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Editorial

MANAGEMENT OF PLANTAR ULCERS— THEORY OR PRACTICE?

Introduction

Nerve damage and its complications are the major causes of stigma in leprosy, caused by reaction against *Mycobacterium leprae* in the nerves.¹⁻³ Solid staining bacilli can be found in trunk nerves long after completed leprosy treatment.⁴ Reactions cause reversible or irreversible, partial or total nerve damage.^{3,5}

Damage in trunk nerves of the extremities causes impairment of sensory, motor and sudomotor function of the hands and feet. Loss of sensation deprives the extremities of protection against injuries, causing ulcers and septic conditions that lead to deterioration of the limbs, and result in severe physical and social disability.^{1,3}

Neuritis and its consequences are preventable but, in spite of this, leprosy patients are still suffering from ulcers. There are programmes where 50% or more of all new patients are reporting with a WHO disability grade of 1 or more.⁶⁻⁸

Ulcers and ulcer care constitute a large problem in the management of leprosy. In leprosy hospitals the majority of beds in surgical units are occupied by ulcer cases. Beds utilized for preventive surgery are comparatively few.

During the courses in ulcer management at ALERT (All Africa Leprosy and Rehabilitation Training Centre), we teach that if we know why, how and when ulcers are formed, how ulcers heal and what prevents some ulcers from healing, we can know how to prevent and treat them.

The prevalence of ulcers in leprosy programmes evokes some questions:

Do the workers in leprosy management have enough knowledge about the pathomechanisms of ulcer formation to prevent and treat ulcers?

- 2 Has recent research contributed essential knowledge to our understanding of prevention and treatment?
- 3 Are present treatment programmes adequate?

Why are ulcers formed?

It is well understood that loss of sensation of a sole of the foot allows various types of trauma to attack the foot unnoticed.¹

Sensory testing helps leprosy workers to identify cases at risk of suffering ulcers.9-11

Such tests have to be performed in the field, where the patients are. Consequently they have to be simple and repeatable.

Threshold values for light touch are measured with nylon filaments calibrated to bend at specific weights.¹² Patients not responding to a 10-g filament are more likely to get ulcers.^{13,14} Some normal feet are not able to feel the 10-g filament, especially in the heel area.¹⁵

The qualities of sensations that are needed to protect a foot from injuries are not fully understood. Vibration perception threshold studies, using fixed frequency and varying amplitudes, have shown that patients with disintegration of the tarsal bones have more severe impairment of vibration sense in affected feet than in the contralateral unaffected feet.^{14,16} In some ongoing studies of the restoration of plantar sensation, patients are reporting returning sensation as an awareness of the ground they are stepping on, qualities of sensation for which it seems to be difficult to design a simple and repeatable assessment.^{17,18}

How are ulcers formed?

Mechanical factors cause ulcers on hands and feet. In the absence of a protective sensation patients injure themselves. A range of mechanical forces from approximately 50 g/cm², closing the capillaries and causing ischaemic ulcers, to above 80 kg/cm², causing penetration of the skin, is in action.¹

The most common force is the repetitive moderate stress caused by normal walking, which ranges from 2 to 5 kg/cm². Occasionally, the mechanical stress may reach peak pressures above 5 kg/cm², especially when feet are unprotected and deformed. During normal walking a foot experiences about 725 impacts per km.¹⁹ A person who has sensation will immediately react with discomfort and pain on overload and alter his/her pattern of walking or take a rest. If that person or people around him are unaware of and do not look for physical signs of overload, damage may happen unnoticed. Consequently, awareness and physical examination *must* compensate for loss of sensation. It seems difficult to create this understanding and make a patient practice what he should have learnt.^{8,20}

Penetrating objects and burns are examples of direct trauma.¹ Classical examples are stones in a shoe or penetrating thorns. Exposure to low heat over a long time can cause severe burns. Walking barefoot on hot stones or riding in a lorry, with the feet on top of the gear box, are typical causes. Blisters, the size of the whole sole, may be the result.

Ischemic ulcers are mainly caused by tight shoes, shoestraps or bandages.¹ All that is needed to cause damage is very low pressure over a long time. New footwear and wrongly applied bandages and splints are typical causes.

Leprosy patients may not feel pain when they step on infected ulcers.¹ Infection is spread to deeper tissues causing the involvement of bones, joints and tendon sheaths, loss of tissue and mobility.

Where are ulcers formed?

The distribution of ulcers depends on mechanical factors.^{1,21} A leprosy worker

understanding this will be able to identify risk factors and give proper advice to patients at risk.

Fortunately three out of four ulcers occur on the forefoot. Ulcers on the lateral part of the sole and under the fifth metatarsal base and on the heel have a high risk of severe complications.

Several methods are available that evaluate the pressure points under the sole, but only the Harris mat is usable under field conditions.²² The rubber mat, with ridges of different heights, gives a footprint on a piece of paper, where higher pressure is registered as a deeper colour. The Harris mat can be applied on top of differing walking surfaces and be cut to fit the inside of shoes. The equipment, consisting of the mat, a rubber roller, stamp, ink and some paper for printing, is easy to carry in the field.

Dynamic and static foot pressure has been studied in insensitive, neuropathic feet, using pressure transducers, barographic images, and image-processing systems which are sometimes microprocessor-controlled.²³⁻³⁰ Some authors²⁷ claim that the measurement of vibration perception threshold alone (considered a bedside method) may be useful in identifying patients who are at risk of suffering from ulcers. Foot pressure studies are used to determine the specific areas that are at risk of developing ulcers. Unfortunately these methods are not usable in the field.

Modern computerized methods that produce graphic displays of position and magnitude of pressure under the sole as well as the position in and proportion of the stance phase are now available. They increase our understanding of dynamic forces during walking. Some authors claim that such investigations provide information about structural changes, e.g. in the metatarsal heads or tarsal bones, and can be helpful in guiding orthopaedic surgeons to suitable early corrective action.^{23,24} Peak pressure studies give valuable information in the design of protective footwear.^{24,25,28}

Studies that give us a deeper understanding of the dynamic forces causing tarsal disintegration and allow us to identify risk factors or very early signs of deterioration are welcome.^{16,30} Our present experience is that once the process of deterioration has started it is difficult to stop. Tarsal disintegration condemns a leprosy patient to wear special footwear for life. Such patients often have to have amputations.⁶²

How do ulcers heal?

Tissue damage heals by scar formation. Primary healing leaves a minimum of scar. Secondary healing leaves a bulky and hard fibrotic lump of a scar. Tissue from the sole of the foot, the palm of the hand, the pulp of the fingers and toes is unique and difficult to replace. Loss of tissue in the heel is serious. The forefoot has some wider margin for tissue loss. Tissue loss must be kept to a minimum.^{1,21,31–33}

Factors preventing healing are infection, sequestra, insufficient immobilization, poor circulation, tension in tissues due to atrophy and scars, and foreign bodies. Such factors also influence the quality of scar. To our knowledge, the healing ability in leprosy patients is not impaired.³³ Can healing be accelerated? Electrostimulation has been tried for many years. Studies indicate that high voltage stimulation and low intensity direct current stimulation of decubitus ulcers does accelerate healing.^{34,35}

Many 'healing agents' have been suggested, but it causes confusion among leprosy workers to claim superiority of a specific agent and deflects them from the essentials in ulcer care—i.e. keeping ulcers clean and immobilizing the ulcerated feet. One author³³ emphasizes that special topical agents add nothing essential to ulcer care. Ulcers usually fail to heal for lack of attention, not for lack of a specific topical agent.

Antibiotics are overused. Many of the strains found in ulcers are resistant to our common antibiotics.^{36–39} Widespread and indiscriminate use of antibiotics only adds to the number of resistant strains and very little to the treatment. Very few cases really need antibiotics. Antibiotics are necessary only in cases who show general signs of a septic condition. Antibiotics should never be a substitute for surgery or general basic care.

The majority of patients with plantar ulcers are not hospitalized but in their own homes. Consequently the time and money currently being spent on developing more or less fancy remedies should be spent on developing and teaching home care.⁸⁻¹⁰

How can ulcers be prevented?

Prevention of ulcers begins with early case detection, a team work that involves everyone from the leprosy control manager to the health worker, the physiotherapist and the surgeon.

A formula that emphasizes that the prevention of disability is a concern and responsibility of every health worker could look like this:

LM = LC + POD + PRS

(LM = Leprosy Management, LC = Leprosy Control, POD = Prevention Of Disability, PRS = Preventive and Rehabilitative Surgery).

- 1 Leprosy management is incomplete without disability prevention.⁴⁰
- 2 Prevention of disability must not only be a concern of the physiotherapy, occupational therapy, orthopaedic workshop and health education staff in their own clinics. It must also be an integral part of leprosy management.^{8,9}
- 3 It must be emphasized that surgery should be used not only to salvage definite disasters but also to prevent disability.³
- 4 To achieve this, leprosy control staff, POD staff and surgeons must all work together.

What a surgeon finally discovers in his operating theatre depends on the efficiency of disability prevention, treatment and case selection in the field. The surgeon should:

- 1 participate in training,
- 2 pay visits to the field to establish and develop routines for patient selection,
- 3 develop surgical treatment in the field, and
- 4 integrate basic surgical activities into peripheral hospitals and clinics.

The leprosy surgeon is an underutilized resource in leprosy management. This may be the fault of the surgeon. The demands in the operating theatre may deflect the surgeon into problem-solving too late. Problems start in the field where the patients are, but surgeons tend to be involved only when problems are large and irreversible, i.e. patients who have major septic conditons or major disabilities in need of extensive surgical intervention.

Early nerve lesions are reversible, but it must be admitted that we still have not

mastered the treatment of neuritis in spite of years of research.⁶² Patients deteriorate during and after neuritis treatment. Many leprosy surgeons believe that nerve decompression, mainly of the ulnar and posterior tibial nerves, combined with corticosteroid treatment, helps in minimizing nerve damage.^{42–45} The timing of surgery is important, and the surgeon is often brought in too late. Great responsibility rests on field staff in identifying and selecting cases early enough. Well-trained field staff and well-developed policies for the treatment of neuritis are essential for success. Neuritis is best treated by a team.

There has been no miracle remedy discovered that keeps dry skin moist or removes excess dry skin other than soaking in water, oiling and rubbing with a stone (pumice, ceramics or any stone cut to obtain a flat surface).⁴⁶ Most dry and cracked feet will become soft and smooth after 1 week of daily treatment.

Insensitive feet must be protected with footwear with a soft insole that evenly distributes pressure and a hard undersole that protects against any penetration of sharp objects. Different types of rocking devices are also essential to further neutralize pressure points. Moulds are a necessary but controversial aid, because ill-fitting moulds do more harm than good.^{1,47}

Patient acceptability of protective footwear is a problem in many programmes, because most leprosy shoes look different from ordinary footwear. Since this creates a new stigma they are often not accepted, but few acceptable alternatives are available.⁴⁷⁻⁴⁹

One publication does describe a plastic sandal which can be mass produced at a low cost,⁴⁸ which seems to be well accepted by the population. It has a controlled rigidity and a certain rocking effect, a hard-wearing plastic sole and a soft insole which can be changed when worn out. The shoe is durable and can easily be repaired either by a local shoemaker in the street or by the patient.

Trials are under way with commercially made shoes that are high enough to allow a protecting soft sole to be inserted. More sophisticated methods of analysing the dynamics of different footwear can facilitate the design of protective shoes acceptable to the patients.^{23–25,58} Local shoe factories can modify commercial models. Simple studies measuring the effect of such arrangements would be welcomed.

How can ulcers be cured and prevented from recurring?

Much of the recent literature on ulcer care is concerned with advanced ulcer treatment and high technology assessment methods. More studies at field level would be welcomed. Has the enthusiasm for MDT and the large reduction of patients in our programmes lessened our interest in the numbers of leprosy patients who are at risk of developing disabilities? There are publications indicating that the incidence rate of disability may be higher after treatment than during treatment.⁷ Are the at-risk patients caught early enough? Is the identification of at-risk patients being delegated? To the community? To the patients themselves? It may very well be that in the future the responsibility for prevention of disability and caring for the disabled should be that of the general health care services or the community,⁴⁰ but until this responsibility has been so delegated it remains with the leprosy control programme.

Ulcer prevention and treatment is basically a management problem. It is a matter of applying simple basic principles:^{1,46,47}

- -Soak and oil the dry skin.
- -Inspect the soles of feet.
- —Use protective footwear. Change the walking pattern. Rest whenever there are signs of tissue trauma.
- -Clean, trim, immobilize and cover ulcers.
- -For deep ulcers, apply appropriate surgical measures.
- —After ulcers have healed, re-evaluate the foot for further preventive measures. Is preventive or corrective surgery needed? Is there a need for prescribing another type of footwear?

It is amazing how much time and effort is spent in looking for *fata morgana*, 'the healing shoe', 'the healing ointment' that should be spent in the training of staff and the development of proper routines for the prevention and treatment of ulcers.

Treatment starts with the field workers identifying and diagnosing the cases.⁵⁰ Today ulcer cases in many programmes are either sent to a central hospital for treatment, or not treated at all. In some field programmes the ulcer care offered ends with a symbolic soaking, superficial trimming and bandaging once every 4 weeks; patients believe in the ritual as performed in the field clinic but do not understand it and make no effort to copy or repeat it at home.

While studying the treatment of ulcers in many programmes and the patients' behaviour, 'classical' health education does not seem to have been very effective.²⁰ Self-examination and self-care must be taught, and practised where the patients normally live. Teaching situations where patients share experience with other patients are recommended.¹⁰

In many institutions the average stay for ulcer patients is as high as 3 months. Patients tend to get dependent on their hospital beds. They show confidence in the nicely-smelling solutions and beautifully coloured dressings, and do not believe that anything essential that is done could be repeated in their homes. Leprosy field workers complain that patients who are discharged from hospitals and treated in the field feel deprived of the special medicines and material used in hospitals and show a lack of confidence in the treatment in the field clinics.

Treatment of simple ulcers

To effectively reach all patients in need it is necessary that:

- 1 Ulcer care should be decentralized to the lowest possible level, and given as close to the patient's home as is possible by:
 - (a) Developing and teaching home care. Since most patients are at home and many institutions are sited far away, relatives or village health workers should treat ulcers. The advantage of this would be quicker treatment, the patients remaining in their homes and developing a better understanding and commitment to the process of care.
 - (b) Encouraging minor procedures to be performed in peripheral leprosy or general clinics either by leprosy control staff or by trained general health staff.
 - (c) Engaging and training staff in peripheral general hospitals to help with the basic surgical management of ulcers and septic conditions.

² Leprosy workers should be taught to differentiate between ulcers suitable for treatment in the field and those which need to be referred to a hospital for possible surgery.

Physical examination has to be taught and practised. Probing is an excellent way of differentiating between superficial and deep ulcers.^{38,51} A simple probe can be made from an unfolded paperclip, sterilized by the flame of the spiritlamp that is used for skin-smear taking, which is normally carried on field trips.

3 Methods of immobilization should be developed and implemented that allow the patients a certain level of mobility but still immobilize the ulcers.

Encouragingly enough there are a number of publications describing different casting techniques, carrying out clinical trials on ulcer healing⁵¹⁻⁵³ and studies on pressure reduction.⁵⁴⁻⁵⁷

The moulded double-rocker plaster shoe is a cheap and simple device which patients accept. It is reasonably effective compared to the classical below knee close contact plaster cast.⁵⁸⁻⁶¹ Both methods are useful in the ambulatory treatment of ulcers.

Several different new materials that have been tested are more durable than conventional plaster (POP), but POP is easier to apply. Self-setting materials like PU-resin-impregnated bandages have better mechanical and physical properties but are significantly more expensive and potentially more hazardous than POP.⁵⁴

Treatment of complicated ulcers

First, the patient must be prevented from walking on the ulcer, and second it must be decided where the patient should be treated.

Most of the cases will need surgical intervention, and depending on the resources and levels of staff training, many patients can be treated in peripheral clinics. We are dealing with simple surgical procedures all aiming at eliminating infection, by drainage and removal of infected tissue, and restoring the plantar surface.

Cases suffering from serious septic conditions or from tarsal disintegration or high risk ulcers need to be treated by a worker who has had training in leprosy surgery.^{63,64}

Grossly deformed feet may need extensive surgical correction and special footwear to prevent further ulcers. This treatment is very time-consuming, and means that the surgeon is occupied a long time treating a small number of patients.^{38,65}

Procedures involving the bone require a lengthy healing time, but this may be necessary in order to prevent the ulcers recurring. Replacement of lost plantar skin is difficult and controversial since the skin outside the plantar surface is inferior to plantar skin.⁶⁷⁻⁶⁹

The damaged heal is the most difficult disability to treat, since it is almost impossible to make a shoe for a foot with a destroyed heel. There are a few publications that recommend a flexor digitorum brevis myocutaneous flap or a fasciocutaneous island flap from the instep of the sole for covering the heel. The reported results are good, but the procedures are time consuming and require training and experience. It is rewarding for the workers involved to observe how well the skin from the instep integrates with the heel skin.^{71,72}

A number of flap techniques that have been reported take skin not only from the surroundings but also from further away on the body.⁷³⁻⁷⁵ Free flaps have been used with success. Since, generally speaking, flap techniques are difficult and time consuming

because microvascular procedures are involved, very few centres will have the skills and resources to perform them. It should also be noted that flaps are insensitive and have a lower threshold for bearing weight than the original skin of the foot, and therefore can easily ulcerate.

After surgery, when healing has started, immobilization in a plaster cast, e.g. a window cast,⁵⁸ and/or with a pair of crutches, may be sufficient. This allows the patient to remain mobile during the period of aftercare.

After the healing of major septic surgery it is important to protect the foot. The patient should not be allowed to put any weight on his/her foot before proper footwear is available. At this stage pressure registration to help design the shoe can be of value.^{38,66}

Malignant degeneration occasionally occurs in plantar ulcers. It has been pointed out that a high proportion of the malignant looking ulcers have been grades of pseudo-epitheliomatous hyperplasia (or epithelioma cuniculatum), which can be treated by local excision. Malignancies are usually of a fairly low grade and can be treated by conservative resections and amputations.⁷⁶⁻⁷⁸

Some feet do not allow any weight bearing. Different devices for taking the weight off the foot have been designed, such as cuff bearing and patella tendon bearing (PTB) devices. They are all difficult to make, and difficult for the patients to wear. Often the only remaining solution is an amputation. Many patients are definitely much more mobile and do better after amputation and limb-fitting.⁷⁹

Programmes for artificial limbs require good material and worker skills. The period between an amputation and the application of the prosthesis can be long and frustrating for the patient. In the ALERT programme there has been a positive reaction to a temporary prosthesis given shortly after amputation. At first the acceptance of the temporary prosthesis is simple, cheap, and easy to produce. It consists of a plastazote-lined PTB-shell with a wooden peg, with rubber on its end. It is intended to be worn for 6 months, when it can be easily adjusted as required. When the time comes to have the permanent prosthesis fitted, the patient's stump is conditioned and his/her walking training easier.

Ideally the best preventive measure would be to restore sensation to the sole of the foot. Previous attempts to restore sensation in the foot have not been very successful,⁸⁰ because, for a start, proper material for nerve grafting has not been available. With the introduction of the freeze-thawed muscle graft, grafting has now become possible. Ongoing clinical trials have shown some interesting and promising results. Sensory qualities are returning after resection and grafting of part of the posterior tibial and median nerves.^{17,18}

Conclusion

In theory it is known why, how, and where ulcers form, and there is experience in basic ulcer management, methods which are simple to understand, cheap and easy to perform and which have been tested and practised for many years. It is recognized that the implementation of such knowledge for the benefit of the patient in need of ulcer prevention and ulcer care is a management problem, and also that many leprosy control units do not have fully-developed programmes for ulcer management. It is a matter of

teamwork, in which leprosy control, POD and PRS staff must participate in a wellplanned and integrated manner.

The necessary infrastructure is basically present in most leprosy control programmes. The implementation, or upgrading, of such an integrated programme is a matter of training of and co-operation with existing staff. In future leprosy institutions should function as training centres for POD and PRS. Integration with general health care services should be promoted. Leprosy surgeons should participate in these developments as trainers.⁸¹

Recent research seems to have concentrated on refining and deepening our understanding of the pathomechanisms for ulcer formation. Unfortunately, treatment research has concentrated mainly on models suitable for the more-developed countries, where the majority of leprosy patients do not live. In a period where the prevention of disability and ulcer management seem to be living in the shadow of multidrug therapy, research projects studying the problems of disability prevention and ulcer management should be promoted. Research aiming at preserving sensation should also be given priority.

It is unlikely that any new leprosy hospitals of the traditional model will be built in the future. New programmes will have to solve their need for hospital beds in other ways. Decentralized ulcer management requires only a modest amount of not very expensive equipment. Existing resources in general health care could be mobilized. In a decentralized model more patients, now without access to ulcer care, can be helped at an earlier stage, thereby preventing complete destruction.

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References

- ¹ Brand PW, Fritschi EP. Rehabilitation in leprosy. *Leprosy. Medicine in the Tropics.*
- ² Jopling WH. Leprosy stigma. Lepr Rev, 1991; 62: 1-12.
- ³ Srinivasan H. Nerve damage, surgery and rehabilitation in leprosy. Trop Med Parasitol, 1990; 41: 347-9.
- ⁴ Pereira JH, Palande DD, Gschmeissner SE. Mycobacteria in nerve trunks of long-term treated leprosy patients. Lepr Rev, 1991; 62: 134-42.
- ⁵ Ross P, Waters MFR. Reversal reactions in leprosy and their management. Lepr Rev, 1991; 62: 113–21.
- ⁶ de Rijk A. ALERT. Personal communication.
- ⁷ Ponnighaus IM, Boerrigter G, Fine PEM, Ponnighaus LM, Russel J. Disabilities in leprosy patients ascertained in a total population survey in Karonga District, Northern Malawi. *Lepr Rev*, 1990; **61**: 366–74.
 ⁸ Watson JM. Disability control in a leprosy control programme. *Lepr Rev*, 1989; **60**: 169–77.
- ⁶ Becx-Bleumink M, Berhe D, Mannetje W't. The management of nerve damage in the leprosy control services. Lepr Rev. 1990; 61: 1–11.
- ¹⁰ Watson JM. Preventing disability in leprosy patients. The Leprosy Mission.
- ¹¹ Bell-Krotoski J. Hand screen for early detection and monitoring of peripheral neuropathy. Part II. *The Star*, 1992; Vol. 5, No. 3, 3–7.
- ¹² Bell-Krotoski J, Tomancik E. The repeatability of testing with Semms-Weinstein monofilaments. J Hand Surg, 1987; **12A:** 155-61.
- ¹³ Birke JA, Sims DS. Plantar sensory threshold in the ulcerative foot. *Lepr Rev*, 1986; **57:** 261–7.
- ¹⁴ Hammond CJ, Klenerman P. Protective sensation in the foot in leprosy. Lepr Rev, 1988; 59: 147-354.
- ¹⁵ Currie HC. ALERT: Personal communication.

- ¹⁶ Klenerman P, Hammond C, Kulkarni VN, Mehta JM. Vibration sense and tarsal disintegration. *Ind J Lepr*, 1990; 62: 422-8.
- ¹⁷ Pereira JH, Palande DD, Subramanian A, Narayanakumar TS, Curtis J, Turk JL. Denatured autologous muscle graft in leprosy. *Lancet*, 1991; **338**: 1239–40.
- ¹⁸ Kazen RO. Unpublished material. ALERT.
- ¹⁹ Sankaran B. Biomechanics of normal foot, gaitanalysis and clinical applications. Proceedings of the National Workshop on Footcare in Leprosy. April 1990, Bombay.
- ²⁰ Wele DS. Health education for the successful implementation of MDT. Ind J Lepr, 1990; 62: 3.
- ²¹ Price EW. The care of the foot. Leprosy in Theory and Practice. Bristol: John Wright & Sons Ltd. 1964.
- ²² Harris RI, Beath T. (1947) 'Canadian Foot Survey', Nat. Res. Council Canada. Appendix B.
 ²³ Patil KM, Srinath MS. New image-processing system for analysis, display and measurement of static and
- dynamic foot pressures. Med & Biol Eng & Comput, 1990; 28: 416–22.
- ²⁴ Arcan M, Brull MA. A fundamental characteristic of the human body and foot, the foot-ground pressure pattern. J Biomechanics, 1976; Vol. 9: 453–7.
- ²⁵ Bransby-Zachary MAP, Stother IG, Wilkinson RW. Peak pressures in the forefoot. J Bone Joint Surg [Br], 1990; 72-B: 718-21.
- ²⁶ Sabato S, Yosipovitch Z, Simkin A, Sheskin J. Plantar trophic ulcers in patients with leprosy. Int Orthopaedics, 1982; 26: 203-8.
- ²⁷ Boulton AJM, Hardisty CA, Betts RP, Frank CI, Worth RC, Ward JD, Duckworth T. Dynamic foot pressure and other studies as diagnostic and management aids in diabetic neuropathy. *Diabetes Care*, 1983; 6: 26-33.
- ²⁸ Pollard JP, Le Quesne LP, Tappin JW. Forces under the foot. J Biomed Eng, 1983; 5: 37-40.
- ²⁹ Hughes J, Clark P, Klenerman L. The importance of the toes in walking. J Bone Joint Surg [Br], 1990; 72-B: 245-51.
- ³⁰ Sims DS Jr, Cavanagh PR, Ulbrecht JS. Risk factors in the diabetic foot. *Phys Ther*, 1988; **12**: 1887–1901.
 ³¹ Srinivasan H. Plantar ulcers and corrective surgery. Determining factors in localization of foot-ulcers in
- leprosy patients. Lepr India, Supplement No. 3A, 1965.
- ³² Srinivasan H. Heel ulcers in leprosy patients. *Lepr India*, 1976; **48:** 355–61.
- ³³ Srinivasan H. Do we need trials of agents alleged to improve healing of plantar ulcers? *Lepr Rev*, 1989; **60**: 278–82.
- ³⁴ Carley PJ, Wainapel SF. Electrotherapy for acceleration of wound healing: Low intensity direct current. Arch Phys Med Rehabil, 1985; 66: 442-6.
- ³⁵ Kloth LC, Feedar JA. Acceleration of wound healing with high voltage monophasic, pulsed current. *Phys Ther*, 1988; **68**: 503–8.
- ³⁶ Palande DD, de Severy C, Rajagopalan MS. Plantar ulcers with osteomyelitis underneath. A bacteriological study. *Lepr India*, 1977; **49**: 322–9.
- ³⁷ George M, Bhatia VN, Kar HK, Roy RG. Anaerobic flora in trophic ulcers in leprosy patients. *Ind J Lepr*, 1985; **57**: 334-40.
- ³⁸ Duffy JC, Patout CA, Jr. Management of the insensitive foot in diabetes: Lessons learned from Hansen's disease. *Military Medicine*, 1990; **155**: 575-9.
- ³⁹ Ganlöv G. Unpublished material. ALERT.
- ⁴⁰ Noorden SK. A look at world leprosy. Lepr Rev, 1991; 62: 72-86.
- ⁴¹ Srinivasan H. *Background notes on foot-care in leprosy patients*. The National Workshop on foot care in leprosy. Bombay 1990.
- ⁴² Rao KS. Neuritis and foot deformities in leprosy. The National Workshop on foot care in leprosy. Bombay 1990.
- ⁴³ Palande D. Some clinical and laboratory signs indicating external compression of a nerve trunk in leprosy: details and rationale. *Lepr Rev*, 1976; **47**: 35–9.
- ⁴⁴ Palande D, Muthuraj M. Surgical decompression of posterior tibial neurovascular complex in treatment of certain chronic plantar ulcers and posterior tibial neuritis in leprosy. *Int J Lepr*, 1975; **43**: 36–40.
- ⁴⁵ Carayon A, Giraudeau P, Disy P. La composante neuro-vasculaire dans les ulceres plantaires de la lepre. *Medecine Tropicale*, 1978; **38**: 453-78.
- ⁴⁶ Premkumar R, Pannikar VK, Fritschi E. Foot soaks for callosities and fissures. *Ind J Lepr*, 1990; **62**: 478–82.
- ⁴⁷ Brand PW. Insensitive feet. A practical handbook on foot problems in leprosy. The Leprosy Mission, 1986.
- ⁴⁸ Antia NH. Plastic footwear for leprosy. Lepr Rev, 1990; **61:** 73-8.
- ⁴⁹ Kulkarni VN, Antia NH, Mehta JM. Newer design in foot-wear for leprosy patients. Ind J Lepr, 1990; 62: 483-7.
- ⁵⁰ Srinivasan H. Newer tasks for leprosy workers. Ind J Lepr, 1990; 62: 409-15.
- ⁵¹ Birke JA, Novick A, Graham SL, Coleman WC, Brasseaux DM. Methods of treating plantar ulcers. *Physical Therapy*, 1991; **71**: 116–22.
- ⁵² Coleman WC, Brand PW, Birke JA. The total contact cast. J.A.P.A., 1984; 74: 548-552.

- ⁵³ Mueller MJ, Diamond JE, Sinacore DR, Delitto A, Blair VP III, Drury DA, Rose SJ. The total contact casting in treatment of diabetic plantar ulcers. *Diabetes Care*, 1989; 12: 384–8.
- ⁵⁴ Wytch R, Ashcroft GP, Ledingham WM, Wardlaw D, Ritchie IK. Modern splinting bandage. J Bone Joint Surg [Br], 1991; 73-B: 88-91.
- ⁵⁵ Kaplan M, Gelber RH. Care of plantar ulcerations: comparing applications, materials and non-casting. Lepr Rev, 1988; **59**: 59–66.
- ⁵⁶ Birke JA, Sims DS Jr, Buford WL. Walking casts: effect on plantar foot pressures. J of Rehabilitation Research and Development, 22: 18-22.
- ⁵⁷ Novick A, Birke JA, Graham SL, Koziatek E. Effect of a walking splint and total contact cast on plantar forces. J Prosthet and Orthot, 3: 168–78.
- ⁵⁸ Borssén B, Lithner F. Plastar casts in the management of advanced ischaemic and neuropathic diabetic foot lesions. *Diabetic Medicine*, 1989; 6: 720-3.
- ⁵⁹ Pring DJ, Casiebanca N. Simple plantar ulcers treated by below-knee plaster and moulded double-rocker plaster shoe—a comparative study. *Lepr Rev*, 1982; **53**: 261–4.
- ⁶⁰ Joseph B, Joshua S, Fritschi EP. The moulded double-rocker plaster shoe in the field treatment of plantar ulcer. Lepr Rev, 1983; 54: 39-44.
- ⁶¹ Diamond JE, Sinacore DS, Mueller MJ. Moulded double-rocker plaster shoe for healing a diabetic ulcer. *Physical Therapy*, 1987; **67**: 1550-2.
- ⁶² Job CK. Nerve damage in leprosy. XII leprosy congress, state-of-the-art lecture. Int J Lepr, 1989; 57: 532-9.
- ⁶³ Mehta JM, Sane SB. *Rehabilitation of foot handicaps*. The National Workshop on foot care in leprosy. Bombay 1990.
- ⁶⁴ Carayon A, Chevallard A. Evolution des lésions osseuses du pied lépreux vers la dislocation du tarse et du métatarse. *Revue de chirugie Orthopédique*, 1990; **76:** 579-82.
- ⁶⁵ Lang-Stevenson AI, Sharrard WJW, Betts RP, Duckworth T. Neuropathic ulcers of the foot. J. Bone Joint Surg [Br], 1985; 67-B: 438–42.
- ⁶⁶ Stein H, Simkin A, Joseph K. The foot-ground pressure distribution following triple arthrodesis. Arch Orthop Traumat Surg, 1981; 98: 263–9.
- ⁶⁷ Palande DD, Rajoo DP, Rajagopalan MS. Skin grafting for plantar ulcers in leprosy. *Lepr India*, 1976; 48: 739–43 (Suppl.).
- ⁶⁸ Shaw WW, Hidalgo DA. Anatomic basis of plantar flap design: Clinical application, with discussion by G. Reading. *Plast Reconstr Surg*, 1986; **78**: 637–51.
- ⁶⁹ Curtin JW. Functional surgery for intractable conditions of the sole of the foot. *Plast Reconstr Surg*, 1977; **59**: 806–11.
- ⁷⁰ Morrison WA, McK Crabb D, McC O'Brien B, Jenkins A. The instep of the foot as a fasciocutaneous island and as a free flap for heel defects, with discussion by D. H. Harrison. *Plast Reconstr Surg*, 1983; **72:** 56–65.
- ⁷¹ Shah A, Pandit S. Reconstruction of the heel with chronic ulceration with flexor digitorum brevis myocutaneous flap. *Lepr Rev*, 1985; **56**: 4–48.
- ⁷² Gravem PE. Heel ulcer in leprosy treated with fasciocutaneous island flap from the instep of the sole. Scand J Plast Reconstr Hand Surg, 1991; 25: 155-60.
- ⁷³ Amarante J, Costa H, Reis J, Soares R. A new distally based fasciocutaneous flap of the leg. Br J Plast Surg, 1986; **39**: 338–40.
- ⁷⁴ Sakai S, Terayama I. Modification of the island subcutaneous pedicle flap for the reconstruction of defects of the sole of the foot. Br J Plast Surg, 1991; 44: 179-82.
- ⁷⁵ Hong G, Steffens K, Wang FB. Reconstruction of the lower leg and foot with the reverse pedicle posterior tibial fasciocutaneous flap. Br J Plast Surg, 1989; 42: 512–16.
- ⁷⁶ Srinivasan H, Desikan KV. Cauliflower growths in neuropathic plantar ulcers in leprosy patients. J Bone Joint Surg, 1971; 53-A: 123-32.
- ⁷⁷ Ochsner PE, Hausman R, Olsthoorn PGM. Epithelioma cuniculatum developing in a neuropathic ulcer of leprous etiology. Arch Orthop Traumat Surg, 1979; 94: 227–31.
- ⁷⁸ Girdhhar M, Mohan L, Arora SK, Gupta M, Basu PK, Mishra SD, Mukhija RD. Squamous cell carcinoma developing in trophic ulcers of leprosy—report of 2 cases. *Ind J Lepr*, 1990; **62**: 126–8.
- ⁷⁹ Fleshmann K, Hill P, Fritschi EP. Rehabilitation by amputation. Lepr Rev, 1976; 47: 41-9.
- ⁸⁰ Mcleod JG, Hargrave JC, Gye RS, Pollard JD, Walsh JC, Little JM, Booth GC. Nerve grafting in leprosy. Brain, 1975; 98: 203–12.
- ⁸¹ Virmond M, Duerksen F, Goncalves A. Report and evaluation of Brazilian experience in the rehabilitation of patients with leprosy. *Lepr Rev*, 1989; **60:** 214–20.