REVIEW OF LITERATURE
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The trophic ulcers are very troublesome and often intractable complications in leprosy. Ulceration of feet undoubtedly causes more unpleasantness for patient as well as his medical attendant than any other complication of leprosy. So much so that patient often comes to their medical attendant saying “Doctor my feet are killing me”, as very correctly quoted by Ross (1963). The term 'plantar ulcer' was first introduced by Price (1959) who defined them as 'chronic ulceration of the anaesthetic sole of the foot, situated in well defined areas overlying bony prominences, resistant to local or systemic therapy, and characterised by marked tendency to recurrence'.

Hemerykjkx (1959) in a study of 2,479 patients reported that 9.3% of all the lepers have planter ulcers on one or both feet. Ross (1960) in a similar study at the Schieffelin Research Sanatorium, Karigiri observed very high incidence (25.30%) of plantar ulcers. Whereas in a similar study by him in E. Nigeria, the incidence reported is 10%. Srinivasan and Dharmendra (1978) reported that 10 to 20% of leprosy patients suffer from this condition. If these can be
accepted as indicative of world incidence of plantar ulcers then, taking the W.H.O. estimate of total incidence of leprosy which is about 14 million, we have the alarming figure of 1.4 million patients with trophic ulcers of the sole.

Plantar ulcers occur on the under surface of the foot most commonly seen at the head of the metatarsal bones usually first and fifth. Less frequently they occur at the heel or at the end of the toes (Dharmendra and Chatterjee, 1955).

Price (1959) also observed the head of the metatarsals as the common site for these ulcers, distribution being 2nd, 1st, 5th, 3rd and 4th, whereas Keserwani (1976) observed 1st, 2nd, 5th, 3rd and 4th distribution and Kush Kumar (1979) observed 1st, 5th, 2nd, 3rd and 4th distribution. They also reported the high incidence of these ulcers in 4th and 5th decade of life with male predominance. On the contrary, Mukherjee (1977) reported head of the proximal phalanx of the big toe to be the commonest site for these ulcers.

Whereas forefoot (45 cases), heel (14 cases) and lateral border of foot (6 cases) were the sites for these ulcers as observed by Belsare et al (1979) in a study of 65 cases. In forefoot 2nd and 1st metatarsal heads were the most frequent sites reported. On the
contrary, Srinivasan and Dharmendra (1978) in their study of trophic ulcers found that not all parts of the foot are equally liable to ulceration. 70% to 90% of the ulcers occur in small strip of the sole (about 1" in width) across the forefoot in front of metatarsophalangeal joints. Even here, about half the number of ulcers occur in relation to the head of the metatarsal of big toe.

ARTICULO V AND NATURAL HISTORY

Ulceration of the foot in leprosy patients is seldom if ever, a primary manifestation of leprosy. This does not mean that there are no primary leprosy lesions of bone or plantar surface of foot. There are both, but these per se do not cause ulceration. Ulceration is, almost always, without exception due to trauma or pressure necrosis.

According to Dharmendra et al (1955) in causation and persistence of trophic ulcers, various factors play a part such as sensory, trophic and circulatory changes. These are, due to interference with the nerve supply, trauma to the affected part pressure necrosis, repeated injuries, secondary infection after breach of surface leading to sepsis and necrosis of bone. In the case of ulcers on the end of the toes the important factor is the dragging of the toes along the ground due to foot drop.
Price (1959) is of the view that the appearance of a plantar ulcer is the climax of a series of changes that have occurred in the mechanics of the foot, leading to the breakdown of devices which protect the normal foot from damage during the stress of walking. These changes are represented clinically by damage in two areas which correspond to the surface on which friction pressure are maximal. These surfaces are between the plantar skin and the underlying bone at any given moment of walking roll. Corresponding to these areas of stress, it is possible to recognise a deep and a superficial type of damage to the plantar tissues which culminate in a deep and a superficial type of ulceration.

The natural history of plantar damage falls into three parts.

1. The pre-ulcerative stage,
2. The plantar ulcer,
3. The complications of ulceration.

1. The pre-ulcerative stage:

When there is profound and prolonged deep anaesthesia of the sole, there may be no pre-ulcerative symptoms, the ulcer being the first complaint. With lesser deep anaesthesia, there is first burning sensation at one of the recognised sites of plantar damage,
accompanied by tenderness on walking. If walking continued, the burning sensation becomes accentuated at night in bed, and may disturb sleep. The patient limps, but may continue to walk until a further stage is reached which may include the cracking of a large callosity, or a swelling at margin of glabrous skin of the sole. At this stage, patient usually seeks treatment. The sign of pre-ulcerative stage are -

(i) Localised swelling of the foot.
(ii) Localised tenderness on deep palpation.
(iii) Abnormal callosities of the sole.
(iv) A localised blister at the margin of glabrous skin.

2. The plantar ulcer:

The plantar ulcer of the neuropathic foot of leprosy occurs either (a) As a deep and often chronic hole corresponding to damage to tissues close to bone, or (b) As a superficial and often acute ulcer, corresponding to damage at the superficial area of friction damage.

(a) Deep type of plantar ulcers:

This is the common chronic ulcer on the sole of the foot in leprosy, though it may be masked by associated long standing secondary infection. When this infection is minimal, it is seen to be a collar-stud
type of hole leading from the skin surface through a
funnel which opens into the necrotic area adjacent to
the underlying bone. Neglected necrosis blisters
frequently initiate a deep ulcer.

(b) The superficial type of ulcer:

This type of ulceration involves a large area
of skin and may or may not include deeper tissue.
It is not as common as deep type, serious secondary
infection is inevitable and the damage to the foot
may be considerable. A similar ulcer is also seen
under the head of the proximal phalanx of big toe,
at the tips of the toes and at heel.

Drisebach (1939) attributed neuromuscular
changes in leprosy to be most significant in the
etiology of the trophic changes of the foot. The
various positions of foot shifts the weight-bearing
to the lateral surface of the foot and away from the
normal weight-bearing area to an area that is
considerably smaller in size. Added weight is placed
on the head and base of the fifth metatarsal and
calcaneus. These bony prominences cause pressure
necrosis of underlying tissues.

Because of foot drop, and the high stepping
gait necessary to advance the foot, and because of
the varus position of the foot, the forefoot and in
particular lateral part at the level of fifth metatarsal head and less often, the great toe, with its associated first metatarsal head, are frequently dragged along the floor, causing repeated abrasions of the integument in these areas, which finally break down, with ulceration and secondary infection, and may eventually go on to osteomyelitis of these bones.

In a normal foot the part of the calcaneus that is weight bearing is usually a relatively flat surface parallel to the floor. There is frequently an outgrowth of bone along the plantar fascia that forms the calcaneal spur. In the normal foot this is also parallel with the floor. In leprosy patients with paralytic pes planus this anatomy is disturbed. The normal longitudinal arch of foot is lost. This results in small spur of bone, or at least an irregular surface of the os calcis, causing much greater pressure on a very small area of integument which may result in a pressure necrosis, ulceration and osteomyelitis of the calcaneus.

The neuromuscular involvement may lead to the formation of a claw- or hammer-toe deformity, this brings the lancet-shaped tip of the bone in contact with the weight-bearing integument. Here again a sharp irregular bony structure is causing pressure on a very small area of integument rather than a large flat area.
This situation leads to ulceration of the tip of the toes. If the claw foot is very marked it will cause the toes to be in marked dorsal flexion. Thus an added burden is placed on the metatarsal heads. Normally toes have not much value in weight bearing. In normal feet however, they bear some weight, more in walking than in standing. They do help the distal transverse metatarsal arch to a considerable extent in its weight bearing functions. If the toes are no longer in contact with floor the whole burden of the forefoot weight bearing is thrown on the metatarsal heads and this added strain may be just enough to cause trophic ulcers. In fact, this is such a common association that one almost always finds ulcers at the level of the metatarsal heads in the foot with claw foot deformities. Similarly Seddon (1960) attributed fixed deformity to be the important factor.

Although there are vascular changes of the foot in leprosy patients these play a rather insignificant role in aetiology of trophic ulceration, whereas Job (1960) states that defective circulation due to irreversible changes in blood vessels play some part.

Ross (1962) states that ulcers on the sole due to lepromatous leprosy per se are very rare, less than 1% of the total. The ulcers occur on feet which are both anaesthetic and walking. He divided ulcers into two groups.
1. First ulcers on virgin feet.
2. Recurrent ulcers on previously damaged feet.

1. THE FIRST UL C ER

(a) Casual trauma: Some ulcers are caused by cuts, burns, thorn wounds, surface friction blisters and injuries due to badly fitting shoes or badly made shoes, e.g. nails, stitching into the sole. The natural history of such an ulcer is break in the skin, often quite minor, unnoticed by the patient who continues to walk. The wound becomes infected. There is local oedema and regional lymphadenopathy and the wound, subjected to trauma of walking, fails to heal.

(b) Plantar warts: Occasionally, ulcers occur under plantar warts and they are sometimes seen after unskilled cutting for corns or careless removal of callouses.

(c) Cracks: Another relatively uncommon cause of ulceration is the infected crack. Deep cracks may extend to the subcutis and provide an opening for bacilli and beginning of an ulcer. These cracks are commonly found at the lateral margins of heel and at the flexor crease in the forefoot.

(d) Subcutaneous necrosis: The casual trauma, plantar warts, and infected cracks account for only a
small proportion of sole wounds. These three show, first a break in the skin, then bacterial invasion and then a septic superficial ulcer which is unless localised, cause spreading inflammation. But the true and common plantar ulcers begins as a deep necrosis, often at its first appearance penetrating from the skin to plantar fascia or tendon sheath.

These ulcers occur only on feet that are both anaesthetic and walking. The friction forces set up within the foot during walking lead to mechanical damage to the tissues and eventually necrosis and ulceration, which is confirmed by the fact that the distribution of the ulcer corresponds to the distribution of friction forces in the foot while walking (Figure 1 b).

The relationship between forces acting on the foot during walking has been studied by Barnett (1956), Brand and Fritschi (1957) and Price (1958 and 1964).

Barnett (1956) divides the period during which the foot is partially or wholly in contact with the ground in walking, about three-quarters of a second for each step, into 5 phases, which together constitute the walking roll.
Fig. 1

STANDING PRESSURES
(OFFER WOOLTON)

DISTRIBUTION OF ULCERS
(RANDOLPH ULCER STUDY)

THE WALKING FOOT
(AFTER WOOLTON)

Fig. 1(a)

Fig. 1(b)

Fig. 1(c)
THE WALKING ROLL

Fig. II (a)

HEEL PHASE 15%

Fig. II (b)

FOREFOOT PHASE 40%

Fig. II (c)

STANDING PHASE 35%

Fig. II (d)

TAKE OFF PHASE 10%

FIG. II
1. The roll begins with the impact of the posterior-lateral aspect of the heel on the ground quickly followed by the complete heel. This phase occupied 15 to 20% of the cycle (Figure 2 a).

2. It is followed by the standing phase in which heel, outer side of sole and metatarsal heads are in contact with ground. This phase occupies 30 to 35% of the total roll and during it the distribution of pressure is much the same as during standing.

3. During the third or metatarsal heel phase which occupies 10% of roll, the weight is borne only on the metatarsal heads, the heel rising from the ground and extension taking place at the metatarso-phalangeal joint is occasionally absent as a separate phase.

4. During the fourth or forefoot phase, thrust is borne on the metatarsal heads and the toes, the hallux and 2nd toe predominantly. The heel continues to rise and further extension occurs at the metatarso-phalangeal joints. The metatarsal heads will rotate as much as 40 degrees and as the skin is fixed to the ground this rotation is absorbed in the tissues between the skin and metatarso-phalangeal joints. It is during this phase that maximum forward thrust occurs, resulting in compression forces behind the metatarsal heads, and
shearing forces beneath them and tension in front (Figure II c).

5. The final step-off phase occupies 3 to 10% of the roll, thrust is borne entirely by the toes, the main part of it by hallux (Figure II d).

The forces just described act at every step in normal as well as in anaesthetic feet. In normal feet no change occurs. In anaesthetic feet proprioceptive reflexes which normally adjust the pressure put on the foot to the minimum necessary and to prevent slipping during walking are out of action. The result is that at each and every step maximum force is exerted on the foot particularly at heel impact and forefoot thrust and the tissues are damaged through shear over work.

The deformities caused by paralysis, is another factor in etiology of these wounds.

II. Recurrent ulcers on previously damaged feet:

In addition to anaesthesia the foot with healed ulceration may display following defects.

(a) Scarring and loss of friction reducing mechanism,
(b) Necrosis blister,
(c) Fixed deformity,
(d) Discrete cyst of purulent material.
(a) Scarring and loss of friction reducing mechanisms :- Scar tissues being rigid and having poor blood supply, is much less able to withstand the stress forces set up in the heel and forefoot during walking than normal. In addition to scarring the smooth cartilaginous surface of the normal metatarsal head has been destroyed, already abnormal tissues are literally ground between the rough metatarsal head and the walking surface. Such feet may re-ulcerate after as little as 15 minutes of walking.

(b) Necrosis blisters :- This is a subcuticular collection of tissue fluids with black necrotic debris floating in it which is usually sterile. The fluid has tracked under the plantar skin from a deep seated sterile necrosis at the site of previous ulcer. If patient continues to walk, the blister ruptures, infection supervenes and an infected wound results.

(c) Fixed Deformity :- Fixed deformity do not appear to play such an important part in causation of ulceration in leprosy patients, but Seddon's (1960) statement serves to underline the difficulties that ensue when secondary fixed deformities are allowed to develop and the extreme importance of detecting the pre-ulcer and thus preventing the bone and joint infection that so commonly lead to fixed deformity.
(d) Intermetatarsal ulcers: Small cysts (Kulonski and Olrman, 1936) containing organisms are commonly present in anaesthetic foot which at one time or another had plantar ulcers may flare up at any time causing acute septic inflammation in tissue of the foot. The nidus of infection may be in the bone, particularly metatarsal or calcaneum, in cartilage or tendon or in subcutaneous tissue. It may contain a small foreign material, such as a thorn or a piece of dressing or it may simply contain purulent material or bacteria. The factors which cause such cyst to become active after as much as 9 years are not known but clinical picture is clear. The patient presents a hot swollen, painful foot, with lymphadenitis.

Anderson (1964) also described 'Roll' to be the crucial factor in etiology of plantar ulcers. The majority of ulcers appear to develop from 'necrosis blisters' which are initially sterile and only after break through become infected. The further development can be described as evil cycle of 'scar, ulcer, scar'. If left unchecked, the deeper structures, bone, joint and tendon are involved.

Price (1964) also described the etiology and natural history of plantar ulcers in four phases
(1) Primary tissue damage, (ii) Primary plantar ulcer, (iii) Collapse of foot, (iv) Appearance of secondary ulceration.
Srinivasan and Mukherjee (1964) classified the ulcer in following groups.

1. Uncomplicated: Those without involvement of bone or other complications but are indolent and not healing with 'routine' treatment. These ulcers show heaped up and overhanging edges and may be deep, penetrating or even perforating the whole foot.

2. Complicated: Ulcer with involvement of bones and joint or tendon sheath or those which present severe deformities.

3. Extensive: Ulcer that extend over a large area. Those may also be considered as a variety of complicated ulcers.

4. Recurrent ulcers:

Srivastava and Keserwani (1976) mentioned following factors responsible for causation of trophic ulcers:

1. Sensory, trophic and circulatory changed in the part due to interference with nerve supply.

2. Trauma to the affected part due to pressure and repeated injuries.

3. Infection after breach of skin surface leading to sepsis and necrosis of bone.
Mukherjee (1977) described three primary causes of neuropathic plantar ulcers.

1. Injuries from outside.
2. Injuries from within the foot.
3. Infection.

TREATMENT

Conservative:

In the treatment of all ulcers whether they affect bone or not, the fundamental principles are to cleanse the ulcer and endeavour to encourage the formation of healthy granulation tissue, and then to protect the granulations and thus aid healing.

Chaulmoogra oil had been used in the treatment of leprosy in India from ancient days. Sushruta (600 B.C.) mentioned "tuvarka" as a potent remedy against leprosy.

Paul (1936) recommended a procedure similar to that used for varicose ulcers, namely with the leg raised and vein emptied overlapping pieces of adhesive strapping are firmly placed over the ulcer, the strapping is left until it ultimately separated by the gradual soaking of the exudate from the ulcerating surface. The chief objection to this method is that foul smell from the dressing is usually
complained by the patient and the nursing staff naturally dislike such a method.

Cochrane (1940) used similar method but filled the cavity with 5% sulphanilamide paste (sulphanilamide 5 gram, Adap Lanac - 70 gm and Paraffin liquid - 25 gm) and leaving it for a week. The results of this method were encouraging and where the ulcer is not excessively dirty than this method saves dressing and encourages healing.

Ryzi (1936) advocated the syringing of sinuses with 1:20 solution of Detol.

Low and Chatterjee (1937) and Chatterjee (1955) recommended injecting the trophic ulcers once a week with hydnocarpus oil. ½ c.c. of the medicine is injected at each puncture. After the injection the ulcer is dressed with same remedy used for injection. Similarly, Cochrane (1940) used hydnocarpus oil and cod-liver oil in alternate cases and found that ulcer heal just satisfactorily with either remedy. All the ulcers do not respond to treatment. Where there was much thickening of bone and specially if there was much pain associated with periostitis or if there was not an adequate covering of subcutaneous tissue over the head of the metatarsal bone injection treatment failed.
Mehta (1939) reported very encouraging results in treatment of trophic ulcers by using solution containing Rivanal for injection into subcutaneous tissue near the ulcer and into the neighbour of nerve supply of the part. Das (1940) used same solution with addition of Eflavine and Trypaflavine for intravenouse injection. Healing was obtained only in 14.7% of the patients.

Oberdorffer and Collier (1939) have recommended the ointment containing Mercurochrome (2 oz), Honey (2 oz), cord liver oil (8 oz), Zink oxide powder (4 oz), Bismuth subnitrate (2 oz) and Vaseline (13 oz) but this ointment is extremely expensive.

Khan (1939) tried various local dressings, such as electrolytic chlorine lotion, chaulmoogra oil, Brilliant green, fluorocin in various dilutions, acriflavin, mercurochrome, saturated solution of magnesium sulphate, 30% solution of Dettol and ointments of various kinds and observed little improvement. The results of Dettol were best of these remedies. But as soon as patient start walking the ulcers were same as before. With this he concluded that rest is an important factor in treatment of trophic ulcer.

Lang (1940) used a mixture of Eucalyptus oil with iodoform (grs. 10 : oz 1) for cleaning of ulcers
and when the granulation tissue has commenced equal parts of castor oil or olive oil is added to prevent sticking of dressings owing to tendency of the eucalyptus oil to evaporate.

Cochrane (1940) used Eusol lotion dressing twice or thrice a day and found valuable for cleaning ulcer and encouraging the formation of healthy granulation tissue. Once the granulating surface is clean and healing commenced then a protective dressing was applied. He used either the Eucalyptus oil and iodoform with equal parts of castor oil as used by Lang (1940) or an ointment containing acid boric, oil Eucalyptus, Bismuth subnitrate, Zinc oxide, Castor oil. He further observed that if granulations are excessive, painting with 2½% solution of silver nitrate is sometimes effective. However, he continued dressing with eusol in presence of such oozing.

Sharmendra and Sen (1955) advocated dressing with the by product of Dapsone. He studied 22 cases and found that within a few days of starting the treatment the discharge from the ulcer diminished and was totally eliminated in a short time. In 15 cases the ulcer completely healed and there was no relapse during one year. In the remaining cases although the ulcer become smaller cleaner and drier but they did not heal completely even after treatment
for 3 to 12 months. X-ray examination showed no abnormal changes in first group (ulcer healed). In all the cases of second group (ulcer not healed), chronic sclerotic and destructive changes were found in phalanges and/or metatarsals. Thus correlation between the results of treatment and changes in the small bones of the feet brings out the fact that complete and permanent healing of trophic ulcer cannot be expected if diseased and dead bone lies at its bottom. For permanent healing in such cases the diseased bone has to be removed.

Saxena and Mathur (1963), Mathur and Saxena (1965) and Mathur (1965) have shown beneficial effect of local application of priscol in leprosy deformities and ulcers. Whereas Mathur et al (1966) treated 25 patients of ulcers with perineural priscol injections and observed encouraging results.

Mukherjee (1963) suggested the use of three type of dressings (1) sterile dressings, (2) Sterile dressings with topical antiseptic ointment, (3) Chlorinated lime with basic acid solution wet dressing. He claimed that 80% of the ulcers healed in this way.

Operative:

Paul (1936) was the first who described metatarsalsectomy for these ulcers. The operation was done under tourniquet after devascularising the limb.
Cass (1939) recommended a more radical approach to the extremely bad forefoot with well conserved hind-foot. He sacrificed the whole offending area in order to stop once and for all the tendency of the scar to 'creep backward'.

The use of plaster of paris cast permits healing if it is continued long enough. The earliest reference to this appears to be that of Khan (1939) and represent the application of principles enunciated by Trueta in the treatment of war wounds during the Spanish civil war. He studied fifteen cases in 3 batches.

The first batch: He removed bone in five cases and scraped the infected area and immediately applied the plaster case. Three were cured within 4 to 8 weeks. In one plaster had to be removed.

The second batch: Ulcers were curetted and plaster case given. Three were cured within 4 to 8 weeks, one ran away and one died of pulmonary tuberculosis.

The third batch: Plaster case only. Three were cured within 6 to 10 weeks. In 2, the plaster had to be removed. Here it took longer for ulcer to heal and necrosed bone came out in pieces.
By this he concluded that rest and protection from pressure are important items in the treatment of lepromatous trophic ulcers. Paul (1947), Fisher (1955), Bose (1955) and Price (1959) also confirmed success of this method.

Cochrane (1940) treated these ulcers by metatarsectomy as advised by Paul but packed the cavity left after removal of a metatarsal bone with 5% sulphanilamide paste and leave the dressing for a week. The result were excellent even when a grossly infected bone has been removed.

Drisebach (1959) gave following principles of treatment for trophic ulcers.

1) Prevent further trauma to the foot: This usually means rest. The feet should be elevated especially if there is any evidence of oedema or cellulitis of the feet. If there is associated cellulitis, lymphangitis, or regional lymphadenitis, the appropriate chemotherapeutic agent should be used in full dosage. Continuous hot saline fomentations or frequent hot saline 'soaks' are of great value.

2) In long-standing chronic ulcer it is of value to debride the surrounding hyperkerotic skin and overlying eschar. Hot fomentations and/or soaks and the mechanical cleansing of the ulcer is of greater
importance in obtaining early healing than any
medication that is put on the ulcer. Once the ulcer
is cleaned up, and there are no longer signs of
infection of the surrounding tissues, the foot could
be immobilized in a walking plaster of paris cast.
The pre-requisites for a plaster boot are (1) a clean
healthy ulcer, (2) no cellulitis, (3) no underlying
osteomyelitis of the bones of the foot.

Once these ulcers heal, and foot goes back
to weight-bearing they may occur. Prevention of
further trauma is all that is necessary. Usually an
inexpensive rubber soled canvas shoes is all that is
needed, or a 'native' sandal if it is so made as to
cause no injury to the foot.

In recurring ulcer caused by pressure
necrosis, various types of foot wears have been used
by various workers or alternative is to remove the bony
prominence that cause the pressure necrosis ulcer.
Metatarsal head should be excised with the object of
maintaining as much of the length of the shaft of the
bone as possible. The incision should be away from
the plantar surface of foot. In some cases where
there are multiple ulcers in relation to the distal
metatarsal arch it may be necessary to remove the head
of all the metatarsal, in other words to do a
transmetatarsal amputation. This has been found to be
a good procedure. A technique that saves all the
toes rather than the classical procedure where they
are amputated.

For the ulcers of heel, a fish mouth incision
is made which reflects the heel pad distally. Then
with an osteotome the plantar surface of the calcaneus
is flattened so that there is no protruding bone that
may be source of further pressure necrosis. In case of
osteomyelitis of metatarsal bones or of a single tarsal
bone, it is usually advisable to do a surgical removal
of the sequestrum, or excise the affected bone or a
portion of it, or curette the tarsal bone.

In some cases despite of all efforts, an
amputation of some kind become necessary.

In the case of a large plantar ulcer skin
grafts also have their place, but only in well
selected cases of large ulcers, without much loss of
subcutaneous tissue and in which there is no
osteomyelitis of the bones of the foot.

Price (1940) described treatment of Plantar
ulcers according to stage of ulcer.

1. Pre-ulcerative stage:

The patient is put to bed with foot end of
the bed raised though he is allowed to move with the
help of crutches to perform his toilet. Within a week
or ten days, oedema and tenderness disappear and patient resumes normal activities with rigid sole foot-wear. Alternatively, the patient remains in bed for three days until initial oedema has subsided and a plaster cast is then applied to enable him to be ambulant.

2. Stage of plantar ulceration:

After control of oedema and infection by a few days in bed, the plaster cast is applied. The cast is removed after 6 weeks. In this time ulcer heals, if not healed cast re-applied until healing occurs. To prevent recurrence of ulcer adequate foot wears are provided.

3. Healed ulcers:

For these special foot-wear with a rigid sole and soft insole are provided.

The Standard Rigid sole Sandal:

The sole of the sandal is prepared in wood, and uppers in leather and the insole in sponge rubber. The best materials depend on local conditions.

Wood is the easiest and cheapest material to work for soles. It is easy to obtain and can be worked by local craftsmen using their traditional tools. The shape of the sole is indicated in Figure III a. He gave importance to following:
(a) THE WOODEN SOLE. THIS MUST BE LONG ENOUGH TO PROTECT THE TOES, AND RISE SHARPLY ENOUGH FROM THE METATARSAL PAD TO MAKE POSSIBLE THE ROLLING MOVEMENT NEEDED FOR WALKING.

(b) THE COMPLETE SANDAL. THE LEATHER HEEL PIECE INCORPORATES A HEEL STIFFENER; THE STRAPS ALSO ARE OF LEATHER AND ALL ARE LINED WITH FELT. THE INSOLE IS SPONGE RUBBER BONDED TO CANVAS AS USED FOR CARPET UNDERLAYS. THE HEEL AND SOLE ARE PROTECTED BY RUBBER; SPECIAL SIZES AND SHAPES ARE OBTAINABLE FROM CLOG MANUFACTURERS.

FIG. III
(i) the patient's foot should slope slightly downwards, from heel to metatarsal heads;

(ii) the sole then rises sharply to the toe of sandal;

(iii) the point of contact of the forefoot underlies the metatarsal heads of the foot, and the sole then rises at an angle of 20° to the ground.

The uppers are made from local leather and must be soft enough not to chafe the skin. The heel is stiffened by a heel stiffener that fits between two layers of leather as in a pocket. The heel piece must project a definite distance up the back of the heel; if it is too short, it tends to leave the heel at the end of the step, while if it is too long it will cause a friction sore at its upper end. The straps are of soft leather and are lined with a half thickness of \( \frac{1}{2} \) in. grey orthopaedic felt, using an adhesive.

The insole is made from one of the type of rubber. The complete sandal is fitted to the patient and may need slight modification. The patient is observed daily for the first few days. In some cases, it is essential to provide short socks, when the skin is particularly susceptible to injury.
Deformed feet can not be satisfactorily fitted with standard sandals, and in most cases it is necessary to make special foot-wear. Grossly deformed feet may need amputation.

Ross (1962) treated his cases on the principles of Truetta (1944). He recommended following treatment depending upon the type of ulcer.

1. THE VIOLENTLY AFFECTED WOUND:

These are found in association with hot swollen feet with inguinal adenitis and with copious discharge from the wound.

(i) Cleaning of the wound: is done by warm water and soap.

(ii) Excision of dead tissue: Anything obviously dead should be removed, along with the fibrous scars at the edge of these wounds to allow epithelium to grow.

(iii) Allowing free drainage: If drainage is not proper even after thorough debridement then further incision and if necessary, excision of healthy tissue, must be done until the wound is no danger of becoming an abscess.
(iv) Immobilisation of the part: Limbs should be encased in a light non-weight bearing plaster of Paris cast.

(v) Prevention of cross infection: Is done by encasing foot in plaster of Paris cast.

(vi) Prevention of repeated trauma: The plaster of Paris cast entirely cuts out the walking roll which is the underlying cause of most of these ulcers. If the discharge has not ceased and the small subsided in two to three weeks after the application of the plaster, then the plaster should be removed under aseptic conditions and the wound re-examined. It is usually found either the drainage is inadequate or that a previously undetected sequestrum is present.

2. THE CLEAN DRY ULCERS:

They may be treated along the same lines but bed rest is not necessary and they can be sent home in a weight bearing plaster of Paris cast, or "Karigiri Boot". It is cheap, costing approximately 5/-, effective, acceptable to the patients and can be applied to out-patients who have to go home the same day.

3. THE MILDLY INFECTED ULCERS:

were treated by cleaning the wound with soap and water soaks, excision of dead tissue, providing free
drainage and immobilization of the part in plaster boot. These patients are put on strict bed rest "patients must not walk on wounded foot".

4. **THE INFECTED CRACK**

The large majority of these heal in 2 to 4 weeks if the edges are excised, the wound cleaned and then immobilised by strapping a wooden rocker with a felt insole on to the foot by means of elastoplast. If there is sinus associated with a crack, then excision to provide free drainage and immobilisation in a weight bearing plaster of Paris cast is essential.

There are three adjuncts to the treatment.

(i) **Antibiotics.**

(ii) **Preventive surgery:** Claw toes and foot drop secondary to paralysis should be corrected. Toes which are twisted and rigid, should be removed. The only bone cutting operation which commends itself is the removal of spur of bone from the underside of the calcaneus which is commonly present following an ulcer on the heel. If sequestration occurs, remove the sequestrum but normal metatarsal heads should never be removed.

(iii) **Ascorbic acid.**

Ross (1964) treated 502 cases of plantar ulcers on this basis and reported good
results in all except six, 4 of them were treated later by metatarsal head resection and 2 by amputation.

Anderson (1964) is of the view that when active ulceration is present surgery should be limited to securing free drainage only. In this stage one should never remove anything obviously dead tissue, at the number of times evidently dead tissue when left under a Plaster of Paris cast, proves very vital indeed.

While the ulcer is freely draining, and while there is oedema and swelling, bed rest with elevated foot is the only sensible treatment. As soon as oedema subsided and ulcer is dry patient may be made ambulant in a well fitted Plaster of Paris cast, with a rocking device preferably a Schlar iron. The majority of the ulcers will heal within six weeks. To prevent recurrence of the ulcer protective foot-wears were used.

Srinivasan and Mukherjee (1964) described a more radical approach in the management of chronic foot ulcers. They recommended thorough debridement of all aspects of ulcer i.e. edge, floor, track, abscesses, base and underlying bone. They performed sequestrectomy or sanerization rather than metatarsectomy. In severely destroyed and deformed foot they have recommended salvage
procedure, so that the removal of offending part
will make the patient ambulant with or without
prosthetic device.

Bhasin and Antia (1972) performed radical
resection of metatarsal bone through a dorsal approach
in cases of recurrent ulceration in leprosy. They
resected the metatarsal subperiosteally close to the
base of the bone, preserving tendon and vessels.
Wound was closed only by few catgut sutures, skin was
not sutured and was allowed to heal by secondary
intention in a below knee plaster. To prevent
recurrence after healing of ulcers they used footwear
a shoe with a build up of the forefoot and toe cover.

They treated 16 feet of recurrent plantar
ulcers by this method and reported that 10 cases have
remained free of plantar ulcers, 5 of these have been
ulcer free for 2 years or more, and 4 have remained
ulcer free for 4 years.

Andersen (1975) recommended the transverse
metatarsal head resection as a surgical approach to
the problem of recurrent forefoot ulceration. The
indications are any kind of forefoot ulceration or
forefoot scarring. Contraindications are ulceration
and/or scarring of the mid or hind foot since such
conditions would not permit safe weight-bearing on the
reduced foot.
The operation aims at removing sufficient amount of skeleton from the forefoot so that the cut ends of the metatarsal bones are well proximal to the proximal edge of the ulcer/scar. This permits a trouble free take off phase.

The incision is made transversely on the dorsum of the foot, at the level of proposed osteotomy of the metatarsal bones. The incision is carried down to the bone. The metatarsal bones are divided subperiosteally in a straight transverse line. The distal portion of the bones are twisted out, including the metatarsal heads. Sesamoid bones if any must be removed. The resulting gap is loosely packed with plain vaseline gauze. The foot is dressed in a bulky dressing. However there are few variations to the technique.

In cases of septic arthritis removal of metatarsophalangeal joint as a whole is indicated. Another variation being removal of all the remnants of bone distal to metatarsophalangeal joint along with distal part of metatarsal. The indication being absorbed forefoot, that externally look rather like a forefoot amputation, but has a very thin, adherent-plantar tissue in the take-off area.

Srivastava and Keservani (1976), Keservani (1976) treated 32 and 64 cases respectively of plantar
ulcers by pre-operative plaster of Paris cast to
give rest for 2 weeks under cover of systemic
antibiotic therapy. When the discharge stopped,
cedema subsided and granulation tissue started
appearing, the cases were subjected to thorough
debridement of the ulcer and metatarsectomy through
the dorsal incision; they excised head and neck of
metatarsals preserving the tendons and immobilised
the foot post-operatively in below knee Plaster of
Paris cast, till the ulcer healed. To prevent
recurrence patients were advised to use micro-cellular
chappals. They reported very gratifying results of
this technique and observed that gait was not affected.

Kumar (1979) treated 68 plantar ulcers under
the metatarsal heads. Superficial ulcers were treated
by plaster immobilisation and split thickness skin
grafting if needed. Uncomplicated deep ulcers were
treated by curettage and debridement whereas deep
ulcers were treated by pre-operative Plaster of Paris
cast, metatarsectomy through a dorsal incision, as
done by Rosenvi (1976). He reported best results
with metatarsectomy.

Wynn Williams (1953) treated plantar ulcers
by split skin graft and reported replacement by full
thickness skin was not necessary. Even the large ulcers
of the heel healed by this method and there was no
problem even after three years.
Walkey and Williams (1965) treated 128 cases of plantar ulcers by excision and thin split skin graft and reported that healing occurred in 20 days in 2/3rd of his cases. 75% of the total cases healed after first operation and 90% after the two operation. In the later case the period has been four to five weeks. Majority of remaining 10% of cases had large deep lesions of the heel, the size of ulcer reduced by the procedure and eventually healing took place.

D'Hoooghe and Hendrickx (1975) compared the results of treatment following skin grafting and plaster immobilisation. Plaster of Paris treatment has the advantage that it enables the patient to be ambulatory. Disadvantage being that it prevents control of wound and the early detection of complications. Further the wound heals by scar tissue, which is an inadequate substitute for skin, which especially in a weight-bearing area, must be strong and elastic. The skin grafting of ulcers therefore must be performed whenever possible. They are also of the view that skin graft should not be taken from insensitive area of the leg, because denervated dermis however is usually thinner than normal and the adnexae, hair follicles and skin glands which contribute much to the healing and re-epithelialization of the donor area may
also be atrophic, so that delay in healing of the
donor area and sometimes loss of full thickness skin.

Palande and Ashaguraj (1975) recognised
the changes in the posterior tibial nerve and vessels
because of compression in the fibro-osseous tunnel
and used decompression of the posterior tibial
neurovascular complex for the treatment of chronic
plantar ulcers with posterior tibial neuritis. They
reported that 53 of 58 cases of plantar ulcers healed
in short period after the decompression.

Delsear et al (1979) treated 65 cases of
plantar ulcers in leprosy by various surgical
procedures.

(i) Curettage with or without decompression of
posterior tibial neuro-vascular complex in
superficial ulcers.

(ii) Metatarsectomy with or without decompression of
posterior tibial neuro-vascular complex in deep
ulcers, where bone was infected or presenting as
a pressure point.

(iii) Curettage with skin grafting with decompression
of posterior tibial neurovascular complex in
heel ulcers.

(iv) Amputation of toes in gangrenous cases.
They encased the feet in plaster cast till the ulcers healed. Best results were observed after decompression of posterior tibial neuro-vascular complex along with other surgical procedures. The recurrence rate was 25 to 30%, maximum being in first six months.

Pati et al (1981) reported his observations following treatment of 57 metatarsal head plantar sores in 43 patients treated by excision of the head. Removal of offending metatarsal bone was done either by plantar (9 cases) or dorsal approach (43 cases). Recurrence was not observed even in a single patient. However, 5 cases had metatarsal head pressure sores at other sites. The ulcers healed faster by dorsal approach.