-drop. Right claw hand	30 APR 57 swollen left ankle stated to be due to injury	AUG 57 chip fracture of talus head	10 DEC 59	Good arterial filling round talo-navicular area	Flattened arch but functional foot. No real disability (See history page 168)
No. 780 M/34 LL type B.I. 4.0	Multiple nerve lesions. Stocking anaesthesia below knees	19 SEP 59 swollen left ankle. On 16 APR 59 had amputation of left fifth toe and metatarsal head	16 OCT 59 navicular disintegrating	12 NOV 59	Good arterial filling	In 1971 lesion stable and no real disability (See history page 170)
No. 1650 M/29 BL type B.I. 4.0	Left leg, stocking anaesthesia below knee and foot-drop. Left claw hand	Left foot No complaints	SEP 64 slight irregularity of talus. Slowly progressed without treatment till 1969	MAY 69 after talus collapse	Complete obstruction of dorsalis pedis artery (Fig. 20, page 77A)	Collapsed talus healed by JUN 70. Function fair, but residual "boat shaped" foot (See history page 188)

TABLE 3

PREVALENCE OF TARSAL BONE LESIONS SEEN IN 400 LEPROSY PATIENTS

See page 44 for Materials.

See page 53 for Methods.

TABLE 3

	FOOT	MALES	FEMALES	TOTAL	
Tarsal bone lesions healed on admission	LEFT	34	7	41	
	RIGHT	28	7	35	76
Tarsal bone lesions active on admission	LEFT	7	2	9	
	RIGHT	8	2	10	19
SUB - TOTAL		77	18	95	
Tarsal bone lesions that appeared while patient was under observation	LEFT	6	5	11	
	RIGHT	10	1	11	
SUB - TOTAL		16	6	22	
TARSAL BONE LESIONS SEEN IN SELECTED GROUP		93	24	117	

TABLE 4

DISTRIBUTION BY AGE ON ADMISSION OF THE INVESTIGATED PATIENTS

TABLE 4

AGE	AFFECTED PATIENTS	UNAFFECTED PATIENTS	TOTAL
0 - 9	0	4	4
10 - 19	4	43	47
20 - 29	19	77	96
30 - 39	20	76	96
40 - 49	16	47	63
50 - 59	22	36	58
60 and over	13	23	36
TOTAL	94	306	400

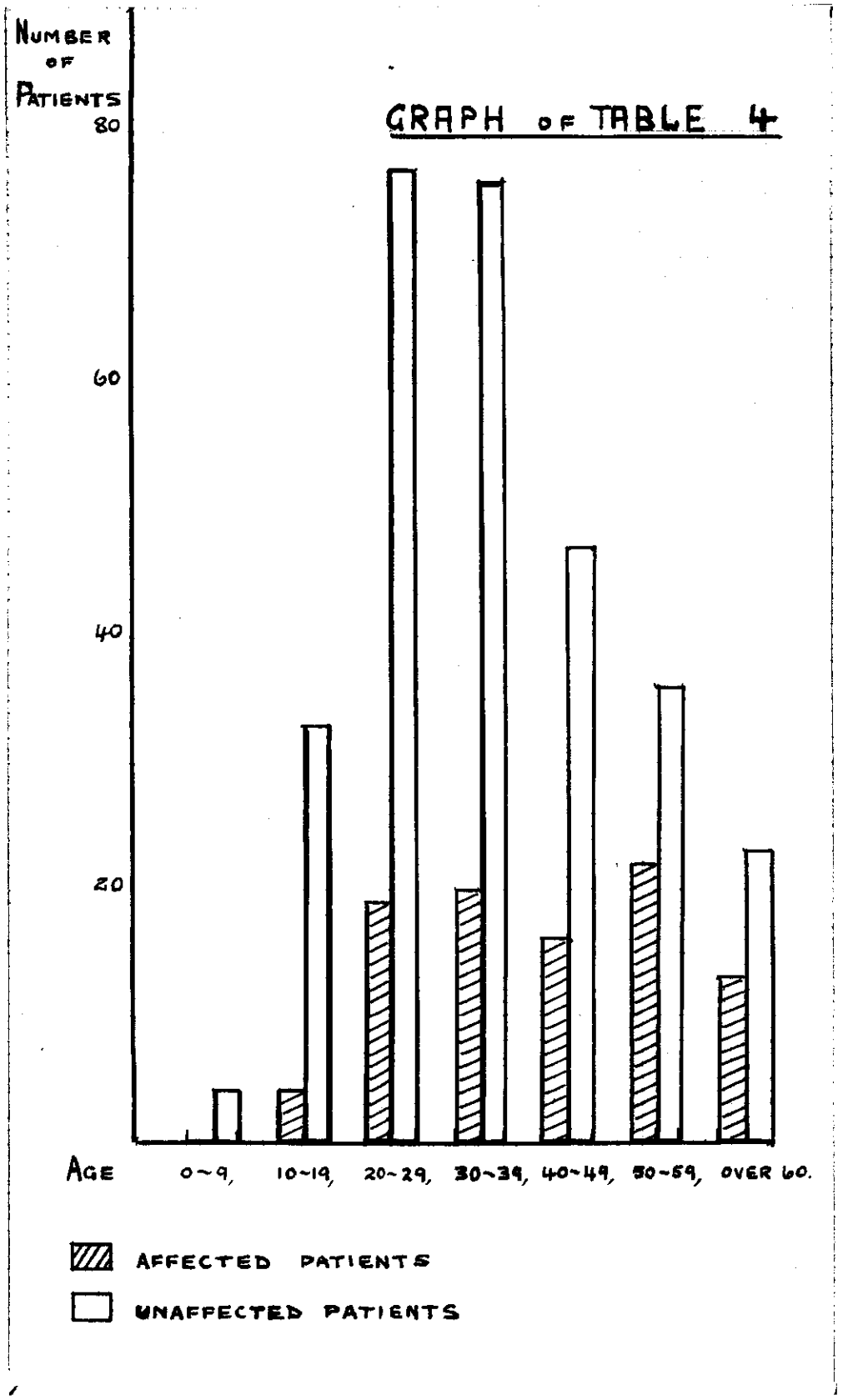


TABLE 5

DISTRIBUTION BY THE LEPROSY TYPE OF THE INVESTIGATED PATIENTS

See page 9 for classification of types.

TABLE 5

LEPROSY TYPE	AFFECTED PATIENTS	UNAFFECTED PATIENTS	TOTAL
LL	32	123	155
BL	15	70	85
BB	29	71	100
BT	17	25	42
TT	1	17	18
TOTAL	94	306	400

GRAPH OF TABLE 5

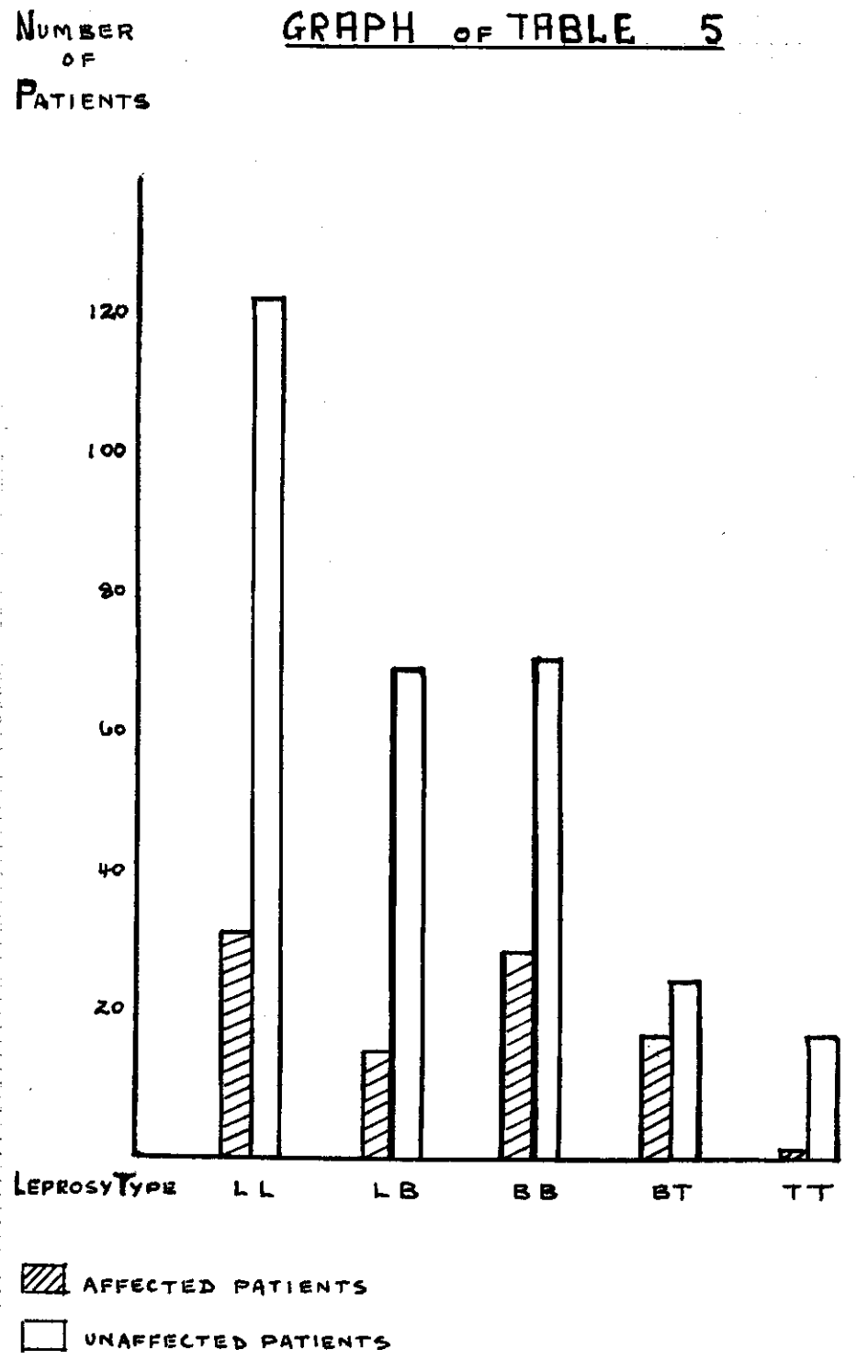


TABLE 6

DISTRIBUTION BY BACILLARY INDEX AS RECORDED ON ADMISSION FOR THE INVESTIGATED PATIENTS

For estimation of and significance of Bacillary Index, see page 157..

TABLE 6

BACILLARY INDEX	AFFECTED PATIENTS	UNAFFECTED PATIENTS	TOTAL
0.0 - 0.9	27	57	84
1.0 - 1.9	7	43	50
2.0 - 2.9	11	43	54
3.0 - 3.9	21	59	80
4.0 and over	28	104	132
TOTAL	94	306	400

GRAPH OF TABLE 6

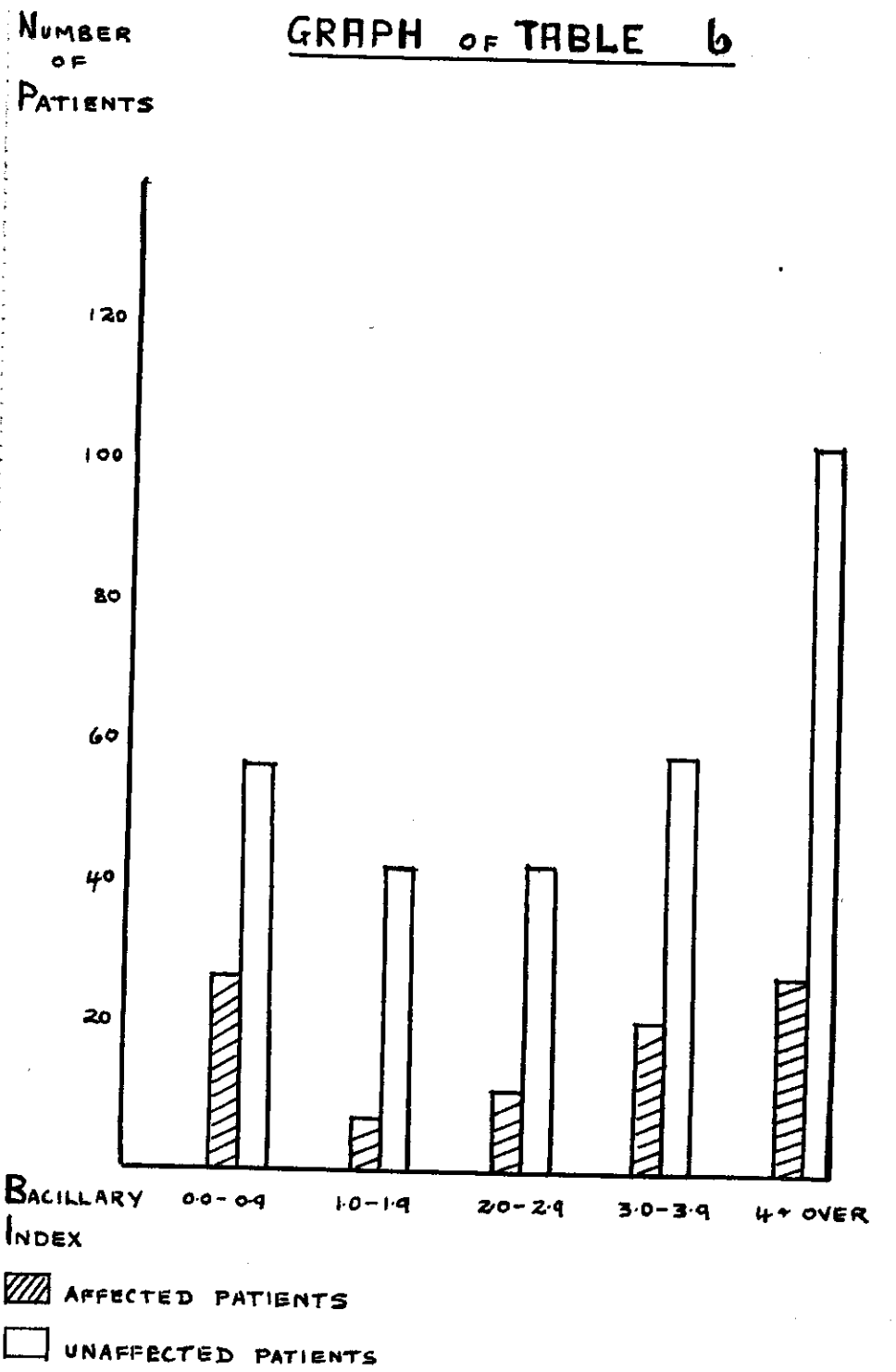


TABLE 7

THE DISTRIBUTION OF AFFECTED PATIENTS ACCORDING TO THE BACILLARY INDEX READING AT TIME OF DIAGNOSIS OF AN ACTIVE LESION

For significance of Bacillary Index, see page 157.

TABLE 7

BACILLARY INDEX	AFFECTED PATIENTS
0.0	13
0.0 - 0.9	3
1.0 - 1.9	2
2.0 - 2.9	5
3.0 - 3.9	5
4.0 and over	6
TOTAL	34

GRAPH OF TABLE 7

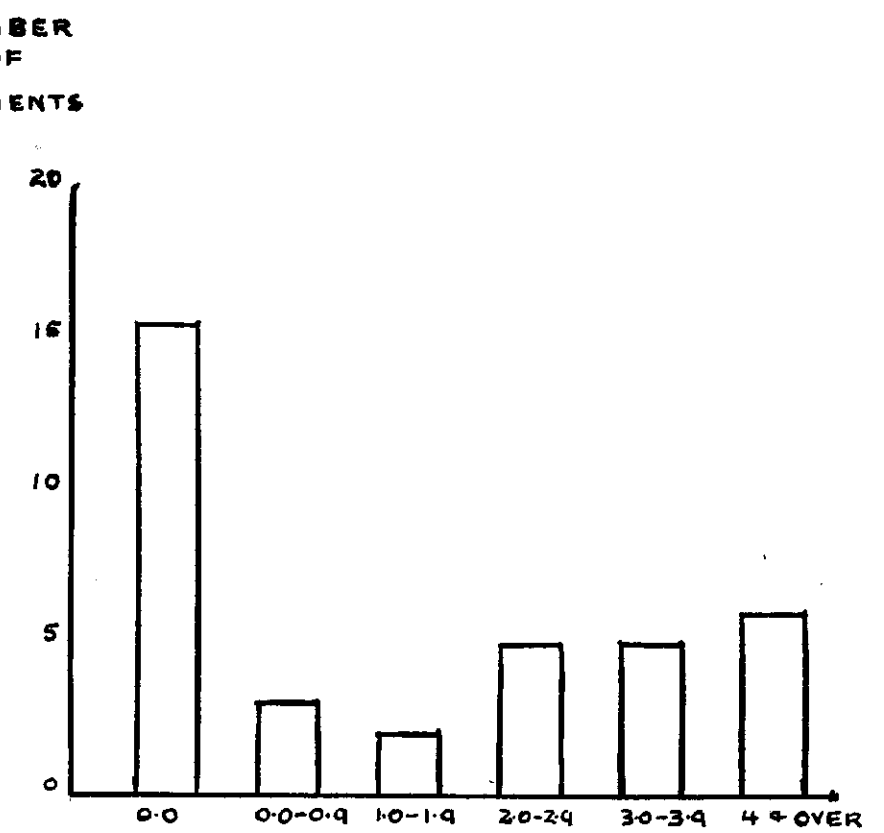


TABLE 8

SENSORY INVOLVEMENT OF THE LOWER LIMBS OF THE PATIENTS IN THE SELECTED GROUP AT TIME OF ADMISSION

TABLE 8

	SOLE OF FOOT	DORSUM OF FOOT	KNEE TO ANKLE	BELOW KNEE
THE LOWER LIMB AFFECTED BY A TARSAL BONE LESION				
No obvious anaesthesia	30	25	25	19
Patchy anaesthesia	10	13	44	50
Total anaesthesia	72	74	43	43
Number of legs				112
BOTH LOWER LIMBS OF AFFECTED PATIENTS				
No obvious anaesthesia	70	55	49	40
Patchy anaesthesia	11	22	70	80
Total anaesthesia	106	110	67	67
Number of legs				187 (1 amputation)
LOWER LIMBS OF UNAFFECTED PATIENTS				
No obvious anaesthesia	426	321	273	227
Patchy anaesthesia	12	31	126	271
Total anaesthesia	174	260	213	114
Number of legs				612

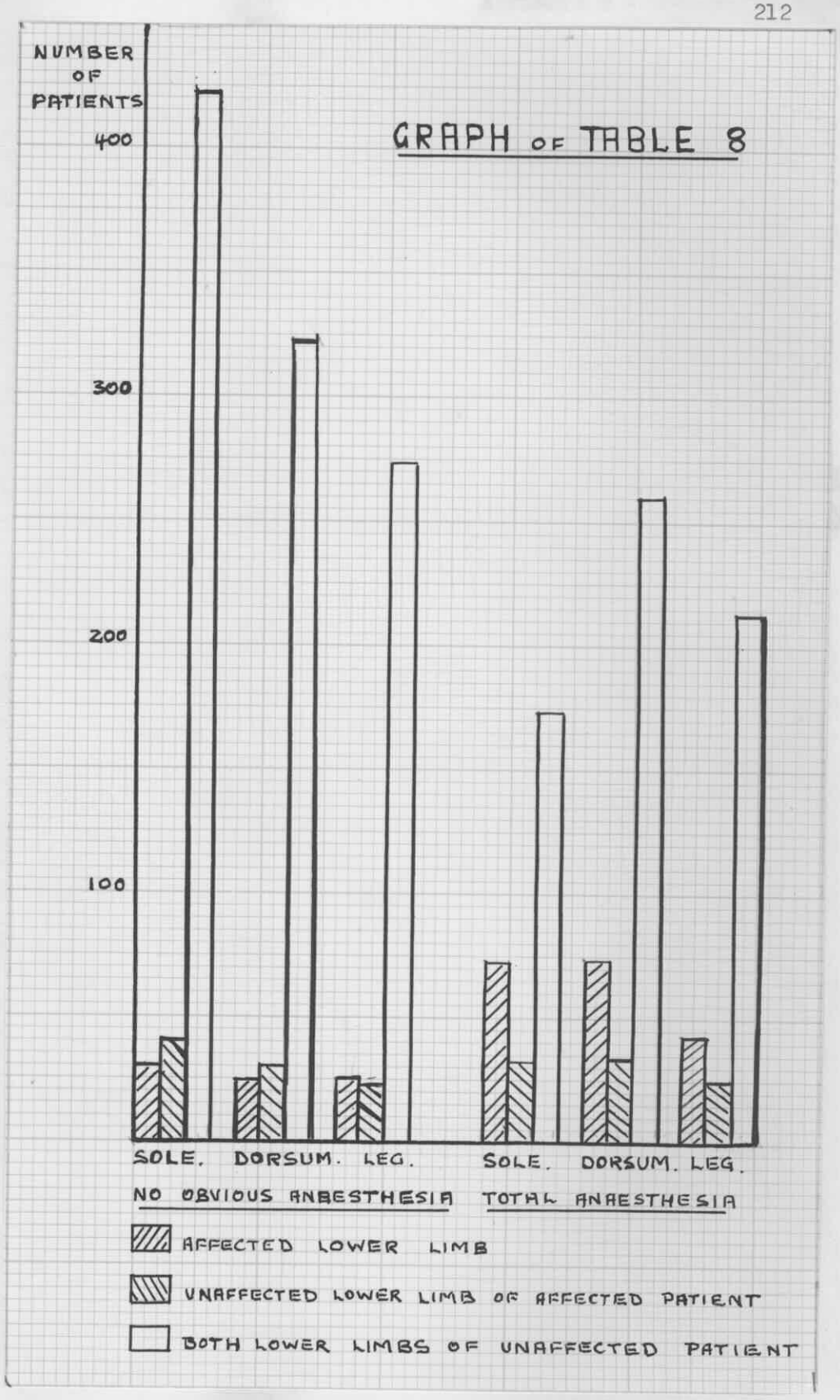


TABLE 9

PREVALENCE OF MUSCLE WEAKNESS ON ADMISSION AS SHOWN BY ABILITY TO DORSIFLEX THE FOOT

TABLE 9

	NORMAL FUNCTION	WEAK DORSI-FLEXION	ABSENT DORSI-FLEXION	TOTAL
The lower limb affected by a tarsal bone lesion	64	9	39	112
Both lower limbs of affected patients	122	14	51	187
Both lower limbs of unaffected patients	554	1	57	612
TOTAL	676	15	108	799 legs

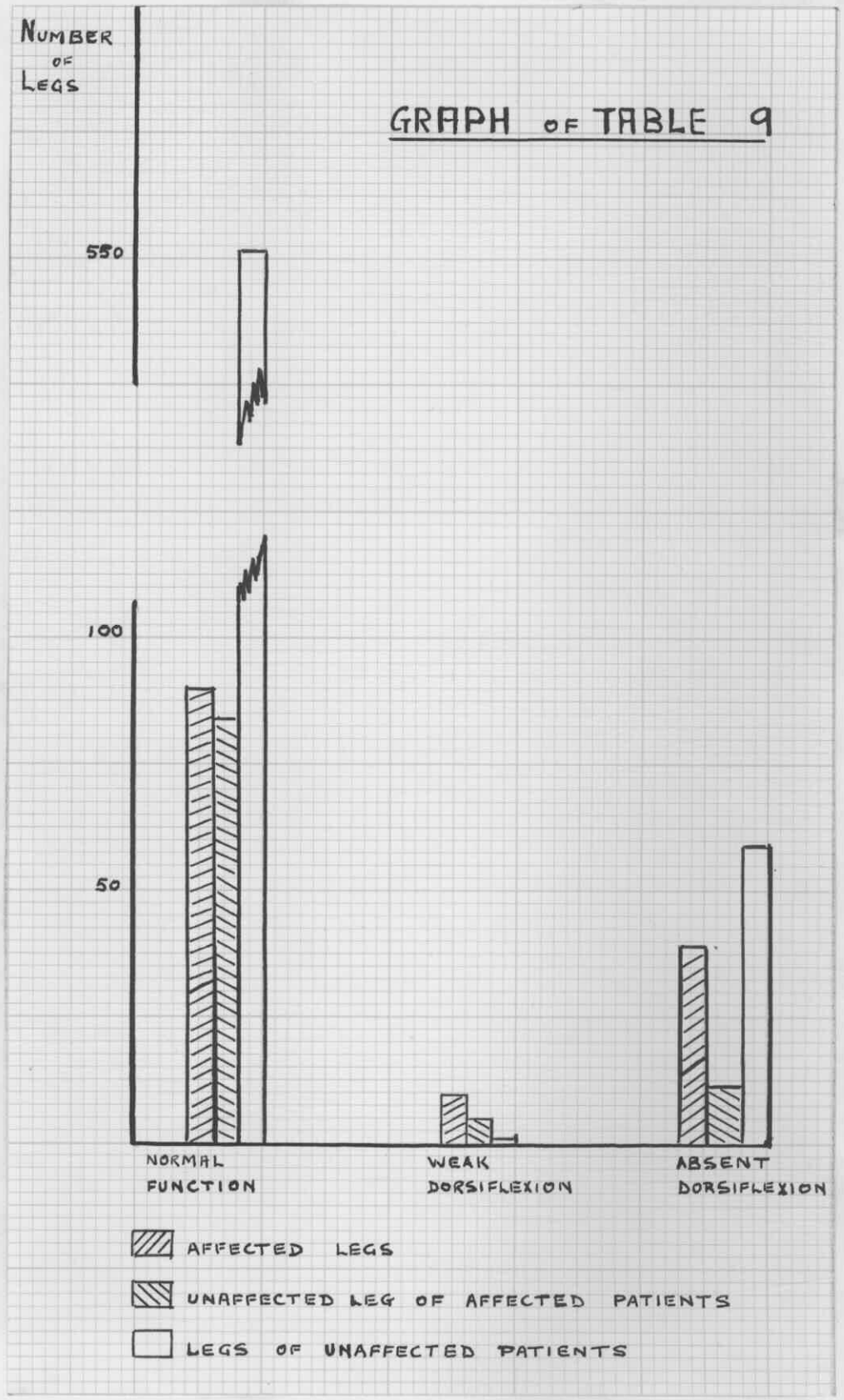
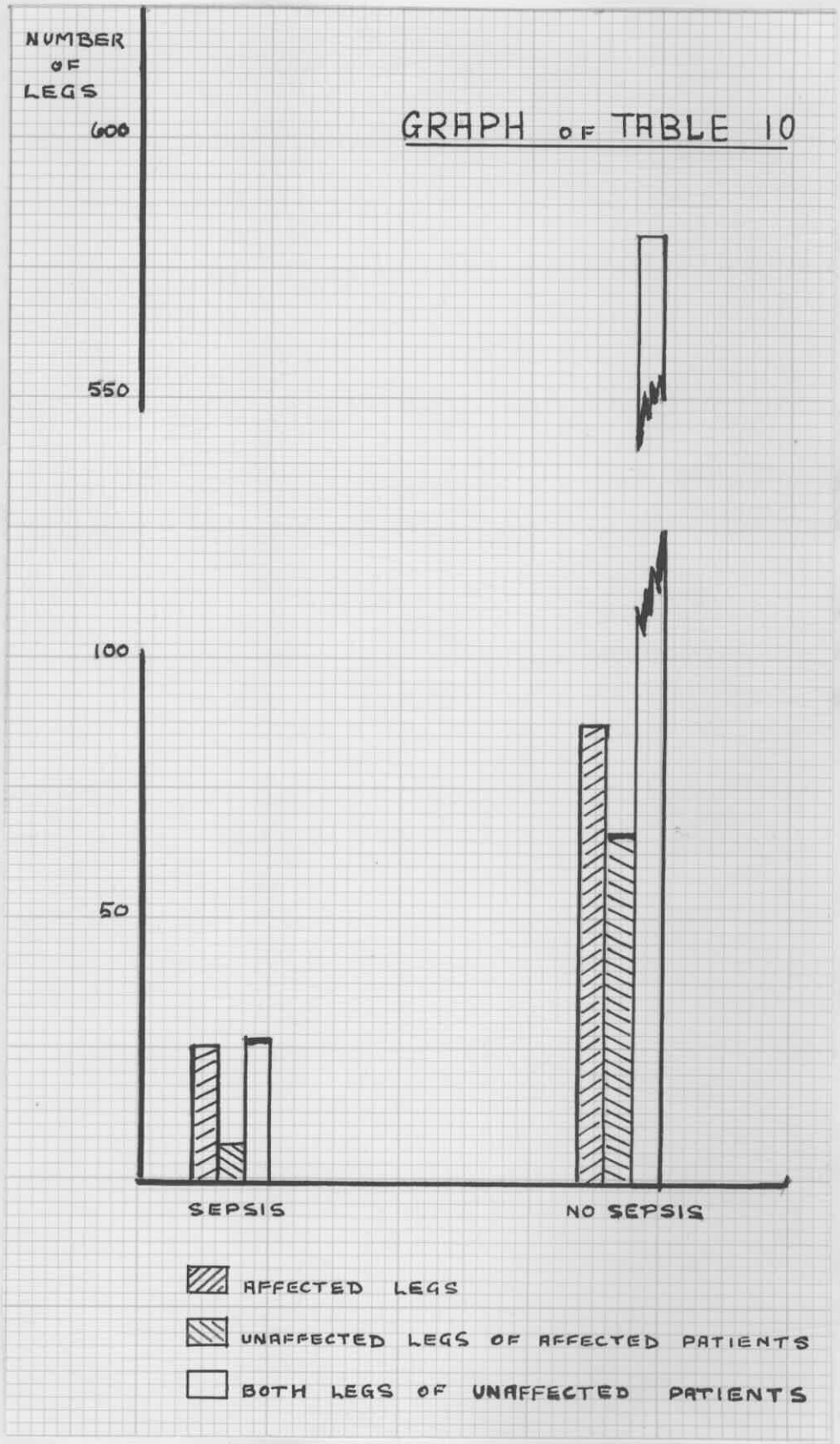


TABLE 10

PREVALENCE OF ULCERATION OR SEPSIS AT TIME OF DETECTION OF A TARSAL BONE LESION

TABLE 10

	WITH ULCERATION OR SEPSIS	WITHOUT ULCERATION OR SEPSIS	TOTAL NUMBER OF LEGS
The lower limb affected by a tarsal bone lesion	25	87	112
Both lower limbs of affected patient	32	155	187
Lower limbs of unaffected patient	29	583	612
TOTAL	61	738	799



GRAPH OF TABLE 10

TABLE 11A

PATIENTS ADMITTED WITH ACTIVE BONE LESIONS BUT WITHOUT OBVIOUS SEPSIS OF THE AFFECTED AREA

Patient No. 1723 had received twenty years of irregular antileprotic drug therapy. The other patients had not received significant amounts of antileprotic drugs before admission.

Patient's Number, Sex, Age, Type, B.I. on admission	Neural involvement of legs Ulceration	Other neural involvement	Bone involved, Side affected	Relevant history	Therapy	Residual deformity and/or disability	Comments
No. 1650 M/20 BL type B.I. 4.0	Left leg: - Stocking anaesthesia below knee, foot-drop for four years; ulcer on fifth metatarsal head with chronic sinus	Nerves large and tender, but no other deformity	Initially the left talus only - a minimal lesion was present JUN 64. Later all tarsal bones and the metatarsal bases were involved (APR 69)	MAR 64 chronic sinus of left fifth toe - healed by AUG. Bone lesion progressed slowly till DEC 65 when it was recognised but patient absconded before therapy was instituted	Therapy instituted on readmission in APR 69. A walking plaster cast was applied and immobilisation maintained for nine months	Limited ankle movement, due to collapse of the talus and a flattened arch. Tendency to "boat shaped" foot	A difficult patient. He refused therapy 1966-69. He has now lost the left forefoot due to sepsis in 1968
No. 1657 F/15 BL type B.I. 3.0	Bilateral patchy anaesthesia below groin. No muscular weakness or ulceration of feet	Slight bilateral ulnar nerve enlargement and muscle weakness	Left navicular. There appeared to be a chip off the dorsal surface	Nil available	A walking plaster cast for six months	No residual disability. Slight deformity of navicular	After the cast was removed she damaged the medial cuneiform (see Appendix H, Table 15)
No. 1680 M/31 BB type B.I. 1.2	Right leg: - Stocking anaesthesia below the knee, foot-drop for two years and ulceration of sole	Many nerves tender on admission; multiple deformities present	Right navicular fracture which showed some healing	Treated for a fractured ankle for three months before admission. Had lepra reaction on admission	A walking plaster cast for six months	Very little disability. Slight loss of arch	May have been a primary lesion or the result of inadequate immobilisation of a fracture
No. 1705 M/45 BL type B.I. 5.0	Right leg: - Patchy anaesthesia below calf. Foot-drop with claw toes and ulcers on fifth metatarsal head	Many peripheral nerves were large and tender	Posterior lip of right talus, possibly a fracture	Nil relevant	A walking plaster cast for four months	Slight collapse of body of talus with resultant tendency to varus of calcaneum	Constant problem with forefoot, eventual loss of fourth and fifth toes due to chronic ulceration aggravated by inversion
No. 1723 M/63 LL type B.I. 5.0	Bilateral stocking anaesthesia below knees. No muscular weakness. No ulceration	No obvious motor nerve lesions but much anaesthesia	Bilateral periosteal irregularities of the lower end of tibia and the tendo Achillis insertion	Chronic reaction - twenty years. Oedematous ankles and cardiac insufficiency	Expectant only, as early lesions and patient not very active	No real deformity or disability	Settled on regular therapy

TABLE 11B

PATIENTS ADMITTED WITH ACTIVE BONE LESIONS BUT WITHOUT OBVIOUS SEPSIS OF THE AFFECTED AREA

These patients had not received significant amounts of antileprotic drugs before admission.

Patient's Number, Sex, Age, Type, B.I. on admission	Neural involvement of legs Ulceration	Other neural involvement	Bone involved, Side affected	Relevant history	Therapy	Residual deformity and/or disability	Comments
No. 1727 M/13 BB type B.I. 2.3	Right leg: - Stocking anaesthesia below the knee. Foot-drop for one year, no foot ulcers	Peripheral nerves large but not tender	Head, neck and body of the right talus showed slow change in shape	Swelling of the ankle was present two weeks before admission	Nil, as not recognised in the first radiographs	Ankle instability due to altered shape of the talus was reduced after foot-drop correction was performed	A slow alteration in the talus shape occurred, probably influenced by the muscle imbalance
No. 1754 M/21 BL type B.I. 4.2	Right leg: - Very slight anaesthesia of forefoot. No muscular weakness or ulceration	All nerves were tender on admission. Later he had acute neuritis and paralysis	Right talus head only, already healing on admission	A local acute reaction of the dorsum of the right foot was present eight months preadmission with resultant anaesthesia	Expectant only	Minimal bone deformity, no disability	Further episodes of neuritis and reaction occurred but no further bone lesions
No. 1975 F/57 BB type B.I. 0.0	Right leg: - Stocking anaesthesia below knee, foot-drop present, no ulceration	No other nerve lesions	Mild activity of right navicular, cuneiforms and cuboid. Probably a long standing lesion. Collapse of the arch had occurred	None known	A wedge osteotomy was performed to reshape the foot. Then a walking plaster cast was used for eleven months	Very little functional disability after a tibialis posterior transfer was performed to correct the foot-drop	Lesion was probably slowly progressive. It appeared to heal completely after surgery
No. 2007 M/62 LL type B .I. 4.0	Left leg: - Stocking anaesthesia below knee, muscular weakness present	Nerves not tender or enlarged, multiple deformities present	Cuboid, cuneiforms and navicular of left foot	Very poor general condition. Anaemia. Amoebiasis. Tuberculosis. Syphilis	Walking plaster	Some expected, but: -	Reasonable healing was progressing when he died within three months of admission

TABLE 12

PATIENTS ADMITTED WITH ACTIVE BONE LESIONS ASSOCIATED WITH MARKED SEPSIS OF THE AFFECTED AREA

All patients had stocking anaesthesia below the knee of the affected foot.

All patients, except No. 1996 and No. 2011, had a foot drop, and all except No. 2011 had claw toes.

Patient's Number, Sex, Age, Type, B.I. on admission	Affected foot and degree of sepsis	Neural involvement	Bone lesion	Treatment	Comments
No. 1611 F/52 BB type B.I. 0.8	Left foot, large ulcer over cuboid, osteomyelitis	Multiple deformities of hands and feet for many years	All tarsal bones involved. Osteomyelitis of cuboid, marked "boat shaped" foot deformity	Antibiotics and rest for eight months then wedge osteotomy and ten months plaster immobilisation. Then foot-drop correction was performed	Functional ulcer free foot. Wears normal shoes
No. 1822 M/42 BB type B.I. 0.0	Right foot gross ulceration with loss of forefoot. Left foot loss of forefoot by ulceration	Multiple lesions, nerves infiltrated, of long-standing	Bilateral involvement of all tarsal bones. Osteomyelitis, with loss of both forefeet	Antibiotics and rest only - patient confined to bed because of gross deformities and poor physical condition	Sepsis was controlled when the patient died eight months after admission from gastric haemorrhage and cholangitis
No. 1831 F/58 BB type B.I. 0.3	Large ulcer on instep of right foot. Osteomyelitis.	Multiple deformities of hands and feet	All tarsal bones showed disintegration with osteomyelitis of the cuboid area	Antibiotics and rest, and then walking plaster immobilisation. Foot-drop correction had to be followed by modifed triple arthrodesis because of varus of calcaneum	Fair result - heel is now satisfactory, but ulceration over cuboid is recurrent and general condition does not allow further surgery
No. 1866 M/31 BT type B.I. 0.0	Large ulcer over cuboid area, right foot	Multiple deformities hands, feet and face	All tarsal bones were involved. There was osteomyelitis of cuboid area. Marked "boat shaped" foot deformity	Antibiotics and then walking plaster immobilisation for six months, then wedge osteotomy and immobilisation for a further twelve months	Good functional foot, normal shoes
No. 1965 M/56 BT type B.I. 0.0	Gross gangrene lateral side of left forefoot	Right hand and foot only affected by paralysis and anaesthesia	Osteomyelitis and gangrene of most of forefoot, loss of cuboid and lateral ray of the foot	Antibiotics and then immobilisation for a total of eight months then double wedge osteotomy of tarsals. Immobilisation for a further nine months, then foot-drop correction performed	Small functional foot, required prosthesis for stability
No. 1981 M/32 BT type B.I. 0.0	Deep ulcer of cuboid area, left foot	Right hand and left foot only affected	All the tarsal bones were involved. Osteomyelitis probably only in the cuboid. Foot inverted, and markedly "boat shaped"	Antibiotics and rest. Then immobilisation for eight months. Double wedge osteotomy and immobilisation for further nine months followed by foot-drop correction	Functional, though deformed foot, wears normal shoe
No. 1996 M/45 BB type B.I. 2.7	Multiple sinuses, whole of left foot affected by osteomyelitis	Active neuritis of arms, anaesthesia of legs	Gross osteomyelitis of all foot bones, some sequestra removed later	Antibiotics, rest, local treatment and then immobilisation for eighteen months allowed healing with eversion of heel	Patient walking in normal shoes but residual ulceration under metatarsal heads
No. 2011 M/27 BT type B.I. 0.0	One large ulcer on sole over calcaneum and cuboid area, right foot	Multiple deformities of hands; retained deep sensation of the affected foot	Osteomyelitis of calcaneum and cuboid present. Talus and navicular also involved and collapsed. Resultant foot was "boat shaped" with a deformed calcaneum and almost absent cuboid	Two months antibiotics and then nine months of immobilisation. Double wedge osteotomy and further twelve months in walking plaster cast	Functional ulcer free foot, wears normal shoes

TABLE 13

PATIENTS WHO DEVELOPED TARSAL BONE LESIONS WHILST UNDER TREATMENT

a) IN ASSOCIATION WITH LEPRA REACTION

In Patient No. 1618 there was stocking anaesthesia below mid-calf.

In Patient No. 1762 the skin lesions only were anaesthetic.

In the other affected feet there was also stocking anaesthesia below the knee.

Patient No. 1762 complained of a painful ankle. The others complained of swollen feet.

Patient's Number, Sex, Age, Type, B.I. on admission	Reaction	Affected foot and B.I. at time of lesion	Muscular involvement of affected foot	Radiographic findings	Other factors	Therapy	Deformity, Disability	Duration of radiographic follow-up
No. 1618 M/54 LL type B.I. 3.3	Chronic mild ENL, slight swelling of foot, pain in feet, repeated minor sepsis	Right B.I. 3.3	Right claw toes, but normal dorsiflexion	16 MAR 64 showed a lesion of first metatarsal base and cuneiform but not diagnosed until 22 SEP 64	Chronic sepsis of ankle and toes present at time lesion diagnosed	Walking plaster for six months, then intermittent use of crutches for recurrent ulcers	No deformity of tarsal area. No disability	Six years
No. 1640 M/34 BB type B.I. 4.0	Chronic mild lepra reaction, with several acute severe episodes of neuritis. Self-medication with Prednisolone in mid-1968	Left B.I. 2.7	Claw toes and weak dorsiflexion	SEP 68 compressed navicular	Surgery to toes in MAR 68 was followed by sepsis and prolonged immobilisation. Patient self-medicated with Prednisolone at that time	Walking plaster for seven months	No deformity of talus area, but loss of toes. Disability due to scars on toes	Two and a half years
No. 1762 M/56 BT type B.I. 1.8	Localised lepra reaction of skin lesions and large tender nerves	Right B.I. 1.5	Some weakness of dorsiflexion which later recovered	31 AUG 65 displaced tendo Achillis due to fractured calcaneum	The patient was hospitalised for three months prior to diagnosis of the lesion. He had marked osteoporosis	Splint to maintain dorsiflexion. No walking. Hospitalised for pulmonary tuberculosis	Some heel deformity. No disability	Twelve months
No. 1897 M/25 BL type B.I. 3.5	Chronic swelling of the feet. Acute neuritis with left foot-drop in MAY 67	Left B.I. 3.3	Left foot weakness two years. Increased paresis MAY 67	22 NOV 67 compression fracture of calcaneum	Sepsis on admission, none for nine months prior to diagnosis of bone lesion	Walking plaster for five months	Some compression of calcaneum but no real disability	Three months then discharged
No. 1913 F/61 BT type B.I. 0.0	Chronic severe lepra reaction. Given Prednisolone for three weeks from 25 JUL 67 for reaction	Right B.I. 0.0	Foot-drop present for eighteen months	18 NOV 67 all tarsals involved	Osteoporosis may have been due to senility and/or Prednisolone	Walking plaster for twelve months then foot-drop correction 21 JAN 69	Some arch collapse. No real disability	Nine months after surgery
		Left B.I. 0.0	Foot-drop for eight months	18 NOV 67 navicular compressed	"	Walking plaster for six months then foot-drop correction 20 JUN 68	Minimal deformity. No disability	Fifteen months after surgery
No. 1916 M/35 BL type B.I. 3.5	Chronic severe reaction associated with marked swelling of the feet	Right B.I. 2.8	nil	15 NOV 68 chip of talus neck	Sepsis of toes on admission. No sepsis for twelve months prior to lesion	Walking plaster for five months	No deformity. No disability	Two years
		Right B.I. 2.8	nil	NOV 69 healing fracture of right first proximal phalanx diagnosed four months after removal of walking plaster for talus lesion	No sepsis for twenty-one months prior to diagnosis of lesion	Bandage support	No deformity. No disability	Eighteen months

TABLE 14

PATIENTS WHO DEVELOPED TARSAL BONE LESIONS WHILST UNDER TREATMENT

b) DETECTED FOLLOWING SURGERY

All patients had borderline type leprosy and were B.I. negative at the time of surgery.

All patients had tibialis posterior transfer for foot-drop correction.

Patient No. 1656 had claw toe correction (Girdlestone procedure) at the same time (each foot).

Patient's Number, Sex, Age	Duration of foot-drop	Anaesthesia	Other lesions	Side affected and date of surgery	Patient's complaint	Radiograph date and findings	Therapy and results	Comments
No. 1625 M/27	1 year	Stocking type below knee	No ulceration of feet, multiple deformities of all limbs due to paralysis	7 JUL 64 right foot	14 AUG 64 swelling (Haematoma)	SEP 64 avulsion of tendo Achillis	Walking plaster for six weeks. No disability	Limited activity for six months pre-surgery due to post-operative infection of left foot
No. 1646 M/29	11 years	Stocking type	Ulcers on right fifth metatarsal head and multiple deformities of all limbs	26 JUN 64 left foot	Nil (a)	JAN 65 navicular disintegration	Walking plaster for five months	Biopsy taken of lesion. For report, see page 201. Limited activity for previous four months due to osteomyelitis of right foot
					(b)	27 AUG 65 first metatarsal and cuneiform disintegration	Walking plaster for ten weeks. No resultant disability. See also Table 15.	
No. 1656 M/14	Right 15 months	Patchy on calf, total on foot	No ulcers on feet. Bilateral foot-drop. Left hand paralysis	22 SEP 64 right foot also claw toe correction performed	NOV 64 pain in right ankle	15 DEC 64 talus head flattening, talo-navicular instability	Walking plaster for four months, then talo-navicular arthrodesis. Slight flattening of arch	Young boy with mobile feet. Removal of tibialis posterior support may have reduced talo-navicular stability and increased abnormal stresses on developing bones and ligaments. Both still stable in 1971
	Left 18 months	Patchy on calf, total on foot		27 OCT 64 left foot, also claw toe correction performed	JAN 65 intermittent swelling of left foot	24 MAR 65 calcaneo-cuboid joint irregularity	Walking plaster for four months. No resultant disability	
No. 1743 F/51	6 years	Stocking type below groin	Ulcers on both feet, shortened left toes. Multiple hand and face deformities	1 MAR 66 left foot	Swelling of left ankle	4 MAY 66 avulsion of tendo Achillis insertion	No walking. Plaster back slab support for three months and physiotherapy. No disability	Limited walking for six months pre-surgery due to infected right foot
No. 1882 M/34	3 years	Total of foot, patchy on calf	No ulcers on feet. Increased nerve involvement in preceding year due to acute lepra reaction and acute neuritis	21 NOV 67 right foot	JAN-FEB 68 infected haematoma of right fifth metatarsal head with ulceration. Swelling of the ankle in APR 68	SEP 68 healed avulsion of tendo Achillis insertion	No disability. No specific treatment. Patient was on limited activity and on a rest splint for the infected toe	This lesion may have occurred before surgery, at the time patient was given Prednisolone for lepra reaction

TABLE 15

PATIENTS WHO DEVELOPED TARSAL BONE LESIONS WHILST UNDER TREATMENT

c) AFTER USE OF A WALKING PLASTER

Patient's Number, Sex, Age, Type, B.I. on admission	Foot-drop	Clinical findings on admission	Side affected and bone lesion. Reason for original plaster	Lesion seen	Lesion	Therapy and result	Comments
No. 1646 M/29 BB type B.I. 0.0 History page 186	Complete drop of eleven years duration and claw toes. Surgically corrected 26 JUN 64	Stocking anaesthesia below mid-thigh. Ulceration right fifth metatarsal head. Multiple nerve lesions of face, hands and feet	Walking plaster for left navicular disintegration. Duration five months. Removed 7 JUL 65	27 AUG 65	First metatarsal base and cuneiform	A walking plaster for ten weeks. No deformity	Healed well. Remained stable for two years
No. 1657 F/15 LL type B.I. 3.0	Weak dorsiflexion	Patchy anaesthesia of both legs below groins. No ulceration. Right ulnar paralysis	Disintegration of left talus was present on admission. A walking plaster cast for six months achieved healing	24 JAN 65 six weeks after removal of plaster cast	Medial cuneiform	Walking plaster for six months and slow supervised resumption of normal activities. No disability	Very active teenager. She probably resumed activity too rapidly after removal of the plaster
No. 1921 M/32 BL type B.I. 4.7 See history page 191	Right foot-drop for one year	Stocking anaesthesia of both legs below the groins. No ulceration. Progressive paralysis and anaesthesia during lepra reaction	Right foot walking plaster cast for 4½ - 5 months for an ulcer of:				Very unco-operative patient always walking when not closely supervised
			(a) Right fifth metatarsal head with osteomyelitis. Plaster removed 20 FEB 68	28 MAR 68	Medial cuneiform and first metatarsal base	A walking plaster for four months, then two months bed rest	
			(b) For above lesion, limited walking allowed on 27 AUG 68. Full walking NOV 68	12 NOV 68	Navicular compression	A walking plaster for eight months	
			(c) For above lesion, plaster removed 22 JUL 69	16 SEP 69	Compression of calcaneum	Nil, as already healing when detected. No real disability, though some residual deformity	Foot has remained stable for eighteen months since last bone lesion

TABLE 16

PATIENTS WHO DEVELOPED TARSAL BONE LESIONS WHILST UNDER TREATMENT

d) OTHER PATIENTS WHO DEVELOPED LESIONS

No patient had a foot ulcer.

Patient No. 1766 showed patchy anaesthesia of the affected leg but normal muscle power.

Other patients showed stocking anaesthesia and a foot-drop.

Patient's number, Sex, Age, Type, B.I. on admission	Duration of Disease	B.I. when lesion found. Affected side	Condition of affected leg	Other neural lesions	Patient's complaint	Date of radiograph, Lesion	Therapy and result	Comments
No. 1699 M/40 BL type B.I. 3.8 M.I. 8%	8 years	B.I. 3.2 right	Right foot-drop, stocking anaesthesia. No ulceration	Bilateral ulnar and right median paralysis	Swollen painful foot on 15 MAR 65. Stated he "twisted it"	1 APR 65 compressed talus body and impacted fracture of calcaneum	A walking plaster for seven months. Removed 2 NOV 65 for trial walking but patient absconded. No obvious deformity then	Appearance of lesion suggested talus lesion of several months duration at least - and in retrospect it was obviously not healed. On readmission in 1970 there was total ankle disorganisation (See Fig. No. 3, page 69A)
No. 1766 F/74 LL type B.I. 5.0 M.I. 10%	6 years	B.I. 4.0 left	No muscle weakness. Patchy anaesthesia. No ulceration	Nil detected	Intermittent pain and swelling of feet for months. Radiograph of MAY 65 was normal, except for osteoporosis	27 DEC 66 impacted fracture of calcaneum, but healing well when first seen	No treatment as healing well. No deformity	Elderly lady, not very active. Had osteoporosis on admission. No history of known trauma
No. 1772 M/42 BL type B.I. 3.3	2½ years	B.I. 2.0 left	Foot-drop present. Stocking anaesthesia. No ulceration	Generalised large peripheral nerves. No other paralysis	Nil	Found on routine examination. Radiograph in MAY 67 showed compression of neck of talus, not present in JUN 65 films	Nil, as already healed. No disability	History does not suggest time of lesion
No. 1831 F/57 BB type B.I. 0.3	20 years	B.I. 0.0 left	Foot-drop present. Stocking anaesthesia. No ulceration of affected foot	Bilateral foot-drop, right claw hand. Facial weakness	Pain and swelling of left leg and ankle	12 Mar 69 fractured left calcaneum with slight displacement	A walking plaster for four months. No real disability	Had been in bed for ulcer of right foot OCT 68 to JAN 69, usually inactive, osteoporosis present

Appendix I

CALCIFICATION OF THE INTEROSSEOUS MEMBRANE OF THE LEG

Calcification of the interosseous membrane between the tibia and fibula is seen in some patients under treatment for leprosy. As it has been seen in patients with chronic foot sepsis and lepra reaction, and in association with tarsal bone lesions, investigation into its incidence was carried out.

MATERIAL AND METHODS

Ninety-nine patients under treatment at Hay Ling Chau, selected by consecutive admission numbers as far as available, were radiographed to show the interosseous membrane of the leg. These radiographs were examined by an independent person (Dr. S. D. Sturton) and classified according to the degree of interosseous membrane calcification present. Periosteal proliferation along the tibia and fibula, adjacent to the interosseous membrane, was considered to be a mild degree of calcification.

RESULTS

A total of twenty-nine legs (14% of those examined) showed definite interosseous calcification. Patients frequently showed calcification of one leg only. The lesions were most marked in the middle and lower third of the leg, rarely involving the whole width of the membrane but usually appearing as an irregular calcification extending outward from the bones into the membrane, see Fig. 44, page 225A. The lowest centimetre of the membrane and the articular surfaces of the tibia and fibula were rarely involved unless there was associated involvement of the talus.

Of these twenty-nine legs, fifteen showed interosseous calcification associated with a tarsal bone lesion; ten of these also showing definite evidence of chronic sepsis of the associated foot, resulting in loss of soft tissue and sometimes loss of bone as well. Five legs,

in which no scars of past sepsis were obvious at the time of examination, showed interosseous calcification in association with a tarsal bone lesion.

Thirteen legs showed a tarsal bone lesion without interosseous calcification. Three of these showed definite evidence of chronic ulceration or sepsis, and ten showed no obvious sign of past sepsis.

Fourteen legs showed definite interosseous calcification without evidence of a tarsal bone lesion. None of these showed marked tissue loss as a result of sepsis, but some did show signs of previous infections and ulceration, and ten were sufferers of chronic lepra reaction.

It was noted that the most severe calcification was present in patients whose feet had been severely involved by sepsis.

Twenty feet showed definite tissue loss as a result of chronic sepsis, and half of these feet showed both interosseous calcification and a tarsal bone lesion. In this series of ninety-nine patients the common factors in the patients with interosseous membrane calcification were chronic lepra reaction and soft tissue loss, indicating previous chronic sepsis. Both of these conditions are associated with hyperaemia and periostitis. Once the lepra reaction is controlled or the infection has subsided, there is little change in the degree of calcification of the membrane, even though the leprosy is still active.

Previously, in 1961, the investigator checked a group of ten patients showing interosseous calcification of the leg. All the patients investigated were in good general health and had no complaints relative to the abnormal calcification. Some of them did still have chronic infection of the feet. Radiographs of the forearms showed no evidence of abnormal calcification of those inter-

osseous membranes. The V.D.R.L. test was negative. Wasserman tests were not done as these frequently give false positive results in active lepromatous leprosy patients. Serum calcium and urinary calcium levels were within normal limits. It was deduced that the isolated calcification of the interosseous membrane of one leg was not due to a general metabolic disturbance, but to some local factor.

In these ten patients chronic sepsis was a common factor, though the original calcification had been detected on routine radiography and they were not selected because of the sepsis, which had in some cases been healed for a number of years. The calcification was frequently only present in one leg, but no careful examination was made for tarsal bone lesions. Some of these patients did not have any lateral radiography of their feet so it is impossible to say in retrospect that they did not have tarsal bone lesions at that time. It was assumed that the interosseous calcification was a result of the chronic foot sepsis and this impression has been confirmed over the years. Follow-up of some of the patients with interosseous calcification has shown that this lesion is not progressive. It has been included in the Appendix of this dissertation as its presence suggests that the patient has had lesions of the feet which should be investigated.

DISCUSSION

Interosseous membrane calcification was not specifically mentioned by Paterson (1961) when he described the other bone lesions of leprosy, although he listed leprous periostitis of the tibia as occurring in the Hong Kong patients. It is, however, known that very few patients in Hong Kong, prior to 1960 when he visited Hay Ling Chau to review our material, had been radiographically examined in a position to display the whole membrane. In retrospect small amounts of calcification can be seen in some of the films available at that time but it was not commented upon.

In "Studies in the Medieval Diagnosis of Leprosy in Denmark", by Andersen, 1969, he mentions that in examining the skeletons from the graveyards, Møller-Christensen found periosteal deposits on the tibia and fibula and supposed them to be evidence of specific disturbances of nerves and vessels. Andersen himself observed these deposits in the legs of patients at the Purulia Leprosy Home in India, and states that all patients who had these deposits showed signs of plantar ulceration or the scars of previous ulcers. It is understood that these deposits are the same as the lesions this investigator calls interosseous calcification.

CONCLUSION

Interosseous membrane calcification is a separate entity from tarsal bone disintegration. It may, however, occur in association with tarsal bone disintegration or with chronic infection of the affected foot, or in association with chronic lepra reaction.

APPLICATION

The presence of interosseous membrane calcification is of clinical significance in reconstructive surgery when a decision must be made regarding the route of tendon transfer for foot-drop correction (see page 150). Its presence should warn the clinician to examine the feet carefully.

Fig. 44 Calcification of the interosseous membrane, associated with marked sepsis of the foot in Patient No. 1154.

Appendix J

REPRINTS OF ARTICLES ON RELATED SUBJECTS BY THE INVESTIGATOR

THE CORRECTION OF FOOT DROP IN LEPROSY 227

A FOOT DROP SPRING 228

FACETS OF LEPROSY OF ORTHOPAEDIC INTEREST 229

PLASTER CASTS 230

TARSAL BONE DISINTEGRATION IN LEPROSY 231

THE CORRECTION OF FOOT DROP IN LEPROSY*

A. GRACE WARREN, HONG KONG

From the Hay Ling Chau Leprosarium, Hong Kong

Foot drop from paralysis of the muscles of the anterior and lateral compartments of the leg is common in leprosy. Often associated with anaesthesia, it is a challenge to the surgeon who seeks to rehabilitate the patient. The patient, unaware of unusual stresses on his foot because of the neurotrophic lesions, may allow a deforming force to continue until an ulcer is formed; hence it is of the utmost importance that any deformity of the foot in a patient with leprosy should be so completely corrected that no other deformity or disability will occur as a result of surgery (Fritschi and Brand 1957).

Many methods of correcting the functional problems associated with a foot drop have been tried with the object of providing a foot that is stable and socially acceptable. Surgical means provide the most permanent correction and give the best cosmetic results (Ober 1933). Transfer of the tibialis posterior tendon to the front of the foot through the interosseous membrane has proved satisfactory, and in most patients is devoid of serious complications (Gunn and Molesworth 1957, Brand 1964). Nevertheless, in the Chinese patients at the Hay Ling Chau Leprosarium a regularly recurring list of complications has followed the routine use of this method, when followed up for five to six years.

COMPLICATIONS OF TIBIALIS POSTERIOR TRANSFER

The commonest complications encountered are as follows. 1) Inadequate elevation of the foot because of undue lengthening of the tendon. 2) Inversion or eversion of the foot related to the placement of the new insertion and the pull on any remaining muscles; 3) talo-navicular collapse (Andersen 1964, Paterson and Job 1964, Harris and Brand 1966), which may occur without operation and is not of necessity associated with any obvious infection. In reconstructive surgery the first indication occurs some two to four weeks after the removal of the plaster, when the patient starts to walk again. The foot becomes warm and slightly swollen but there is usually no discomfort. A radiograph may show osteoporosis only at this time but if walking is allowed freely and radiography is repeated in six weeks the early signs of bone breakdown will often be seen. Early detection and immobilisation for up to six months in a walking plaster can lead to complete recovery without deformity; neglect may lead to increasing bone destruction and to loss of the arch with lateral deviation of the forefoot. This condition of talo-navicular collapse is believed to be caused by unnoticed injury to the anaesthetic foot. However, it has been assumed that surgical intervention with the tarsal bones must increase the tendency to bone breakdown after surgery. The new technique described has therefore avoided tendon-to-bone union and has utilised a tendon-to-tendon technique, which requires only three weeks in plaster and therefore there is less osteoporosis. 4) Instability of the talo-navicular joint which may be associated with some breakdown of the cuboid bone or the calcaneus and results in lateral deviation on the forefoot. This is assumed to be connected with removal of the support of the talo-navicular joint that is provided by the tibialis posterior tendon (Last 1963). 5) Dropped toes caused by laxity of the tendons of the paralysed extensor digitorum longus when the ankle once again assumes the right angled position and the pull of the tendons of the flexor digitorum longus shortened by the dropped foot position. This may cause the patient to walk on the nails of the toes

* Based on a paper presented at the Combined Orthopaedic Meeting in Singapore in August 1966.

TABLE I

COMPARISON OF THE OLD AND NEW METHODS IN CASES 1 AND 2

Case number	Side	Sex	Age in years	Method and date of operation	Movement	Angles before operation in degrees
1	Right	Male	70	Old October 1964	Passive dorsiflexion	85
					Active dorsiflexion	—
					At rest	115
					Active plantar-flexion	120
					Range	5
	Left			New May 1965	Passive dorsiflexion	85
					Active dorsiflexion	—
					At rest	115
					Active plantar-flexion	128
					Range	13
2	Left	Male	31	Old June 1964	Passive dorsiflexion	75
					Active dorsiflexion	—
					At rest	125
					Active plantar-flexion	140
					Range	15
	Right			New May 1965	Passive dorsiflexion	88
					Active dorsiflexion	—
					At rest	110
					Active plantar-flexion	120
					Range	10

which are turned under. 6) Clawed toes, which may result if too strong a pull is given to the toe extensors by suturing these to the mobile section of the active tendino-muscular transfer when there are no muscles to provide flexion of the metatarso-phalangeal joints (Andersen 1961).

PRINCIPLES OF TREATMENT

With these points in mind a new method of foot drop correction has been evolved with the object of: 1) maintaining the muscular support of the talo-navicular joint; 2) providing tendon-to-tendon union to reduce immobilisation to three weeks; 3) leaving the bones of the foot intact; 4) reducing the tendency to dropped toes and clawed toes.

The principle of the modified operation is to leave the distal five centimetres of the tendon of tibialis posterior *in situ* and to attach this stump to the flexor digitorum longus as the two tendons lie together above the medial malleolus. This means that the flexor digitorum longus will pull on the insertion of the tibialis posterior and so maintain some muscular support of the arch of the foot and the talo-navicular joint. Its power of pull on the toe tips is thus reduced, so that dropped toes are not so likely to occur.

The proximal part of the tendon of tibialis posterior is still available for transfer with the muscle belly, but will not of course reach to the dorsum of the foot. To meet this deficiency further variations of technique have been evolved. The tibialis posterior is attached to the tendon of tibialis anterior at the junction of the middle and lowest thirds of the leg and the tibialis anterior tendon therefore prolongs the tendon of tibialis posterior so that it reaches the medial side of the dorsum of the foot. The lateral side of the foot is provided for by a peroneal slip that is woven through the tibialis anterior to form an inverted Y distal to the ankle when the tibialis posterior muscle contracts and dorsiflexes the foot—hence there is no active pull on the toe extensors if they are stitched to this peroneal slip and the effect is that of a tenodesis.

Good results cannot be expected if the ankle will not passively move through an adequate range. It is desirable that after surgery the patient should be able actively to lift the toes off

TABLE I—*continued*

COMPARISON OF THE OLD AND NEW METHODS IN CASES 1 AND 2

Angles after operation in degrees						Comment
3 weeks	7 weeks	4 months	7 months	12 months	20 months	
70	70	65	70	70	73	
80	78	70	78	86	85	
90	85	75	85	90	90	
92	90	95	95	95	100	
12	12	15	17	11	15	Walks very well now. Feet previously very unstable and needed crutches to walk at all
68	70	70	70	70	75	
75	78	82	82	85	90	
80	90	90	90	95	95	
85	95	95	95	100	105	
10	17	13	13	15	15	
75	75	70	70	70	70	
83	83	84	96	90	94	
90	95	95	110	105	105	
95	100	104	115	110	110	Right foot excellent result.
13	17	20	19	20	16	Left foot revised
60	65	65	60	65	70	in December 1966
73	80	80	80	80	80	at patient's request.
76	88	90	90	90	90	Now walks very well
80	90	95	95	100	100	
7	10	15	15	20	20	

the ground when he is standing with his heel on the ground. A range of 10 degrees of movement on each side of the midpoint of ankle movement is probably ideal but not necessary in practice. To achieve this it is necessary to be able to dorsiflex the ankle passively to 75 degrees with the knee straight; if this is not possible it is advisable to lengthen the calcaneal tendon.

Measurements—All measurements are taken with the knee straight and the leg parallel with the ground, with increasing values as the foot passes from dorsiflexion (extension) to plantarflexion (flexion). By this method all changes follow a regular transition and one does not need to state if the movement is flexion or extension in each case.

TECHNIQUE

Through an incision on the medial side of the leg the tendons of tibialis posterior and flexor digitorum longus are displayed and the calcaneal tendon is lengthened if necessary. The tendon of tibialis posterior is divided obliquely so that the distal slip can be sutured into the lateral side of the flexor digitorum longus. There should be no tension on the suture when the ankle is dorsiflexed to 80 degrees and the toes are straight.

The anterior surface of the interosseous membrane is displayed at the level of the junction of the middle and lowest thirds of the tibia, through a curved incision based medially, and the membrane is incised along the tibia. The proximal part of the tibialis posterior tendon is pulled through to the front of the leg. Sufficient fibres of the tibialis anterior muscle are separated from its tendon to allow the tendon of the tibialis posterior to be passed through the hole so made so that the two tendons lie side by side and burying of unsatisfied tendon ends is simplified.

A slip is passed from the peroneus tertius or from the insertion of peroneus brevis to the tibialis anterior tendon at the ankle, and sutured to it so that the tibialis anterior tendon is pulled slightly to the lateral side of the mid-point of the ankle (Fig. 1). This slip can be peroneus brevis, peroneus tertius if it is well defined, or a tendon graft. This provides lateral lift and stability. If the foot is very mobile extra slips may be used passing from the tibialis anterior

at the ankle to the periosteum of the fifth metatarsal neck. The further distal the fixation, the less likely is inversion or instability. With the hallux straight the tendon of extensor hallucis longus is sutured without tension to the tibialis anterior tendon, distal to its junction with the peroneal slip. To the peroneal slip the toe extensors are sutured without tension when the toes are straight. This will help to prevent dropped toes.

With the knee held at about 40 degrees of flexion, and the ankle dorsiflexed to 75 degrees, the tendons of posterior and tibialis anterior are then sutured together with the greatest tension possible, and all unsatisfied ends are buried. This position is held during final skin closure and until the plaster-of-Paris cast is applied to maintain position.

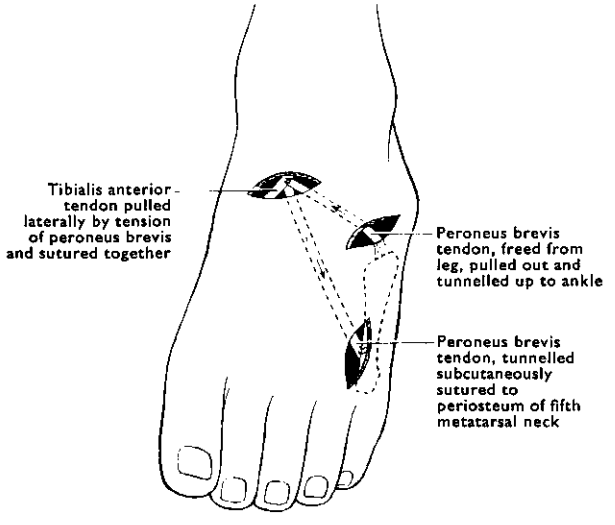


FIG. 1

Diagram to show passage of peroneus brevis from its intact insertion, subcutaneously to the tibialis anterior tendon, and then forward to the fifth metatarsal neck.

The plaster is removed after three weeks, with no weight-bearing, and is converted into a backslab to be worn all the time when the patient is not exercising. Active re-education is started as soon as the plaster is removed, and active control is usually achieved within the first week, when partial weight-bearing is allowed until the patient has learnt to use the transferred muscle against gravity, at which time walking is started. The patient is expected to be walking within three weeks of removal of the plaster—that is six weeks after operation. The backslab is discarded by day once walking is well established but is used at night for a further three or four weeks.

SELECTION OF CASES

The first two patients had bilateral foot drop. They had previously had a corrective operation for one foot, using a previous technique. The second foot was done with the new method. In both patients the peroneus brevis was used for the lateral slip, and in both the toes were still fairly mobile. No complications occurred and both patients preferred the " new method " without knowing it was technically different. In Table I the range of movement of both pairs of feet as recorded over a period of twenty months is set out for comparison. It will be noticed that slackening continues to occur over a long period of time for both methods, but that by the end of four months the foot is fairly stable.

These two patients were out of plaster and evaluated before further cases were done by similar methods. Because the first two feet thus treated functioned well it was decided to do

TABLE II

ANGLES OF MOVEMENT IN RECENT CASES

Case number	Sex	Age in years	Follow-up in months	Angles in degrees					Comment
				Passive dorsi-flexion	Active dorsi-flexion	At rest	Active plantar-flexion	Range	
3	Male	25	13	75	88	95	102	14	Very satisfactory. Walks well
4	Male	33	11	73	85	95	100	15	Good walk after tibialis anterior shortened
5	Male	42	10	75	95	100	110	15	Good function. Consideration must be given to loss of great toe
6	Male	46	9	75	88	95	100	12	Very satisfactory with almost normal gait
7	Female	52	9	75	85	90	97	12	Some inversion after initial surgery corrected by tightening of the lateral slip
8	Female	57	9	70	85	97	117	32	Excellent range and a fair gait. Other foot arthrodesed
9	Male	36	9	70	88	95	105	17	Very slow to re-educate. Now walks very well
10	Female	30	9	70	80	90	97	17	Short foot from loss of toes. Walks quite well
11	Male	36	8	70	80	90	105	25	Some lateral instability corrected when peroneus brevis was used to replace peroneus tertius. Now walks well
12	Male	25	6	73	85	95	98	13	Foot very stable. Patient satisfied though only small range of movement
13	Male	33	6	68	80	86	93	13	Foot rather stiff but patient satisfied and walks quite well

all subsequent operations by this method. A total of fifteen more cases were done in the following ten months. The movements found in those with a follow-up of six months or more are given in Table II with comments on function.

DISCUSSION

It is considered that the range of movement achieved by this transfer adequately provides for the function of the foot. Minor variations in the method of stabilisation of the lateral side of the foot were tried but the peroneus brevis has usually proved the most adequate lateral stabiliser.

One case only had increased warmth and slight swelling about two weeks after removal of the plaster. This lasted for two days only and did not recur when walking was resumed. Radiographs taken at least three months after operation in each case have shown no signs of bone breakdown.

The tendency to drop toes is difficult to assess. No patient so far has complained of the development of dropped or clawed toes but a continued watch must be kept for these complications.

There has been no sign of talo-navicular instability in any case. This usually manifests itself within six months of operation. Only time will tell whether the reactivation of the distal

stump of the tibialis posterior tendon does in fact reduce deformity and if the use of the flexor digitorum longus for activation of this stump reduces the incidence of dropped toes and collapse of the long arch of the foot.

It has been found that this procedure is technically easier than other methods and takes less time.

SUMMARY

1. The complications following standard tendon transfer to provide active correction of drop foot in Chinese patients with leprosy are reviewed.

2. An alternative method of foot drop correction is described in which reactivation of the remaining distal stump of the tibialis posterior tendon is provided to assist in maintaining the stability of the arch of the foot and to help to prevent dropped toes.

3. A review of thirteen patients is given. The indications are that this method is functionally as good as other methods. So far it has shown none of the complications usual in Chinese patients.

I would like to acknowledge the assistance of our patients and colleagues, without whose help it would have been impossible to develop this alternative method. I would like to thank Professor P. W. Brand and Mr G. M. Bedbrook for their encouragement and Mr H. Jackson Burrows for his assistance and advice in the preparation of the manuscript.

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A Foot Drop Spring*

A. GRACE WARREN

Medical Superintendent, Hay Ling Chau Leprosarium, Hong Kong

INTRODUCTION

Active splints have long been accepted as a basic therapeutic measure for the prevention of deformity due to paralysis and in the treatment of paresis. However, in leprosy their use has frequently been neglected. The fitting of an active splint to the hand is difficult, compared with the ease of fitting an efficient active splint that will provide a functional support to allow normal walking in a foot with paralysis of the anterior and or lateral leg muscles (foot drop). Many types of splint have been devised over the years and have proved effective, but some may not be acceptable for the treatment of large groups of persons in poor financial conditions.

The requirements of an efficient active splint for foot drop are that it should be: (1) effective, (2) comfortable, (3) adaptable for making with local materials by patients themselves, (4) cheap, (5) easy to apply, even with deformed hands, and (6) that it can be used with socially acceptable clothes and shoes and can conform to local customs. Over the years the staff at Hay Ling Chau have evolved a foot drop spring that we consider fulfils these conditions in Hong Kong.

DESCRIPTION

A simple cuff is made to encircle the leg immediately below the knee and to fasten in front of the leg. Attached to this by 2 arms is a "Y" piece, to the lower end of which is fastened a metal spring which is hooked on to the shoe; the degree of lift required is adjusted by the length of the "Y" piece, which is provided with a number of holes for use with a pair of buckles on the upper cuff, thus providing a choice of length.

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MATERIAL

(a) Any locally available material can be used. Leather is readily usable, but webbing, canvas, or any similar strong material is equally satisfactory.

(b) The upper cuff, if made of leather, is padded with sponge rubber to assist grip on the leg and reduce localized pressure. A cuff made of other material may not need padding.

(c) Buckles are used for fastening, but eyelets and laces could be used, or "Velcro Magic tape" for patients with bad hands, or "Gripper" fasteners or other patent fastenings as available.

(d) Metal springs are reliable and wear well, but strong elastic may be used or strips of heavy rubber from the inner tubes of lorry tyres will provide adequate stretch.

(e) A dressmaker's hook stitched to the shoe (or sock) will provide the point of attachment, or the spring can be hooked through shoe laces or on to a loop or ring stitched to the shoe.

In countries where shoes are usually removed at the door and left outside, it is possible to attach the spring to firm socks, or to provide a cuff to go round the forefoot at the level of the metatarsal-phalangeal joint so that the spring can be worn in the house. As the paralysed foot is usually associated with an anaesthetic sole it is best to advise these patients to wear some type of footwear at all times so as to prevent trauma.

PATTERN

The Hay Ling Chau design is as follows:

(1) A leather cuff with a sponge-rubber inner lining and furnished with a tongue and a strap to pass through the buckle in front of the leg carries 2 further buckles, one on each lateral aspect, angled forward to provide attachment

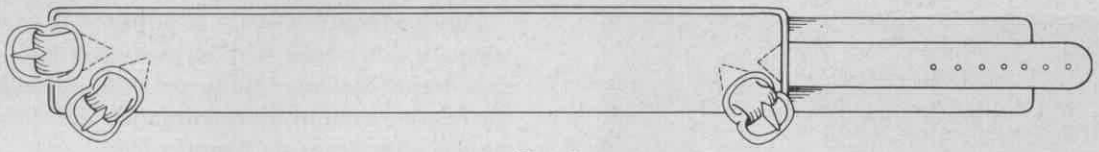

FIG. 1
Upper leg cuff showing tongue lining and placement of buckles.

for the "Y" piece (see Figs 1 and 3). *Note:* All buckles should be on the front of the leg to prevent them damaging the anaesthetic skin of the popliteal fossa when patients sit or squat. The cuff must, of course, be tight enough to hold up above the calf. Cuffs may be pre-made, but should allow for different calf sizes (lengths of 10 and 12 in. (25 and 30 cm) are commonest for adults in Hong Kong).

(2) In acute paralysis, where the lateral popliteal nerve is tender, it may not be possible to tighten this type of cuff adequately without causing pain in the nerve; also, in patients with marked wasting of the calf there will not be enough muscle bulk to hold up this cuff. In such case a double cuff is desirable. The upper cuff can be fastened firmly while the lower cuff is left looser. The latter is essential as it provides the correct site for the pull of the spring. If the spring is attached above the knee its tension will be lost when the knee is flexed; hence to be effective the spring must be attached below the knee. The side bars joining the 2 cuffs should be directly mid-lateral to prevent rubbing during movement of the knee (Fig. 3).

(3) The "Y" piece is also lined with leather for strength and has 4 to 6 punch holes on each upper end to allow adjustment of the length; it has also a metal bracket at the lower end for attachment of the spring (Fig. 2).

(4) The spring is about 5 in. (12.5 cm) in length, 6 or 7 mm in diam. and the 5 in. length should stretch to 6 or 7 in. (15 to 17 cm) when strong pull is applied. Weak springs will rapidly be distorted by the power of the gastrocnemius and soleus muscles. The lower end of the spring is easily bent out to form a ring for use on the hook, or to pass through a ring or lace on the shoe.

(5) The hook on the shoe should be situated over the head of the fourth metatarsal to provide adequate lateral lift in a full foot drop. This site may need to be adjusted if there is paresis in order to obtain a suitable balance and prevent inversion or eversion of the foot. The shoe need not be an expensive or orthopaedic one, provided it fits well and firmly so that it will not be pulled off by the spring (i.e. has a heel counter); it should also have a good sole to protect the anaesthetic foot that usually accompanies foot drop. The final appearance of a completed spring with double upper cuff is shown in Fig. 3. *Note:* For night use a cuff to encircle the foot at the level of the metatarsal-phalangeal joint can be made to attach to the spring and so minimize the chances of contracted Achilles tendon.

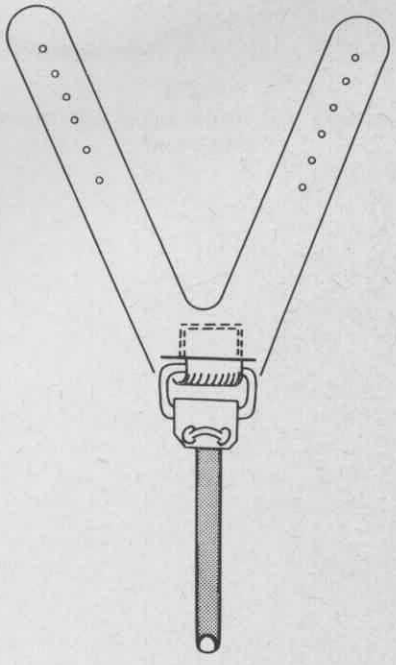

FIG. 2
The "Y" section showing metal attachment and spring.

FIG. 3
Complete spring with double upper cuff, showing shoe attachment.

These springs can easily be adjusted to ensure adequate lift of the foot to prevent a flapping gait during walking. Many patients appreciate the relative comfort of walking with one of these springs after years of "flapping feet".

SUMMARY

A description is given of a simple form of foot drop spring that is cheap and easy to make and has proved effective in controlling the paralyzed foot and preventing further deformity.

ACKNOWLEDGEMENTS

The design of this spring has gradually evolved over many years of co-operation by staff and patients. We merely present the version we are using today to help others to help themselves. Our thanks go to Miss K. Collett for the illustrations and to the Hong Kong Government physiotherapists for their encouragement and assistance in improving the design.

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Facets of Leprosy of Orthopædic Interest

GRACE WARREN
Hay Ling Chau Leprosarium, Hong Kong

Facets of Leprosy of Orthopædic Interest[1]

GRACE WARREN
Hay Ling Chau Leprosarium, Hong Kong

In many countries leprosy is regarded as a disease that the average medical practitioner is unlikely to see unless he travels. However, in this jet age, the long-accepted geographical distribution of disease is being upset by tourists, workers and other travellers, who bring home illness and pose problems of diagnosis that are unexpected. Although in leprosy the primary lesion is frequently one of the skin, a proportion of patients do not seek medical advice for a relatively minor skin lesion. They may, however, present with secondary complications involving the hands and feet.

PATHOLOGY

LEPROSY is often considered to be a dermatological disease. The skin lesions are characterized by granuloma, which may be well localized or may form extensive sheets with ill-defined margins. As the granuloma regresses the lesions heal, leaving characteristic scar tissue devoid of normal resilience and sensation and prone to injury. In some patients there are episodes of acute ulceration. The scar tissue resultant from both of these processes may lead to contractures and deformities that may require surgical correction to restore function. In the acute stages physiotherapy will do much to prevent or minimize such deformities.

Leprosy can also be considered as a disease of the peripheral nerves with cutaneous manifestations. The earliest lesion is an inflammatory cellular exudate in association with the fine nerve fibres. This may develop into an acute inflammatory process, typified by the presence of epithelioid cells and Langhans giant cells, and presenting clinically as an acute paralysis.

The affected nerve fibres eventually degenerate and may undergo necrolysis. In patients with less immunity, there may be a more chronic inflammatory process typified by a slowly increasing granuloma containing many lepra cells. The nerve sheaths and Schwann cells disintegrate, and a hyaline degeneration of the nerve progresses slowly to the stage where the nerve is firm and fibrosed, with impaired function. These patients may present with a chronic progressive neuritis which has continued for many years.

The paralysis and the anæsthesia resultant from this nerve damage are responsible for most of the deforming and disabling sequelæ that create the big problems in the rehabilitation of the leprosy patient. Even after reconstructive surgery to correct the deformities, the patient has to learn to live with the anæsthesia.

NEURITIS

Neuritis, acute or chronic, may be the presenting symptom. Involved nerves may be enlarged, tender or painful, but there is usually some paræsthesia or muscle weakness if careful testing is undertaken. Patients may even present requesting plastic surgery for apparently symptomless enlarged unsightly nerves such as the supra-orbital and supratrochlear. Alternatively, they may present requesting plastic surgery to restore the appearance of a wasted first interosseous space resultant on an ulnar neuritis.

The commonest sites of nerve involvement are the ulnar nerve at the elbow and the lateral popliteal nerve at the head of the fibula. The picture following involvement of the median nerve at the wrist may resemble the carpal tunnel syndrome. Recovery of some motor power can be expected in over 70% of patients with an acute paralysis who receive adequate early treatment. Recovery cannot be expected in patients who show a chronic slow loss of power characteristic of low-immunity leprosy.

[1] Read at a meeting of the Combined Orthopædic Associations, held in the Royal Australasian College of Surgeons in April, 1970.

DEVELOPMENT OF DEFORMITY

In the treatment of an acute foot-drop, a most important therapeutic measure is the provision of a toe-raising spring, as without this the patient may walk on the lateral border of an anæsthetic foot. Because of lack of pain, an ulcer may develop, which will be neglected, and secondary osteomyelitis may supervene. The orthopædic surgeon may be consulted about an inverted ulcerated foot with osteomyelitis, partial paralysis and possibly other secondary deformities. Routine orthopædic measures can be applied to provide a functional non-ulcerated foot. However, although soft tissue healing is not usually delayed in leprosy, it has been found that bone healing may be delayed, and the surgeon must be prepared to continue immobilization for considerably longer periods than are normally considered necessary with the non-anæsthetic foot. Healing of neuropathic joints will occur if immobilization is contiuued, but may require from six to 18 months.

SURGERY

It was the large number of leprosy patients with marked deformities that encouraged Mr Paul Brand, while working in India, to develop a number of surgical procedures. Although other methods may give good immediate results, his technique of lumbrical replacement is preferred by a large number of leprosy surgeons because of the lack of long-term complications. These complications frequently cause disabilities worse than the original deformity, when other procedures are used for leprosy patients. Many of these procedures and derivatives of them are also applicable to patients with deformities from other causes.

THE PROBLEM OF ANÆSTHESIA

However, it is not only reconstructive surgery that is needed to rehabilitate leprosy patients. Because of anæsthesia, even hands with normal function are prone to be misused, and work is being continued at present on methods of training patients to use their hands without damage. Research at the Carville Centre in Louisiana has produced gloves incorporating dyes of different colours which are released at various pressures, so that the patient learns to use the correct pressures of grip while at work to achieve a uniform colour stain. Variations of colour will indicate where he has exerted too much pressure.

In the foot anæsthesia is also a problem, as minor degrees of trauma are not noticed by the patient. Continued use of a foot may be enough to cause a minor chip fracture of the tarsal bones to develop into a neuropathic joint. Treatment may be delayed until gross instability has developed. Sometimes the patient may present early with heat and swelling of the affected foot, but no obvious deformity. Anæsthesia can usually be detected on the affected limb, but how many of us do check for sensation if presented with a suspected sprained ankle? Radiographic examination may not reveal any bone abnormality until four or six weeks after the time of injury, so that serial films are desirable if swelling persists. Even a gross bone lesion will heal if the limb is adequately immobilized, but great care must be taken to achieve a good functional position and to continue the immobilization until healing is complete.

TROPHIC ULCERATION

Research programmes at various leprosy centres have increased our understanding of the problems of anæsthetic feet and the reasons for trophic ulceration. Trophic ulceration occurs in areas that are submitted to pressures of sufficient intensity and duration to cause anoxia and death of tissue. The tendency to trophic ulceration is increased by muscle imbalance and lack of pain sensation.

Trophic ulceration can be prevented, but this requires:

(1) The proper care of neurotrophic skin, including replacement of water and oil and the removal of hard skin callosities so that they do not cause increased localized pressure. Patients can learn to undertake this themselves, and it should become an essential part of their programme.

(2) The provision of suitable footwear, with special attention to the resilience of the sole.

(3) Attention to the patient's gait. Short steps will help to prevent excessive local pressures.

(4) Surgical correction of muscle imbalance and reshaping of the deformed foot to spread pressures more evenly.

(5) Encouragement of the patient to pursue a suitable occupation to minimize the risks of trauma.

In conclusion, it is well to realize that by the time deformities develop, most leprosy patients are already non-contagious and can be treated in routine clinics alongside patients with other diseases. It is only a small proportion of patients who at any one time are contagious, and with modern antileprotic drugs even the worst of these can be rendered non-contagious in a few months.

Coordination of the treatment of the various aspects of this disease is highly desirable. If surgery is attempted during the acute phases of the disease, an undesirable reaction may be precipitated. It is necessary to keep in mind the progressive nature of the disease when planning surgery. A new paralysis can occur years after apparent quiescence, and this may negate a reconstructive procedure if the possibility has not been considered.

Leprosy is an endemic disease in many countries. In spite of the availability of effective drug treatment, statistics suggest that the numbers of sufferers are still increasing. More and more patients are being found in the English-speaking countries where once it was considered a rarity. Let us look for it in our own communities and we will find it. The lessons that we have learnt from leprosy can be applied to patients with other diseases, so they too may benefit from our experiences in leprosy.

The patient with leprosy has every right to early diagnosis, correct treatment, and recovery without obvious deformity or disability.

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Plaster Casts*

GRACE WARREN

Hay Ling Chau Leprosarium, Hong Kong

A step-by-step description is given of a method of applying a plaster of Paris walking cast that can be performed by one technician with minimal assistance.

Plaster of Paris walking casts are recommended for the treatment of foot ulcers in leprosy. They are also an essential part of the treatment of tarsal-bone disintegration in leprosy. To be effective, a cast must be strong, light, and well fitted so that it does not rub and cause an abrasion which, because of anaesthesia, may remain unnoticed until a deep ulcer has developed. Since a plaster applied to an oedematous leg or foot becomes loose as the swelling subsides, steps should be taken to reduce swelling before the walking plaster is applied.

Introduction

A simple method of applying a walking cast developed by the staff at Hay Ling Chau is here described in the hope that it may interest medical orderlies and other field workers. Alternative appliances for enabling the patient to walk are also available.

Method

1. POSITION

(a) The patient lies face down on the couch with the knee of the affected side held at 90° of flexion. This means that the leg is not being supported on the horizontal plane and so reduces the risk of isolated pressure spots being caused by the assistant inadvertently pressing on the wet plaster (Fig. 1).

(b) The ankle and foot are easily controlled. (1) The patient can hold the foot correctly himself, in a good position. (2) Finger-tip pressure on the toes will maintain an anatomically normal foot. (3) If the foot arch needs much moulding, a strip of cotton bandage may be passed across the arch and pulled by the assistant to exert downward pressure on the arch while the assistant pushes the forefoot plantarwise. The combined action will give maximum arch moulding while the plaster is moulded over the bandage (Fig. 1). (4) Where there is marked heel inversion because of contracting soft tissues, an assistant forcibly moulds the foot until sufficient plaster is applied to hold the foot in the desired position. (5) If the metatarsals or phalanges are damaged, it may be advisable to use adhesive plaster and/or small splints to straighten the toes, and thus enable healing to proceed in a position of function.

N.B.: In most leprosy patients, it is important to apply the plaster with the

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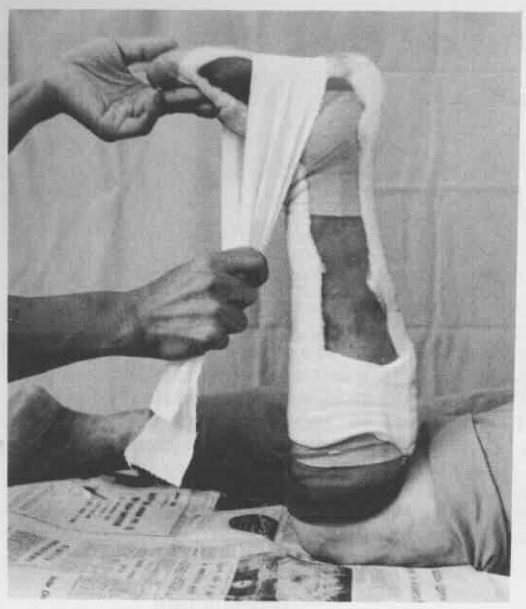

Fig. 1. The patient lies face downward on the plinth with the affected foot prepared for plastering. The position of the foot is held by use of a cotton bandage as described under Position (b) 3.

foot at right-angles to the leg, or even slightly dorsiflexed. If the plaster is applied while the foot is in plantar flexion, contracture of the tendo Achilles may occur, especially if the dorsiflexors of the foot are weak. Furthermore, the foot should be kept slightly everted (and not inverted), and the toes should be straight.

2. SKIN PROTECTION

Adequate protection must be provided at any points subject to pressure or friction, such as the front of the ankle and the upper end of the plaster, that is, near the knee. (a) Elastoplast has proved an excellent material, in that it moulds to the contours of the limb and does not move. A figure-of-eight bandage protecting the malleoli and the front of the ankle, combined with a cuff at the proximal limit of the plaster, is adequate. The upper limit of the plaster is determined by the patient's habits: if squatting is part of his way of life, the cuff needs to be lower than is usual. (b) Orthopaedic felt is satisfactory as a protector. (c) Rolls of cotton wool may be used, but care must be taken that the application provides a uniform layer that is not too thick and will not allow movement of the whole plaster. A combination of 2 or more of these "pressure point protectors" is probably best. (d) Old nylon stockings provide an excellent substitute for tubular stockingette; indeed they are in some ways preferable, since being already shaped they do not wrinkle in front of the ankle. Also the intact stocking toes keep dust and dirt from the ulcerated area.

At Hay Ling Chau, the routine for a plaster application is as follows:

(1) Elastoplast is applied around the ankle as a figure-of-eight. (2) A circular

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cuff is made of the same material at the level of the upper edge of the plaster. (3) A nylon stocking is rolled on and pulled up over the knee. (4) A layer of wool is applied along the sole and 3 in (7.5 cm) up the back of the leg to protect the heel (which is very vulnerable to pressure), carried over the tibial ridge, and over the Elastoplast at the level of upper edge of the plaster. (5) A second nylon stocking put on over the wool holds this firmly in position. (6) The position is checked and maintained–(as described in Section 1). (7) Plaster of Paris is then applied. (8) A cuff of nylon stocking and wool is turned down over the proximal edge of the wet plaster. This provides a nice cuff and helps to reduce rubbing at the proximal end of the plaster.

3. PLASTER APPLICATION

(1) A back slab of 8 to 10 layers of 6-in (15-cm)-wide plaster bandages is made, big enough to reach from the toe tips to the proximal (upper) edge. After being soaked, rubbed in and smoothed on a flat surface it is then applied carefully, moulded well on to the arch of the foot and round the heel and the tendo Achilles to ensure a good fit.

(2) A 3- or 4-in (7.5- or 10-cm) bandage is soaked and applied to the ankle and arch area to hold the foot and heel in position. If a cotton bandage is used to hold position (as described in 1, b, 3 above) this is not removed till the plaster is dry, but it can be worked around and later cut.

(3) A 6-in (15-cm) bandage is then soaked and applied proximally to give good support and fit around the calf, to form the upper limit of the plaster, and to complete the leg.

(4) When the plaster is dry enough to maintain the desired position of the foot, any moulding can be removed and the plaster completed by further 4- or 6-in bandages. It is important when applying the plaster to the toes that they be plantarflexed. Care must be taken that in applying the plaster the fifth toe is not extended at the metatarso-phalangeal joint.

(5) The proximal edge of the cast is smoothed off and the stocking or stockingette turned over to provide a smooth edge.

(6) The plaster must be completely dry before walking is allowed. This takes at least 24 h. If possible, the walking appliance should be applied only after the plaster is dry to ensure that the patient does not walk too soon.

4. WALKING APPLIANCE

(a) Bohler-type walking irons should be used for any patient with osteoporosis or tarsal-bone disintegration of the talus and/or ankle joint to minimize compression of the ankle region. A simple metal walking iron can be made by a local metal worker. Double cross bars on the long side pieces are desirable and these pieces should be malleable in order to fit the cast well (Fig. 2). The total weight of the iron must be considered in selecting a suitable metal or it may be so heavy that the patient cannot walk.

(b) Wooden or rubber rockers can be applied to the foot. Wooden ones can be made from a wooden packing case by a jobbing carpenter as shown on the diagram (Fig. 3), and shod with car tyre or made like the Karigiri shuffle board described by Ross (1962).

(c) For patients with bilateral foot lesions, or others for whom stability when standing may be a problem, a flat sole can be made on the plaster. Care must be taken that the plane of the sole is at 90° to the tibial line and that the foot is not

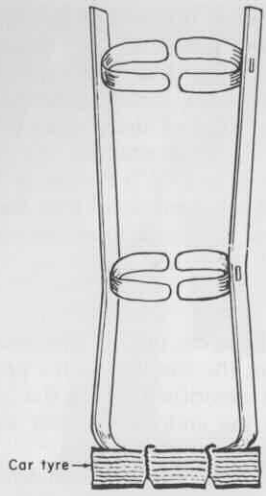

Fig. 2. A Bohler type walking iron made by a local metal worker and shod with car tyre.

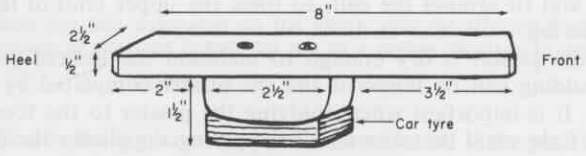

Fig. 3. A wooden rocker (showing dimensions) shod with car tyre.

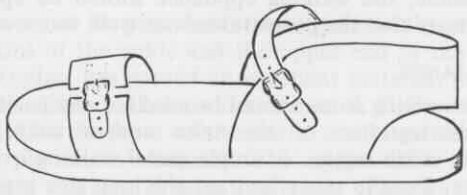

Fig. 4. Rubber-soled sandal with canvas straps and buckles to wear over a plaster cast.

forced into eversion or inversion. The sole is built up with plaster and smoothed off with a flat board. When it is dry the patient is provided with a sandal with a rubber or leather sole and adjustable straps that can fit over the plaster (Fig. 4). In wet weather a plastic bag can be used between the sandal and the plaster. These flat plasters have proved very useful and effective, especially in the elderly and less active patients and those in whom both feet need treatment at the same time (Fig. 5).

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Fig. 5. Sandalled plaster casts on a patient with bilateral foot lesions.

Comments

The use of walking plasters may cause osteoporosis, the degree of decalcification being proportional to the length of time the plaster is worn. Any foot that has been in plaster for 6 weeks or more should be observed carefully for several weeks after removal of the plaster. Unsupported and unrestricted ambulation should not be allowed at once, as the development of tarsal-bone disintegration following the use of a walking plaster is a very real possibility.

Early treatment of lesions due to tarsal-bone disintegration can result in complete recovery in an undeformed position. On the other hand, neglect may result in a grossly deformed foot and much disability.

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Lepr. Rev. (1971) 42, 60-68

Prosthetics in Leprosy*†

RONALD J. GARST

Associate Professor of Orthopaedics, Christian Medical College, Ludhiana, Punjab, India

This paper deals with the implications for amputation of the lower extremity of the patient with Hansen's disease, the preparation of the prosthesis, and the care of the stump. Particular attention is given to the making of a hard socket to prevent breakdown of the skin of the stump. Inexpensive methods of producing useful and acceptable prostheses are described.

Introduction

The management of amputees suffering from leprosy (Hansen's disease) offers a fascinating challenge to the prosthetist as well as to the orthopaedist who does the amputation and follows the care and fitting of the stump. The knowledge gained and practised regarding patients with Hansen's disease can be carried over into use for any amputee with sensory paralysis of the lower extremities. This is especially true in the case of paraplegia and spina bifida.

As an orthopaedist I will not try to tell the prosthetist how to make an artificial limb. However, there are a few points that we have observed which may be of help, or which may stimulate further study. One of the best monographs on the subject of orthopaedic and prosthetic appliances for leprosy is that written by J. A. E. Gleave, published by the United Nations Organization (Gleave, 1968). Most workers devise their own short cuts and simplifications, but all have not been published. By far the majority of leprosy patients in India who have undergone amputation go without the benefit of prostheses. Some of the reasons are the cost, the lack of prosthetic facilities available, unsatisfactory fitting followed by rejection of the limb, and the psychology of a begging community who get more alms by displaying grotesque deformities—the amputee leaves his prothesis at home when he goes out to beg. In this paper we will deal with our management of some of these difficulties.

Pathology

Any type of leprosy may result in loss of sensation, and the problem of fitting a prosthesis is the same. The loss of sensation creates a host of problems, such as

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The article on "Tarsal Bone Disintegration in Leprosy" has been accepted for publication in the November 1971 Volume of the Journal of Bone and Joint Surgery.

Only a galley proof is available as yet, and in this the illustrations lack clarity and are not included. However, the case histories and illustrations referred to in the article are included in this thesis as follows: -

Case No. 1	Patient No. 402	page 163
Case No. 2	Patient No. 546	page 167
Case No. 3	Patient No. 780	page 170
Case No. 4	Patient No. 771	page 168
Case No. 5	Patient No. 1996	page 194

LIVINGSTONE—BONE SURGERY—ARTICLE 53/48—1

TARSAL BONE DISINTEGRATION IN LEPROSY

GRACE WARREN, HONG KONG

From the Hay Ling Chau Leprosarium, Hong Kong

Many of the foot deformities that cause marked disability in patients with leprosy have resulted from tarsal bone disintegration. This condition has been observed by a number of workers in different countries, but is often accepted as a progressive disorder that does not respond to treatment.

These lesions have been only briefly dealt with in the literature. Some of the references were mentioned by Harris and Brand (1966), who stated that it was difficult to stop the rapid destruction of the foot once tarsal bone disintegration had begun. This is the general impression given by other workers, but a detailed study of the natural history, prevalence and effects of treatment of tarsal bone disintegration in Chinese patients with leprosy has shown that treatment is effective and that long-term results are satisfactory. The results of this study are summarised here.

MATERIAL

The radiographs and case histories of over 1,500 leprosy patients treated at the Hong Kong Leprosarium since 1959 were available for the study of tarsal bone disintegration. Of these patients, 400 admitted consecutively have been examined to provide prevalence rates and to indicate the persons at risk.

NATURAL HISTORY

The presenting signs are usually heat and swelling of the foot or ankle, without pain. Sepsis is not commonly present. Rarely a history of injury, such as a sprain, is elicited.

The first radiographic evidence is detectable four to six weeks after the presenting signs are observed. There may be a minimal fracture, either chip or crack (Paterson and Job 1963), rarefaction, fragmentation or partial collapse. These bone lesions progress sometimes with absorption or displacement of the fragments and collapse with consequent deformity of the foot.

The clinical signs of heat and swelling persist for several months, but as the process becomes more chronic the heat subsides and deformity becomes more obvious. Eventually the foot may show no heat and little or no swelling but the patient may have marked deformity, hypermobility or instability that is incompatible with normal function.

ILLUSTRATIVE CASE REPORTS

Case 1—A man aged thirty-eight was found to have slight irregularity of the talus in September 1957 (Fig. 1). The treatment recommended was relief of weight-bearing by use of crutches, but as his hands were badly deformed this was not practicable and limited walking continued. The lesion progressed over four months until a marked deformity of the ankle was present (Fig. 2). The foot was later amputated.

Case 2—A man aged twenty-five was found to have destruction of the talus and adjacent bones (Fig. 3). In spite of irregular immobilisation and periods of relief of weight-bearing the lesion progressed to complete disorganisation of the ankle and adjacent tarsal bones (Fig. 4).

Osteoclastic and osteoblastic activity have been seen to occur side by side in biopsy material, and it has been observed that healing tends to occur when the affected foot is completely immobilised so that no movement can occur between the bones or at the fracture sites.

Careful observation has shown that in some patients episodes of bone breakdown have alternated with periods of healing. This healing has occurred during immobilisation in walking plaster or enforced rest in bed, but has been interrupted by resumption of normal activity and breakdown has occurred again. Lesions have been seen in which severe deformity and instability have occurred within three years of a radiograph showing normal appearances. In other patients in spite of a degree of low grade activity continuing for many years the foot has remained basically functional while showing slowly increasing deformity. Radiographs covering a period of thirteen years are available for one patient, who retains a functional though deformed foot.

PREVALENCE

For the determination of prevalence rates all types of tarsal bone lesion are included (Table I).

The lesions appeared to be more common in patients with unstable dimorphous leprosy than in patients with other forms of leprosy. This may be due to the high incidence of lepra reaction and nerve involvement in these patients.

It was observed that of the forty-four patients in the selected group who had walking plasters, 11 per cent developed tarsal bone disintegration on removal of the plaster.

A study of these 400 patients has indicated that the "patients at risk" are those with conditions that predispose to osteoporosis. Some of these conditions have been implicated by other workers (Table II).

TABLE I

PREVALENCE OF TARSAL BONE LESIONS IN PATIENTS SUFFERING FROM LEPROSY

Total number of patients	400
Patients showing tarsal bone lesions	94 (23·5 per cent)
Patients showing unilateral foot involvement	76 (19 per cent)
Patients showing bilateral foot involvement	18 (4·5 per cent)
Total number of affected feet	112 (14 per cent)
Total number of tarsal bone lesions seen	119
Number of lesions active on admission of patient	19
Number of lesions that developed whilst patient was under treatment	22

TABLE II

CONDITIONS PREDISPOSING TO OSTEOPOROSIS

Hyperaemia of the feet caused by	1) lepra reaction
	2) sepsis (Harris and Brand 1966) (Karat and colleagues 1968)
Prolonged limitation of walking causing disuse atrophy and osteoporosis (Price 1964) caused by	1) lepra reaction (Karat and colleagues 1968)
	2) ulceration of the feet treated by complete bed rest
	3) surgery to the foot
	4) intercurrent illness
Corticosteroid medication for	1) lepra reaction
	2) intercurrent illness
Plaster for treatment of	ulceration of the foot and other conditions (Price 1964) (Harris and Brand 1966) (Lennox 1965)

The osteoporosis so caused is sufficient to increase the risk of a stress fracture from microtrauma (Paterson 1961). Tarsal bone disintegration has on a number of occasions been seen to follow attempted arthrodesis of the ankle or fracture of the ankle or foot when immobilisation has been discontinued before healing was complete.

ETIOLOGY

The radiographic evidence supports the view that tarsal bone disintegration is the result of microtrauma (Harris and Brand 1966), possibly in the shape of stress fractures (Karat, Karat and Foster 1968) which are neglected because of the diminution of pain perception (Paterson and Job 1963). The incidence of microtrauma and stress fracture is probably increased by the osteoporotic condition of the feet of the patients at risk (Table II). Disintegration of tarsal bones is often regarded as a neurotrophic lesion. This is supported by clinically detectable anaesthesia in 79 per cent of the affected legs in the selected group although lesions have occurred in patients who have no clinically detectable nerve deficit.

TREATMENT

Healing of these bone lesions will occur if the foot is adequately immobilised for long enough. This is reported by Harris and Brand (1966) who also mentioned that disintegration progressed during the use of calipers and other weight-relieving appliances.

ILLUSTRATIVE CASE REPORTS

Case 3—A man with navicular disintegration (Fig. 5) was treated by immobilisation of foot and calf in a plaster-of-Paris walking cast for six months. Complete healing of the lesion occurred and the foot remained stable for ten years (Fig. 6).

Case 4—A girl aged sixteen had a small chip fracture of the head of the talus (Fig. 7). Irregular periods of treatment were instituted over two years: there were periods of complete immobilisation on several occasions during which bone healing occurred; at other times, when weight-relieving calipers and crutches were used, further disintegration and collapse was observed. Healing eventually resulted but after further destruction with collapse of the head of the talus and loss of the longitudinal arch of the foot (Fig. 8).

It is important that a well planned plaster be applied to prevent the development of further collapse of the osteoporotic bones. In those lesions in which hypermobility of the foot exists it may be possible to mould the foot into a better functional shape while the plaster is being applied. The writer does not recommend operation for early lesions or when the foot can be moulded into a functional position. Lesions will heal without operation and this is encouraged when possible.

In patients with healed or partially healed lesions but grossly deformed feet that cannot be moulded into a functional shape, it may be advisable to improve the shape by operation. Osteotomies or bone grafts can be used as necessary (Lennox 1965). Healing does occur but may take twelve to eighteen months before it is complete. Adequate immobilisation must be maintained till the lesion is fully healed.

In the early minimal lesion healing may be complete in four to six months. Most moderately severe lesions require about nine months of immobilisation and surgical cases at least nine months.

Disintegration rapidly begins again if the lesion is not fully healed when walking is resumed. Hence it is essential that radiographic bone healing be achieved and after that a period of trial walking be undertaken. The patient is allowed to walk for short periods, three to five minutes at a time the first day, and the time is gradually increased if there is no heat or swelling. The appearance of heat and swelling suggests renewed bone activity and should be treated by replacement in a walking cast for a further six to eight weeks, and then trial walking is again begun.

If sepsis is present it is important to maintain a functional position during its resolution or more deformity may result. It is desirable to immobilise completely as soon as the discharge decreases enough to allow a plaster cast to be applied. Final healing is not usually delayed provided the position is satisfactory.

Case 5—A man aged forty-five was admitted with sepsis of the left foot and marked bone involvement (Fig. 9). Local treatment, antibiotics and complete immobilisation resulted in bone healing with a resultant useful though deformed foot (Fig. 10).

RESULTS

Healing of lesions has been seen in all patients who would cooperate and persist with walking plasters. Some early lesions have now remained healed for over ten years. Although in some patients a second bone has been involved in the same foot, in no one has the same bone been involved on a second occasion, once complete healing and walking were achieved.

A number of markedly deformed feet have been reshaped by operation, and no further breakdown in the affected areas has occurred during a five-year follow-up.

CONCLUSIONS

Tarsal bone disintegration can be a very disabling condition and left untreated it predisposes to many of the chronic foot problems of leprosy patients.

Most patients with tarsal bone disintegration can be treated satisfactorily to ensure a useful foot. Amputation is only necessary in neglected patients with gross deformity, extensive loss of bone or widespread scarring.

SUMMARY

1. Tarsal bone disintegration is a progressive disorder that affects nearly 10 per cent of leprosy patients.

2. Early detection and treatment by immobilisation will result in healing with minimal deformity or disability.

3. Feet involved in advanced lesions can be treated so that there is a satisfactory outcome and amputation is not needed.

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