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MAINLY A SYMPOSIUM ON PLANTAR ULCERS

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Marianum *Antigen*

Letter to the Editor

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Dr. C. M. Ross, o.b.e.

Editorial

I.—DEATH OF A GREAT LEPROLOGIST

DR. CHARLES M. ROSS died on 24th June, 1964. The death of this good, great man has deeply affec

Obituary notices prepared by Dr. T. F. Davey and ourselves, reprinted from the *British Medical Journal*, 25th July, 1964, page 254, by the kind permission of the Editor.

C. M. ROSS, O.B.E., M.B., B.CH., B.A.O., S.T.M., C.P.H.

The unexpected death of DR. C. M. ROSS at Nairobi on 24th June at the age of 61 was a grevious loss to a wide circle of friends and to the cause of leprosy control.

CHARLES MCCONAGHY ROSS was born in 1903, a member of a family well known in medical circles in Northern Ireland. He graduated M.B., B.CH., B.A.P. at Belfast in 1926, and after working for two years in the Dublin Medical Mission he offered his services to the Qua Iboe Mission, being appointed medical officer at the Etinan Hospital in south-east Nigeria in 1928. In addition to an extensive surgical practice his responsibilities there also involved the care of leprosy patients at the neighbouring leprosarium of Ekpene Obom, and the great need of these sufferers led him to the decision to specialize in leprosy.

In 1940 he offered his services to BELRA and became my colleague at Uzuakoli Leprosy Settlement. At that time rural leprosy control had begun to develop in Eastern Nigeria, and the possibility of its widespread expansion was opening up. In this pioneering atmosphere his great gifts found rich fulfilment. His boundless energy and common sense, his professional skill, his transparent sincerity, and his love of the countryside and its people not only endeared him to innumerable patients but inspired confidence and respect among chiefs and local authorities.

Quite apart from sufferers from leprosy, his professional services were much sought after by members of the general public, both African and European, and in the midst of an exceptionally busy life his courtesy and skill were always at their disposal. In 1947 he was appointed medical superintendent of the Rivers Area of the Nigeria Leprosy Service. Much of the planning, both of a central leprosarium and of a widespread anti-leprosy organization in the Niger Delta, devolved on him, and development was so rapid that ten years later leprosy had become of secondary importance in the area. In 1953 he had already accepted the post of specialist leprologist in the Northern Region Leprosy Service. Here he faced a formidable task. A few small stereotyped Government leprosaria and a number of Mission leprosaria were only touching the fringe of a vast problem, aggravated by religious sanctions and the wide expanse and large population of the territory. Fact-finding surveys led to unconventional methods of leprosy control, in which he both sought and

won the co-operation of Government and Mission doctors, nursing sisters, and lay-workers on an unprecedented scale, so much so that when he retired in 1961 200,000 leprosy patients were receiving treatment and a nation-wide campaign was in progress.

His pioneering spirit could not be satisfied with retirement, and in 1963 he became the director of leprosy research in the East African Common Services Organization. His contributions to leprosy journals were numerous. Over the years his unique experience of leprosy survey work enabled him to undertake, at the request of the Governments concerned, surveys in Sierra Leone and Gambia, on which subsequent leprosy control programmes were formulated.

Workers in tropical public health with the indomitable spirit of Charles Ross can ill be spared, but there are many who, while mourning his loss, remember with admiration and gratitude those personal qualities which expressed so nobly the Christian faith which meant so much to him.

To his wife, his constant and equally devoted companion over many years, we offer our deepest sympathy.

T. F. DAVEY

DR. J. ROSS INNES *writes:*

DR. ROSS's leprosy work was particularly notable for reliable leprosy surveys in Gambia, Sierra Leone, and Bornu in Northern Nigeria. In December 1952 he went to Kaduna as leprologist to Northern Nigeria. There his work was stupendous. He succeeded in training paramedical workers and set up a dispensary and leprosarium control scheme for over 200,000 patients. I personally visited Northern Nigeria and saw his work at first hand, and the energy and hard work and practical effectiveness of the Northern Nigeria campaign under Dr. Ross were outstanding. He also went to Ceylon under WHO as adviser in the leprosy programme there.

The scientific contributions of Dr. Charles Ross were numerous and valuable in trials of certain new drugs. Above all he had a flair for reliable assessment of leprosy incidence. These surveys were done with a small number of personnel, including his wife, who brought her nursing knowledge and practical knowledge of leprosy to the task. The result was that if one really wanted to know the leprosy incidence of a country or area Dr. Charles Ross having surveyed it gave the truth.

His whole life was directed, therefore, to leprosy investigation and relief, and countless patients in Africa will now be grateful for his hard work and efficiency. As a colleague Dr. Charles Ross was known to all leprologists for his integrity, grace, and lovableness.

DR. T. F. DAVEY who knew DR. CHARLES ROSS very well in Nigeria has kindly written the following additional intimate reminiscences.

DR. C. M. ROSS – *A colleague remembers*

I first met Dr. and Mrs. Ross at Uzuakoli railway station late one night

in February 1940, when in the service of BELRA they came to join our small staff. In those days when leprosy work seemed to attract more than its share of unusual people one met newcomers with some trepidation, but here were colleagues after our own heart, bringing with them several years of experience in medical work at Etinan, E. Nigeria. They arrived when village leprosy control work had begun to expand, following a visit by DR. E. MUIR in 1939. At the time of their arrival opposition to leprosy work was still considerable in many areas, and nothing would wear it down but the personal visits of a leprologist with understanding and great friendliness. Charles Ross was ideally suited for this work. He was first and foremost a pioneer, a quality he shared with his wife, and for many years they were never more happy than when on tour in some deeply rural area, undertaking surveys, establishing treatment clinics, and ministering to the sick.

During the years of our colleagueship we used to alternate our medical duties weekly, so that one of us would be on tour, the other at the Settlement. Touring was no picnic, particularly during the difficult war years, but regularly on alternate Mondays Dr. and Mrs. Ross would set off in an overloaded kit-car, with African nurse or laboratory technician, houseboy, their combined camping equipment, medicines and dressings, and sometimes with a bicycle tied on the side of the car. Travelling over rough unmade roads, sometimes even constructed by the people for our use, they would set up house in a remote unfurnished rest-house, returning at the end of the week with some new advance made, new patients found and treated, the promise of a new clinic, or a site offered for a segregation village. They usually reached their objective, for Charles Ross was no mean motor mechanic, and when they returned there would likely be a very sick patient squeezed into the car somewhere, while during succeeding days a little procession of patients would arrive with their relatives, to receive the help it had proved impossible to give at a lonely outstation. This was work he loved, and amid the heavy administrative responsibilities which later years brought to him it never lost its place in his heart.

Charles Ross's skill as a surgeon was quickly discovered by the local people, and his fame spread far and wide. With Mrs. Ross a highly competent midwife, they made an unusual combination. Obstetric emergencies were brought from miles around, often in extremis, but the life of every mother saved was regarded by the people as a miracle, and the goodwill and personal affection so engendered greatly encouraged, not only the spread of leprosy work, but a new outlook in the area on modern medicine generally. Apart from the Settlement there was for many years only one other medical centre with a resident doctor to meet the needs of an entire county, and it is therefore not surprising that when at 'home', Dr. Ross usually found his hands full, succouring not only leprosy patients, but members of the general public with grave surgical emergencies. This work involved many a night call.

It was characteristic of him that his patients felt that all his interest

and care were being given to them personally. This was the quality of his friendliness, and though it made heavy demands upon him, his physical energy and resilience seemed always sufficient. Children loved him, and our Nigerian staff were deeply attached to him. They appreciated his sense of humour, his sympathetic understanding in their personal troubles, and his utter reliability.

He was a very sociable person, and loved a party. His impersonations of mutual friends, amusing but never barbed, are not easily forgotten. A loyal Ulsterman, he always celebrated Orange Day with a band and a miniature procession, to the mystification of the patients, who out of their regard for him joined in for the fun of it. Endowed with a robust physique, it is not surprising that he enjoyed sport. He saved many a goal while playing in the staff soccer team.

More than all, one treasures the memory of him as an intimate personal friend, with whom hopes and fears could be shared, and common ideals find expression. His enjoyment of life was infectious, and many are the richer for having known him.

T. F. DAVEY

II.—This issue of *Leprosy Review* is by way of being a Symposium on Plantar Ulcer. Several workers who are specially concerned with Plantar Ulcer have responded to our request to contribute to this subject and their papers are printed in this issue. It was necessary to set a limit of time for such papers and naturally those papers are included which arrived in time for printing. By way of preliminary remarks, E. W. PRICE has kindly sent useful introductory remarks on Plantar Ulcer which follow here.

Ulcer on the sole of the foot is one of the commonest disablements of leprosy; it also causes more difficulty to leprosy workers than other lesions because of its persistence and of the number of patients involved. Half or more of the beds available in a leprosy hospital are often occupied in this way and the persistence of the ulcer makes it difficult to send them home even when the leprosy itself is arrested.

It is the purpose of this symposium to clarify the causes of ulcer on the sole, and to give clear indications for treatment. The condition is amenable to modern therapy.

Ulcers on the sole of the foot can be simply classified as follows:

(i) Ulcers which are infected wounds – caused by such things as thorns, nails in footwear, simple infection.
(ii) Plantar ulcers – a specific lesion of the mobile neuropathic foot.
(iii) Ulcers which occur due to friction and pressure on the prominent part of a deformed and rigid foot.

The cause, prognosis and treatment of each of these types is different, and failure is due to the application of wrong treatment.

The specific lesion of the walking and mobile neuropathic foot is described as 'plantar ulcer'; other terms are 'neuropathic ulcer', 'trophic ulcer', 'perforating ulcer'. The term is used to underline the relation to

walking and it can only occur on the sole. No term is completely satisfactory, but 'plantar ulcer' as a term is similar to that of 'duodenal ulcer' where it is generally agreed that it refers to a specific lesion and does not include such ulceration as caused by a fish-bone or by a gall-stone.

In the present state of our knowledge, *all plantar ulcers should become healed and remain healed with modern treatment.* They represent a problem in nerve damage and not in leprosy *per se;* they may persist long after the leprosy is arrested.

It will be seen, from the following pages, that modern treatment includes the provision of *special footwear.* This can be provided at a cost of US $1.00 per pair in most developing countries. It will always be found that some patients in a rural community are familiar with working with wood and can readily copy a model; the first technician used by the writer was the local ju-ju carver. To treat plantar ulcer without having footwear available is deficient medical care.

An *X-ray machine,* to diagnose the extent of bone infection, is useful, and is indispensable if the concomitant lesion of neuropathic joint (a frequent association) is to be recognized. But large numbers of ulcers will heal and remain healed without this facility. The same is true of *surgical facilities;* it is sometimes an advantage not to have a knife available to incise into a neuropathic foot!

Plantar ulcer is always preceded by a pre-ulcerative state which is clinically obvious for ten days before ulceration occurs. It should be considered a criticism of the medical attendant if plantar ulceration occurs in a patient under constant care, since it can always be avoided by *regular weekly inspection* of feet under risk – *i.e.* feet with sensory deficit.

Finally plantar ulcer presents a *problem in psychology and social welfare.* Ulcers often persist because the owner fears that healing will make him suitable for discharge from the security of a leprosarium.

It is surprising how patients soon learn how long they can dispose with footwear daily in order to avoid complete healing of an ulcer.

III.—*Errata* in an important paper by DR. P. ONDOUA, MISS M. TH. PROST, and SISTER M. DE LA TRINITE on Clinical and Immunological Results Obtained with the *Marianum* Antigen after more than Ten Years of Therapeutic Use.

Certain unfortunate *errata* arose in this paper which was published in *Leprosy Review,* July, No. 4, 1964, pages 169–173, and the misunderstanding was on the important matter of what the authors really said. We have asked the authors to send a new correct version which appears in this issue on page 297.

Lep. Rev. (1964), **35**, 4A, 239/244

Frequency and Localization of Plantar Perforating Ulcers of Leprosy Patients

MÉDECIN COL. J. LANGUILLON
Director of the Marchoux Institute, Bamako
(*Original article in French, translated by* DR. J. ROSS INNES)

This study was made at the Marchoux Institute with the help of R. H. BOISSAN on more than 3,000 leprosy patients, of whom 403 patients had 1,049 perforating plantar ulcers. These patients were under treatment at the Institute or came to the daily clinics of the Leprosy Service.

It is worth while to note that the population of leprosy patients studied is not necessarily representative of leprosy as disseminated throughout the bush, but those afflicted with plantar ulcers are more numerous at the Institute than in any African region, seeing that hospitalization is a necessity for many of them.

We point out that we have adopted for this study the nomenclature used by LECHAT in his report on bony lesions in the Rome Congress of 1956, namely:

A = heel
B = lateral border of the foot
C = base of 2nd, 3rd, 4th and 5th toes
D = base of the big toe
E = medial border of the foot
F = plantar arch

The results of our enquiry are summarized in Table I as to frequency and localization, and percentages in Table II.

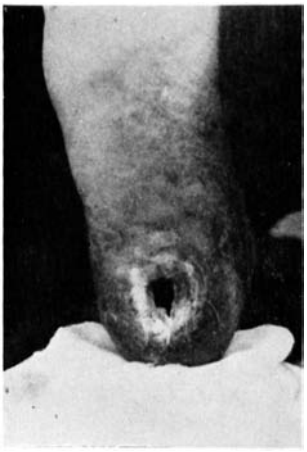
AN ACCOUNT OF THE RESULTS:

1. *Frequency of plantar ulcers*

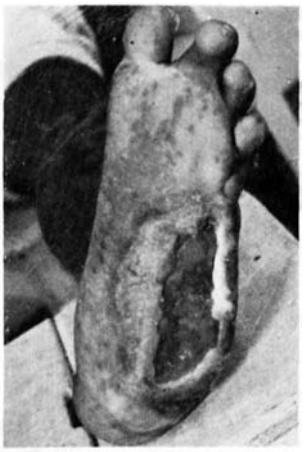
(a) *According to sex* the ulcers are more frequent in the men, 62.6 per cent against 37.4 per cent in women. When one considers the exhausting work of African women and in comparison the three months of work a year by their male companions the results seem astonishing.

In detail, the women did not wear shoes, nor were they pre-immunized better than the men in their sphere.

(b) *Frequency according to age:* In the thousands of leprosy patients examined we have only three times encountered plantar ulcers in those of less than 20 years of age. Also it is possible to state that perforating plantar ulcers are an exclusive complication of the adult, or those patients who are more than 20 years of age. Neuritis has been incriminated in the pathology of plantar ulcers, and in 81.4 per cent of patients nerve lesions are associated. But vascular, and sympathetic nervous pathology, are

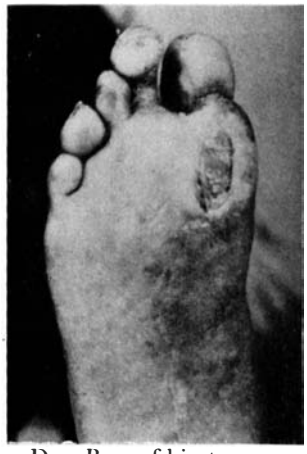


A — Heel

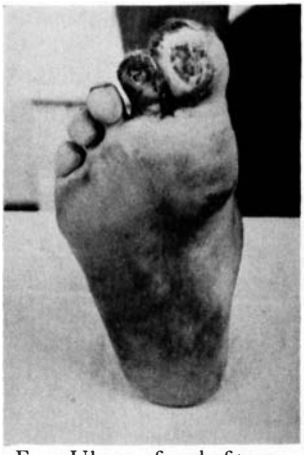


B — Lateral border of the foot

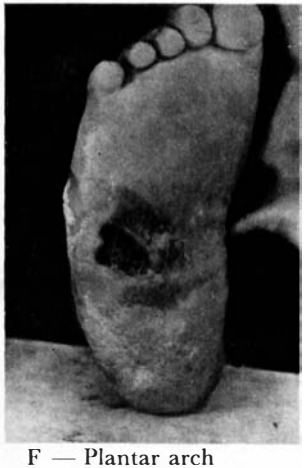
C — Base of fourth toe



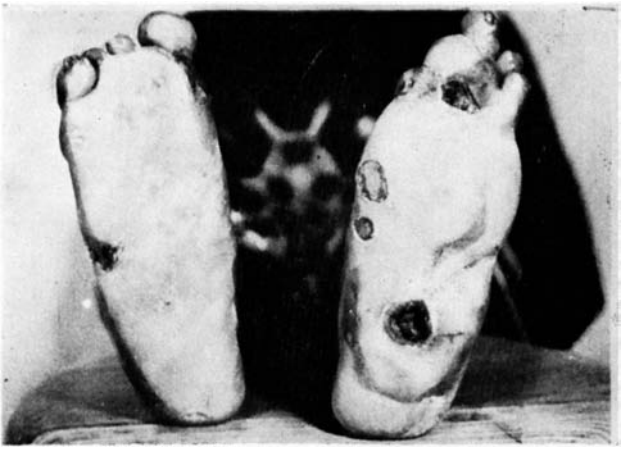
D — Base of big toe



E — Ulcers of pad of toes



F — Plantar arch



B — Lateral border of the foot

equally as important as nerve damage in producing plantar ulcers, even though they supervene slowly in leprosy.

TABLE I

Frequency and Localization

Type		*Total*	*Males*	*Females*
Patients		403	255	148
Plantar ulcers		1,049	657	392
A		126	73	53
B		88	58	30
C		408	254	154
D		399	261	138
E		18	8	10
F		10	3	7
Forefoot	C + D	807	515	292
Mid tarsus	B + E + F	116	69	47
Rear tarsus	A	126	73	53
Victim of	1 plantar ulcer	136	94	42
,, ,,	2 ,, ulcers	86	53	33
,, ,,	3 ,, ,,	77	44	33
,, ,,	4 ,, ,,	50	30	20
,, ,,	5 ,, ,,	31	17	14
,, ,,	6 ,, ,,	14	12	2
,, ,,	7 ,, ,,	4	2	2
,, ,,	8 ,, ,,	2	2	0
,, ,,	9 ,, ,,	3	2	1

TABLE II

Percentages

Type of Plantar Ulcers		*Total*	*Males*	*Females*
A		12.2	11.2	13.9
B		8.3	8.8	7.8
C		38.8	38.5	38.6
D		38.1	39.7	35.4
E		1.7	1.3	2.6
F		0.9	0.5	1.7
Forefoot	C + D	76.8	78.3	74.4
Mid tarsus	B + E + F	11.0	10.6	11.7
Rear tarsus	A	12.2	11.1	13.9

(c) *Frequency according to the form of leprosy:* Here also the results are very definite. In our 403 patients who had plantar ulcers we found 381 tuberculoid or indeterminate patients, and only 22 lepromatous, pointing to the conclusion that plantar ulcers are almost exclusively a complication of the first two types.

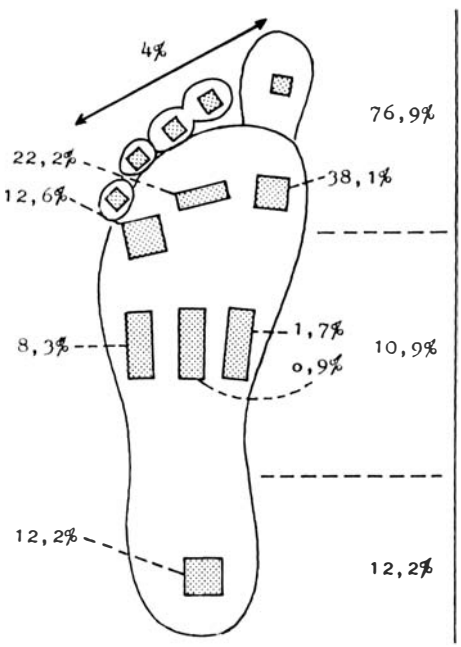
2. *Sites of Plantar Ulcers*

(a) *Ulcers of the base of the toes* are the most frequent. JEANSELME said 'the perforating plantar ulcer of leprosy shows this peculiarity, that it occupies the forefoot almost exclusively', and more recently E. W. PRICE said 'the

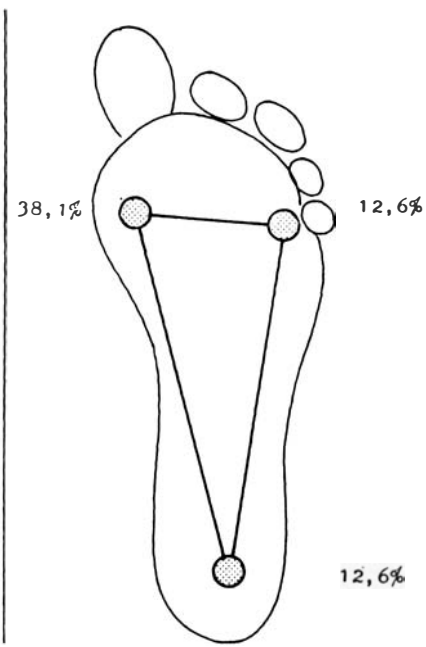
most typical character of these ulcerations is their predominance on the forefoot'. In our data we found 807 plantar ulcers of this kind out of a total of 1,049 patients, that is 76.8 per cent of ulcer patients. As regards to site, sex plays an unimportant role (78.3 per cent in males and 74.4 per cent in females).

In fact the complete list of 807 plantar ulcers should be divided into two fairly equal parts, the big toe, and the base of all the other digits, for we found 38.1 per cent of the big toe and 38.8 per cent of all the others. It should also be noted that in our series, ulcers of toes 2, 3, 4, 5, most often involved the base of the fifth (12.6 per cent out of 38.8 per cent). Further it should be noted that almost all these plantar ulcers responded well to the protection in the sole of the distal head of the respective metatarsal bone. The role attributed to static compression seems very solidly based. While the ulcers occur over the metatarsal heads they especially attack the far extremity of the toes in that little plateau which is really a prolongation of the sole and is particularly traumatized when the foot is raised in the course of walking.

(b) *Ulcers of the midtarsus:* We are grouping together the plantar ulcers of the medial and lateral borders of the foot and those very rare ulcers of the plantar arch. In this group we have only met with 11 per cent of patients. Those of the lateral border of the foot only represent 8.3 per cent of the total number of ulcers. These take up a special shape because of their position with their long axis parallel to the main axis of the foot reaching often 8–10 cm. Ulcers on the medial border of the foot are very



Localization of Plantar Ulcer



Dynamic Pressure Triangle of the Sole

rare and only reach 1.7 per cent, and show the same features as ulcers of the lateral border in the plantar arch. Logically, as a result of its concave form the plantar arch seems to be out of reach of numerous spontaneous traumatic or surgical mutilations, and the central part seems to be the part most attacked by ulcers (0.9 per cent and this occurs equally in males and females).

(c) *Ulcers of the heel* which JEANSELME considers rare and some of which are communicated with a concommitant syphylitic affection, have been met with by us with 12.2 per cent of the cases (those of the midtarsus we have found in 11 per cent). Up to the present time sex seemed to have played a negligible role, but we have found that ulcers of the heel are more frequent in the females (11 per cent men and 14 per cent women). It seems that we should incriminate an ordinary custom in Africa, viz. that the carrying of heavy objects on the head and more so the carrying of children on the back, so that the lumbar arch is increased and the distribution of weight is such that the centre of gravity is situated well behind the normal.

3. *Localization of Plantar Ulcer in Connection with the Biomechanics of Gait*

(a) *Pressure triangle on the sole:* The pressures are equally distributed in halves on the hindfoot, and in the forefoot are divided into five parts, one on the base of each toe. We have found that a dynamic footprint of a healthy subject shows that the foot is remarkably flat when applied to a flat surface.

If the foot is strongly pressed on the sole we afterwards see the silhouette of the heel which is imprinted entire. Then the silhouette elongates in the long direction on the lateral border of the foot from which we see the bases of the five toes and still further forward the imprint of the extremities of the toes separated from the rest of the picture by the flexion of the phalanges. We can test this experience on pressing very lightly the foot on the sole. Behind, there is the heel part of the sole then always the line of the lateral border and in front the line of the bases of the toes at the level of the fifth running to the base of the big toe. Thus the first damage to the sole begins to operate when the triangle goes into operation of the base of the big toe, the base of the fifth toe and the heel. This is what we call the dynamic pressure triangle of the sole and its reality is shown by the percentages of ulcers which we have found to occur as follows:

Base of the big toe	..	38.1 per cent
Base of the fifth toe	..	12.6 per cent
Base of the heel	..	12.2 per cent

thus making a total of 62.9 per cent.

(b) *Application of the foot to the sole during walking:* We cannot do better than apply step by step the explanations given by E. W. PRICE. The gait essentially consists of the rolling of the foot on itself (the walking roll) in which the heel first raises itself from the sole, and pressure is made on all

the proximal extremities of the metatarsals. When these are raised they are replaced by the distal heads. The final push is given all the extremities of the toes. This is the line of the metatarso-phalangeal articulations in the active cooperation of which the most important is flexion. Muscular and tenderness intervention is reduced to its most simple degree. It is in the dynamic pressure triangle of the sole where two thirds (62.9 per cent) of plantar ulcers occur.

SUMMARY

1. In a population of leprosy patients the occurrence of plantar ulcers is about 12 per cent.
2. Males are always more subject to ulcers than females (62.6 per cent males, 27.4 per cent females).
3. Adults alone are affected, and those under 20 years of age, never.
4. Plantar ulcers are peculiar to tuberculoid and indeterminate leprosy patients.
5. Three quarters of plantar ulcers occur on the forefoot.
6. The most numerous of the plantar ulcers are those of the big toe (38.1 per cent). The others are:

Those of the bases of toes 2, 3 and 4	22.2 per cent
Those of the base of the fifth toe	12.6 per cent
Those of the heel	12.6 per cent
Those of the lateral border of the foot	8.3 per cent
Those of the medial border of the foot	1.7 per cent
Those of the plantar arch	0.9 per cent

7. Sex intervenes little in the frequency of distribution of plantar ulcers except in the heel which is more attacked in the female than in the male.
8. The first damage in the sole attacks by choice three sites, namely the base of the big toe, the base of the fifth toe, and the heel, and we ascribe this to the dynamic pressure triangle of the sole.

References

1. BECHELLI et DA SILVA-GUIRAMAEZ. Le mal perforant dans la Lèpre. Etude clinique – *Rev Bras. Lep.*
2. BOSE, D. W. Trophic ulcers in leprosy patients and their managements. *Lep. in Ind*, Juillet 1956, vol. XXVIII, no. 3, pp. 77–79; *Trop Dis. Bull.*, Mai 1957, vol. LIV, no. 5, p. 579.
3. BOUSEFIELD, C. E. The ulcers of leprosy. *Int. J. of Lep.*, Janvier 1938, vol. VI, no. 1.
4. JEANSELME, E. Troubles trophiques de la lèpre. In *La Lèpre*, Doin, édit., pp. 419–444.
5. LIANO, L. Classification des ulcères lépreux et leurs traitements. *Rev. de Derm. Syphil.*, Juin 1943.
6. PRICE, E. W. Studies on plantar ulcers in leprosy I et II. *Lep Rev.*, Avril 1959, vol. XXX, no. 2, pp. 98–105.
7. PRICE, E. W. Studies on plantar ulcers in leprosy III. *Lep. Rev.*, Juillet 1959, vol. XXX, no. 3, pp. 180–183.
8. PRICE, E. W. The etiology of plantar ulcers in leprosy. *Lep. Rev.*, Oct. 1959, vol. XXX, no. 4, pp. 242–248.
9. ROSS, W. F. Etiology and treatment of plantar ulcers. *Lep. Rev.*, 1962, vol. 33, no. 1, pp. 25–40.
10. SILVEIRA, L. M. O mal perforante na lepra. *Rev. Bras. Lep.*, Sao Paulo, 1948, vol. XVI, no. 1, pp. 7–32; et *Trop. Dis. Bull.*, Janv. 1949, vol. XLVI, no. 1.
11. SRINIVASAN, H. Trophic ulcers in leprosy. *Leprosy in India*, Juillet 1963, vol. 25, no. 3, p. 119.
12. VESPOLI, M. Leprotic ulcers: their pathogeny and treatment. *Rev. Bras, Leprol.*, 1939, Sao Paulo, vol. VII, no. 3, pp. 295–307; et *Trop. Dis. Bull.*, Mai 1940, vol. XXXVII, no. 5, p. 341.

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A Classification of Leprosy Foot Deformities

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CLAWSON and SEDDON (1960), in a study of the late sequelae of sciatic palsy found that of the patients who suffered from persistent ulceration, less than half exhibited anaesthesia, but all had some fixed deformity of their feet or toes. They concluded that fixed deformity was a more important cause of ulceration than loss of sensibility. In the leprosy patient, an undeformed anaesthetic foot can be kept free from ulceration by the wearing of appropriate shoes. Deformed anaesthetic feet, on the other hand, exhibit a high incidence of ulceration even with footwear, particularly if the deformity concentrates weight bearing onto a small area of skin.

Observation of a large number of foot patients has revealed certain repetitive patterns of deformity, and has enabled a relatively simple classification to be drawn up. It is the purpose of this paper to describe a classification which may provide a useful basis for description and discussion of an apparently complex spectrum of deformity.

It is usual to classify leprosy deformities as either primary or secondary. Primary deformities are those due to the disease process itself. They include loss of eyebrows and all paralytic deformities, such as are unavoidable unless the case comes under adequate medical treatment early. Secondary deformities are due to causes other than leprosy itself, and are preventable. They usually result from septic complications affecting an anaesthetic extremity. This concept is retained in the present classification, and it should be stressed that most secondary deformities in the foot result directly or indirectly from trophic ulceration. This ulceration frequently is a result of neglect of a primary deformity.

FIG. 1

Natural History of the Deformed Leprosy Foot

Leprosy	*Primary*	*Secondary*	*Destruction*
Neuritis	*Deformity*	*Deformity*	*(Amputation)*
	(and Ulcers)	*(and Ulcers)*	

All established foot deformities are associated with a tendency to ulcerate at a specific site. Without surgical intervention, such feet eventually undergo total destruction. The natural history of the untreated deformed foot is summarized in Fig. (1). No worker who has witnessed this sequence of events, can fail to agree with the conclusion reached by CLAWSON and SEDDON.

Figure (2) outlines the proposed classification. The direct relationship between Groups I and II is indicated by arrows, and the site of specific ulceration of each group is also shown.

FIG. 2

I *Muscle Imbalance*	*Site of Ulcer*	
Primary Deformity		
A Dropped Foot	Antero-lateral Border	
B Dropped Foot (Perone Intact)	Antero-medial Border	
C Claw Toes	Dorsum and Pulp of Toes Metatarsal Heads	
Secondary Deformity		
II *Fixed Deformities*	*Site of Ulcers*	
Shortened Equinus Foot	Anterior Border	
Inverted Foot: Destruction Lateral Ray	Lateral Border	
Shortened Foot with Destruction of Medial Ray	Anterior Border	
Shortened Plantigrade Foot	Anterior Border	
III Deformity due to Joint Neuropathy	Centre of Sole or Instep	
IV Heel Deformities	Heel: Multiple Sinuses	
V Calcaneus Deformity	Heel	

GROUPS I AND II

Dynamic deformities resulting from muscle imbalance: Fixed deformities

(a) Dropped foot, due to lateral popliteal paralysis, results in a high stepping gait in which the foot is violently slapped onto the ground at each step. The antero-lateral border bears the brunt of the impact because unopposed action of the tibialis posterior causes a slight varus

imbalance. It is here that the ulcers associated with the deformity occur. Bone and tissue is shown in the destruction of the lateral ray and shortening of the foot follows, and as the deformity becomes fixed, it passes into Group II.

(b) Most examples of foot drop are complete, but in some instances the peronei are spared. With strong peronei and absent tibialis anterior the lateral balance of the foot is upset in favour of the evertors. The pressures along the antero-medial border are accentuated, and ulcers typically occur here, with destruction of the medial bones. If the peronei later became paralysed, and the foot is first seen when the deformity has become fixed, one may be left wondering why a dropped foot sometimes exhibits this atypical pattern of destruction.

(c) Clawing of the toes is the result of paralysis of the intrinsic muscles of the foot supplied by the posterior tibial nerve. Hyperextension of the metacarpophalangeal joints and flexion of the interphalangeal joints brings the tips of the pulps into contact with the ground, where blisters and ulcers occur, and osteomyelitis of the terminal phalanx. At a later stage, the metacarpophalangeal capsule stretches and the base of the proximal phalanx subluxates dorsally, and ultimately a complete dorsal dislocation occurs, with the acutely clawed toes overriding the heads of the metatarsals. Once subluxation occurs, the toes tend to become stiff in their clawed position, and the deformity passes into Group II. The effects of subluxation are:

(1) The toes are lifted clear of the ground, removing the risk of tip ulceration.

(2) The pressure accepted by the toe pulps during 'push off' in walking cannot now be taken, and passes further back to the metatarsal heads. The extensor digitorum longus tendon shortens and the joint capsule develops contracture.

(3) The fibro-fatty pad (part of the specifically differentiated plantar fascia) underlying the metatarsal head is drawn upwards from beneath the metatarsal heads and thinned out beneath them. The metatarsal heads thus become relatively subcutaneous and subject unprotected skin to high pressure and shear stress during walking. Ulceration occurs under them with a high risk of osteomyelitis. The most characteristic sequel is destruction of the metatarsal heads with shortening of the foot, but the subluxed toes are spared, and remain perched along the anterior margin of the foot.

Group I deformities are passively correctible, but they pass into Group II when the deformity becomes fixed by contracture of the tendo-Achilles, joint capsules, and fibrosis of subcutaneous tissues. X-rays of Group I deformities show a normal foot skeleton, except when a claw toe deformity is seen. In Group II, bone and joint changes are apparent, notably concentric atrophy or absence of the distal parts of the metatarsals. Rarefaction of bone indicates active bone inflammation, and illustrates the mode of evolution of the deformity.

The plantar surface in Group II deformities is frequently reduced in area, but is usually free from scars (except at the anterior or lateral margin). The plantar surface is preserved because it is protected from weight bearing; this is an important fact from the point of view of treatment.

GROUP III

Deformity due to bone and joint neuropathy

Any bone or joint in the foot may undergo destruction as a result of continued function after injury in the absence of pain. This aspect of leprosy deformity is not well documented, but a forthcoming contribution (BRAND and HARRIS) is likely to prove helpful. In the meantime, attention should be drawn to certain clinical features of the condition, and to a not uncommon deformity resulting from it. The foot is swollen and warm, and exhibits excessive subtalar or midtarsal movement, with crepitus. The sole is convex, with flattening of the medial arch, and palpable descent of the head of the talus and navicular. There is contracture of the tendo-Achilles, which draws the posterior tuberosity of the calcaneum backwards and upwards, and contributes to dorsal hinging of the foot at the mid-tarsal plane. A pressure area develops in the centre of the sole or in the instep, and it is here that the ulcers associated with this deformity appear. Descriptively, the deformity has been named 'Boat Foot'. By means of the warmth and swelling, the process may be distinguished from osteomyelitis and cellulitis. X-ray shows crumbling and fragmentation of one or more bones, with roughening of joint surfaces and irregular sclerosis. These appearances may also be attributed to old septic arthritis, but the absence of sinus scars is helpful in making the distinction.

GROUP IV

Heel Deformities

These constitute a separate surgical problem, and are therefore put into a group of their own. Most cases result from ulcers under the heel. The primary ulcer has a particularly obscure aetiology, but once deformity of the calcaneum has occurred, it is perpetuated by localized high pressures, or by rupture of an unyielding scar by shear stresses. The spectrum of deformity in this group ranges from erosions and spurs on the undersurface of the calcaneum, to bizarre deformities resulting from partial or total destruction of the talus, and calcaneum. These patients may have no heel at all, and may be transmitting weight direct from the lower end of the tibia through scarred skin to the ground.

Destruction of these bones may result from osteomyelitis, septic arthritis of the subtalar joint, neuropathic disintegration, or a combination of these processes. Clinically, the heel is scarred, the plantar pulp may be destroyed and multiple sinuses may be present. X-rays show calcaneal irregularities, or partial or complete loss of talus and/or the calcaneum (Fig. 2).

GROUP V

Calcaneus Deformity

This deformity also poses special problems of treatment, and merits a category of its own. It results occasionally from muscle imbalance following foot drop surgery, or to avulsion fracture of the insertion of the tendo-achilles. It has also been seen in calf weakness following poliomyelitis in patients who have also contracted leprosy. It carries with it a high risk of heel ulceration. On X-ray the foot skeleton may be relatively normal, or there may be absorption and flattening of the posterior tuberosity of the calcaneum.

DISCUSSION

This classification embraces the majority of leprosy foot mutilations, but in some instances a combined type of deformity may be seen, for example, a bizarre heel deformity, and a fixed claw toe deformity with shortening of the metatarsals.

SUMMARY

A descriptive classification of leprosy foot deformities is outlined, and the close relationship of deformity to trophic ulceration is stressed. No discussion of management is attempted, but the clinical features of each group are summarized, and their relationships are described.

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Bibliography

CLAWSON, D. K., and SEDDON, H. J. The Late Consequences of Sciatic Nerve Injury. *Journal of Bone and Joint Surgery*, 42-B, 1960, 213.

BRAND, P. W., and HARRIS, J. *Patterns of Disintegration of the Tarsus in the Anaesthetic Foot.* In course of publication.

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Treatment and Prevention of Plantar Ulcers: A Practical Approach

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SECTION I

The term 'plantar ulcer' was introduced by PRICE (5) in 1959 and was defined as 'a 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 a marked tendency to recurrence'. This definition is the result of careful empiric studies. Properly interpreted, treatment and prevention may be deduced from it by the application of simple biological facts.

The great advances in our understanding of the aetidogy and natural history of plantar ulcers in leprosy which we have seen during the last, relatively few years, have been brought about by an application of the knowledge gained in other diseases with neural damage, and from careful studies of the static and dynamic forces at play in the normal and diseased foot. (1, 5, 6, 7, 8, 13.)

Based on this understanding a therapy has been developed, which shows a close relationship to the simple biological treatment of war casualties, developed by TRUETA (15), and CHIEWITZ (3), which is standard knowledge. (1, 7, 8, 9, 13, 16, 18.)

The same application of basic biological principles has led to the development of methods of prevention, easily applicable everywhere. (1, 9, 12, 14, 17, 18.)

The final achievement, or rather challenge to achievement, was boldly stated by the WHO Scientific Meeting on Rehabilitation in Leprosy at Vellore: 'If our present knowledge is *properly* applied, plantar ulceration should not occur'. (18.) (Italics by the author.)

This paper will attempt a presentation of a practical approach to the problem of plantar ulceration as actually met today.

AETIOLOGY AND NATURAL HISTORY

The aetiology is misuse, or rather misunderstood use, of a foot with plantar anaesthesia. The crucial factor would appear to be the 'roll' of the foot in the normal springy gait. (1, 5, 6, 7, 12, 13.) The large majority of ulcers appear to develop from 'necrosis blisters' (6), which are initially sterile and only later, after break-through become infected. (8.) The further development can be described as an evil cycle of 'scar-ulcer-scar'. (1, 6, 7, 8, 13.) If left unchecked, the deeper structures, bone, joint, and tendon, will become involved. The result is not only a shortened and

deformed foot, but a rigid foot with infinitely greater tendency to ulceration. ([1], [8], [9], [12], [13], [14].) A rigid foot has different mechanics from a pliant foot, with a different distribution of plantar ulceration. ([12].)

TREATMENT

A primary necrosis blister is a sterile, but potentially infected lesion. A developed ulcer is an infected wound. A secondary necrosis blister is a potentially, often actually, infected lesion. The hot, swollen foot with scarring is potentially, and very likely actually, infected. ([1], [6], [8], [13].) A peculiar feature, for which so far no satisfactory explanation has been put forward, is the rarity of violent infection, general sepsis and tetanus. ([1].) Successful treatment is based upon the principles laid down by TRUETA ([15]): Rest, elevation, and free drainage. ([1], [7], [8], [9], [13].) Although there can be little doubt that administration of the correct antibiotic in many cases would hasten healing ([13]), the magnitude of the problem and the rapidly developing resistance to all antibiotics make it imperative to avoid antibiotics and metabolic bacteriostatics to the greatest possible extent. ([1].) Experience has taught us that it is extremely difficult to differentiate between dead and living tissue in these cases, and while securing free drainage, it pays to be extremely conservative in excision of apparently dead tissue. ([1–13].)

POST ULCER CARE AND PREVENTION

They are practically identical. The key points are regular inspection, both by the medical officer respectively, physiotherapist and the patient himself, education in detection of warning signs, issue of proper footwear, and constant, intelligent use of the shoes. ([1], [7], [9], [12], [13], [14], [16], [17], [18].)

SURGERY

When active ulceration is present, surgery should be limited to the minimum, only securing free drainage. In this stage, one should never remove anything other than obviously dead tissue. ([1], [7], [13].) The author is still surprised at the number of times evidently dead tissue when left under a plaster of Paris cast, proves to be very vital indeed. The scarred, but hot and swollen foot is a contraindication to any form of surgery. It should be left alone under protection of a plaster of Paris cast. In the scarred, but quiescent foot a number of indications for corrective surgery may be seen. The aim is to remove severe, additional danger points where thin, adherent scar overlies a bony prominence. Several procedures have been recommended, but the general consensus is that such cases must be left for an experienced surgeon to deal with. The results are not encouraging at all. The indiscriminate excision of offending toes and metatarsal bones cannot be condemned strongly enough. ([1], [2], [8].) A radical approach to the extremely bad forefoot with well preserved hindfoot, a common finding, was recommended a long time ago by GASS ([4]). He scarifies the whole offending area in order to stop once and for all the tendency of the

scar to 'creep' backwards. This is a sound approach, but requires careful and expert surgical evaluation and handling.

TECHNIQUES FOR OBTAINING REST

Given free drainage and absence of general sepsis, which must of course be treated with appropriate drugs, any wound will heal if the affected foot is kept permanently elevated and at rest, while cross infection is being prevented. ([1–13].) The magnitude of the problem, and the scarcity of hospital beds has made it imperative to find other methods of securing the necessary rest of the affected leg. While an ulcer is freely draining, and while there is oedema and swelling, bed rest with elevated foot is the only sensible treatment. ([1, 9, 13].) As soon as the ulcer is dry and local oedema has subsided, the patient may be made ambulant in a well fitted plaster of Paris cast with a rocking device preferably a Bohlar iron. The majority of ulcers will heal within six weeks. If indicated, the leg may be kept in plaster cast for any length of time. ([1, 7, 9, 13].) This strongly emphasises the contention that the interruption of the walking roll is the most important part of the treatment. ([7, 13].) The need for a water resistant device for use where it is necessary or desirable to send the patient to his home, while under treatment, led to the development of ' the Karigiri boot' ([13]) where the foot rests on a platform with some shuffling movement on an intervening layer of absorbent felt, the whole device contained by a boot of gauze bandages impregnated with prevulcanised rubber latex. While not quite so effective as a well preserved plaster cast, it is cheaper, and it is waterproof. We have at present difficulties in India about obtaining the correct grade of rubber latex, and experiments are going on to find a suitable preparation. A number of cases, notably necrosis blisters, very small, early ulcers, and cracks of the sole heal remarkably well while the patient is made ambulant on a simple shuffleboard with interposed felt, kept in place round the ankle with lengths of elastoplast. ([13].)

PROTECTIVE FOOTWEAR

The long and winding road of experimentation that has led to our present understanding of this problem was started in 1956 by ROBERTSON (quoted by WARD, [17]). The following principles are recognized: (1) a correct fitting of the shoe ([1, 17]), (2) rigidity of the shoe ([1, 5, 7, 9, 12, 13, 14]), (3) an impervious undersole ([1, 17]), (4) moulding of the inner sole ([1, 12, 14, 17]), (5) a resilient inner sole ([1, 14, 17]).

(1) It is self-evident that careful fitting to avoid 'shoe bites' is absolutely essential in an anaesthetic foot. It is worth remembering that the shoe should be longer than the foot to allow for expansion when weight is placed on the foot.

(2) In the rigid foot with previous bone involvement, this is a *sine qua non*, but even in the pliant foot, the concept of the roll as the causative factor in occurrence of ulcers makes it desirable. This can be achieved either by using a plane or slightly moulded rigid sole with a rocker

incorporated. This is the standard method so far in India. ([1], [17].) This is a cumbersome shoe with poor inherent balance and little attraction for the patient. The wooden clog ([12]) appears to fulfil the demands for a solid, nice looking, acceptable shoe. A number of reasons have so far prevented us from introducing this pattern in India.

(3) When a flexible shoe is prescribed, it becomes important to prevent nails and thorns from perforating and causing damage to the foot. This is usually done by applying an undersole of split tyre rubber. ([1], [17].)

(4) The original idea was to mould the inner sole accurately to the sole of the foot, thus making sure of an even distribution of weight. This is true in the static phase, but cannot be obtained during the dynamic phases of gait. Also if the moulding is less than absolutely perfect, it militates against its very purpose and creates new and dangerous pressure areas. A slight moulding of the instep may be desirable, but this will usually be taken care of by the resilient insole. The metatarsal bar ([1], [17]) is a device that aims at removing pressure from the metatarsal heads and transferring it to a wider area further back. This is a logical concept when the stance is contemplated, but since the roll is the important factor and also because a large number of the feet with fairly extensive scarring of the forefoot and good mid- and hindfoot, which constitute the traditional indication for a metatarsal bar, will also exhibit a certain degree of rigidity, the metatarsal bar loses most, if not all, of its importance.

(5) The rationale of the resilient inner sole is primarily to supply an artificial resiliency where the original resiliency of the sole is lost due to scarring ([1], [13], [17]). By derivation this has been applied as a preventive in anaesthetic, but unscarred feet. Experience has supported this. All workers seem to agree that microcellular rubber of hardness shore 15 degrees is the most universally adoptable material ([1], [13], [17]).

SECTION II

In section I, a highly condensed outline of the principles of the aetiology and natural history of plantar ulcers, and of the logical treatment and prevention of these, has been given. The author is very well aware that neither is this complete, nor is it the final word in this field. This section deals with the practical application of this knowledge, as found in a leprosarium, and as seen at roadside clinics and outpatient departments.

THE LEPROSARIUM

In such an institution, where no full scale ulcer programme has been instituted previously, and where the admission and discharge policy is still largely patterned on the old idea of the asylum, *i.e.* admit when the patient cannot manage the social problems outside – and 'once in, never out', one is likely to find a fairly large number of highly crippled patients with no real desire for improvement in their often apalling ulcer condition. One is also likely to find a number of patients who are beginning to realize that joint attempts from the administrative and medical section are being

made to rehabilitate patients outside the leprosarium. Not infrequently these patients will be rather reluctant to accept real chances of obtaining restored feet. An ulcer is often regarded as a ticket for continued stay in the leprosarium. Even though valiant, and fairly successful advances are being made from asylum to hospital, these problems will be with us to some extent.

The first necessity for a medical officer who initiates a full scale ulcer programme is a complete understanding of the principles and practice of the matter. He must also be fully convinced, not only that it can be done, but that it is one of the most important steps towards a successful medical and social rehabilitation. He must then win the wholehearted support of the administrative section. It must be clearly understood and accepted, that such a programme will call for certain good facilities in staff and equipment. A reasonable number of hospital beds must be available. Initially, it will quite likely play havoc with the smooth running of the leprosy centre, since we still largely depend on patients for carrying out a significant amount of the unskilled and semi-skilled tasks. If the programme is carried out sensibly and efficiently, this phase will not last long. Soon the patients who used to go to work with poorly applied and bulky bandages, will start out for a full day's work with no bandages, but with well fitting, well kept shoes. Undoubtedly, a number of activities will have to be given up in return for more suitable ones. The most remarkable change is seen where the ulcer programme is geared to a developing social and economic rehabilitation programme. The leprosy patient as coolie disappears and the skilled, rehabilitable worker emerges.

Methods of record-keeping must be developed, which are simple and quick, so that there is a good chance of the actual records being kept. Ancillary staff, either technicians or patients, must be secured, and must be imbued with the right spirit. It should be possible to train such ancillary staff to recognize the various types of ulcers, cracks, blisters, etc., and to record them properly under proper supervision from the medical officer or a trained physiotherapist. The application of the various types of standard treatment and the technique of the necessary therapeutic measures such as plaster of Paris boots, Karigiri boots, shuffleboards with felt, must be taught properly during the actual work. A shoe workshop must be set up, that can turn out the necessary number of simple shoes in sufficient numbers. This also should be run with supervision from the medical officer and/or a trained physiotherapist.

The very first step, which may very well be undertaken to convince patients, as well as administrative and ancillary staff, that it can be done, and that it is worthwhile doing, should be a careful selection of a few intelligent, co-operative patients with minimal ulcers on otherwise good feet. Only when it is possible to point to known successful cases, can one expect full support on all sides.

Once this stage has been achieved, a full scale regular foot inspection of every patient in the home is necessary irrespective of the state of the

disease, with careful recording of the findings. At this point, the problem is likely to be too big for the existing facilities, so it will be wise to concentrate on children, younger patients, and those with a good chance of return to normal society. Of prime importance is prompt action on the findings from the foot inspection.

If active sepsis is found, the patient is put to bed with a protective dressing, the foot is kept elevated, and appropriate drug therapy is instituted.

If no active sepsis but local swelling and heat is found, and the ulcer is freely discharging, the patient is put to bed, the foot is kept elevated, therapy is instituted with soaks in lukewarm soapy water, followed by protective dressing with 4 per cent mercurochrome. Provided a careful 'no touch' technique is followed, along with a separate soak bowl for each patient, this has been found to be the simplest and quickest way of preventing cross infection, while preserving free drainage. A notable effect of mercurochrome solutions is that dead tissue is left mummified, thus presenting one with a crisp, inoffensive ulcer, ready for the next step.

When the ulcer is crisp and dry with no localized swelling nor signs of sepsis, it is ready for the ambulatory stage either in a well fitting plaster of Paris cast, or if considered more suitable, a Karigiri boot. These should be left intact for six weeks as a routine. If complete healing is not found, they may be re-applied.

An extremely important part of the regime, unfortunately too often neglected, is measurement for shoes and actual making of the proper shoe so that it is ready the moment the ambulatory device is removed. The idea is to imbue the patient and the staff alike with the understanding that an anaesthetic foot must never touch the ground unprotected by proper footwear. As emphasised, not least by PRICE and ROSS, the ideal footwear is a rigid shoe of the wooden clog variety with resilient insole. Several reasons have made it very difficult to introduce this in India. Quite often we may have to accept that our cobblers cannot make them, and that our patients refuse to wear them. If one can gain the confidence of the patients to the point where they will follow advice about careful walking, it may be perfectly permissible to issue a simple, socially more acceptable chappal (sandal of Indian type).

The whole programme will, in the final assessment, rest on the amount of confidence the medical officer can gain from the staff and patients. Measures of disciplinary action, fines, depriving of privileges, etc., and granting of extra privileges to especially careful patients may all have to be brought into play, but they can never supplant the growth of confidence.

As the programme aimed at the children and younger easily rehabilitable patients gains momentum, and the strain on hospital space and time eases a little, it will become possible to start working with the more recalcitrant ulcers and also the more reluctant patients. It must be understood clearly that while the aim, the possible goal, in the first series, is

eventually to arrive at the point where no ulcers are diagnosed, as a result of the early, preulcerative danger signs being recognized and treated as the potential ulcers they are, the really badly damaged foot can never aim so high. Certainly the incidence of ulcers and particularly the incidence of really bad ulcers can be reduced drastically. But undue optimism is as dangerous and disappointing as undue pessimism.

At this point, the problems of 'cold' surgery will present themselves with increasing frequency and urgency. It is not the intention here to give indications and contraindications which can be found in several readily available papers ([1], [2], [4]). However, it seems timely to underline that 'cold' surgery in the scarred, anaesthetic foot, no less than 'hot' ulcer surgery, is a job for a highly qualified surgeon with wide experience in the field, and with full surgical facilities at his disposal.

An ulcer programme as outlined here is time-consuming, but extremely rewarding, although it is remarkable how soon the strain starts to ease. The gravest danger is to become complacent and think that the problem is over. Unless a constant pressure is kept up *with strict enforcement of regular foot inspection*, prompt action, and steady supervision and encouragement, the situation will deteriorate in a remarkably short time.

THE OUT-PATIENT CLINIC

Since the initial reaction of most patients, certainly in India, usually is extreme reluctance to accept the suggested foot therapy, and since it may not be feasible to introduce it in the early stages anyway for lack of personnel and space, the first and most important problem in the out-patient department is to continue a careful, *regular supervision of the feet and the footwear of the discharged patients*. Every facility should be extended to these patients, including the shoe workshop, for prompt repair of damaged or worn shoes, expecially since the village cobbler invariably will repair with a liberal use of nails. Herein lies one of the real dangers to anaesthetic feet, even if he will accept the shoes of leprosy patients at all for repair.

With the increasing number of discharged patients attending clinics with proper shoes on the feet, and no ulcers, other patients will begin to realize the possibility and later on the benefit of ulcer treatment and prevention. It is very important that facilities are available and are offered promptly when such desire is found.

Usually, the regime will be a short term admission for the specific purpose of ulcer treatment. Certainly a large number of ulcers and pre-ulcerative stages will be seen where it is perfectly possible to apply immediately a plaster of Paris boot, a Karigiri boot or a shuffleboard with felt. There is, however, so great a reluctance to return to the village with these obvious outward stigmata, that until a high degree of confidence has been built up in a large group of patients, advance may be practically impossible.

THE ROADSIDE CLINIC AND THE HOUSE-TO-HOUSE VISITATION

Both offer almost ideal conditions for quick discovery of early damage to anaesthetic feet. One difficulty is that while the patients may be willing to accept unpalatable advice from a senior worker, these are those whom such patients see comparatively rarely. When this understandable reluctance has been overcome, these roadside and domiciliary patients will provide the best possible pabulum of an intensive ulcer programme.

Since a number of ulcers will be found, both in the outpatient department, the roadside clinic, and while visiting the homes of the patients, where it is impractical or simply impossible to give proper treatment without admitting to a well equipped hospital, it is imperative for these branches to have the backing of the hospital section of the leprosarium. Nobody doubts that this could equally well be done in a general hospital, but so far that seems to be an unrealized dream.

SUMMARY

The accepted concepts of the pathogenesis of plantar ulceration in leprosy are presented as the basis for a logical scheme of treatment and prevention. The practical application of this knowledge is described in an ulcer programme in inpatient leprosaria, and under ambulatory conditions.

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References

1. ANDERSEN, J. G. (1961). *Lep. Rev.*, XXXII, 16–27.
2. BRAND, P. W. (1959–1960). Personal communications.
3. CHIEWITZ, O. (1946). Personal communications.
4. GASS, H. H. (1938). 'A Study of the Results of Certain Surgical Procedures in Leprosy,' presented to the Cairo Conference.
5. PRICE, E. W. (1959). *Lep. Rev.*, XXX, 98–105.
6. PRICE, E. W. (1959). *Lep. Rev.*, XXX, 180–183.
7. PRICE, E. W. (1959). *Lep Rev.*, XXX, 242–248.
8. PRICE, E. W. (1960). *Lep Rev.*, XXXI, 97–103.
9. PRICE, E. W. (1960). *Lep. Rev.*, XXXI, 159–171.
10. PRICE, E. W. (1961). *Lep. Rev.*, XXXII, 108–116.
11. PRICE, E. W. (1962). *Lep. Rev.*, XXXIII, 193–201.
12. PRICE, E. W. (1963). *Lep. Rev.*, XXXIV, 16–25.
13. ROSS, W. F. (1962). *Lep. Rev.*, XXXIII, 25–40.
14. ROSS, W. F. (1962). *Lep. Rev.*, XXXIII, 202–206.
15. TRUETA, S. (1944). *Principles and Practice of War Surgery.*
16. VELLUT, C. (1962). Report of the Activities for 1955 to 1962, Leprosy Control Unit, Polambakkam.
17. WARD, D. (1962). *Lep. Rev.*, XXXIII, 94–105.
18. WHO Technical Report Series No. 221 (1961).

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The Etiology and Natural History of Plantar Ulcer

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ETIOLOGY

The lesion called 'plantar', 'perforating' or 'trophic' ulcer is not peculiar to leprosy and is known to occur in several diseases in which there is a neuropathic lesion of the foot, but in which walking is still possible.

It does not occur when walking is not possible; and for these reasons it is seen in tabes, syringomyelia, and diabetic neuritis, but is uncommon in such disabling conditions as spina bifida and transverse myelitis where the patient may be bedridden. In leprosy, the disease is the cause of the nerve deficit which produces the plantar ulceration; leprosy does not cause ulcer directly.

The neuropathic foot is deficient in varying degree of motor, sensory and autonomic function; and all of these play a part in weakening the foot to the extent that walking causes ulceration. The separate deficiencies have been studied by several workers, and the relative importance of each has been discussed elsewhere (PRICE, 1959, 1961). The treatment that gives consistently good results is based on the assumption that the predominating factor in causation is the tissue damage due to the mechanical strains of walking.

The way in which this damage occurs is now well known and is described by several recent writers (ANDERSEN, 1961; ROSS, 1962; ZAMIDIO, 1963) and in the new Edition of COCHRANE and DAVEY, *Leprosy in Theory and Practice* (Wright: Bristol, 1964).

In summary, the crushing effect of body weight on the walking foot is added to a slipping that occurs between the bony framework of the foot and the ground beneath as each step is taken. This slipping does not occur in a foot with competent musculature; but when sensory and motor loss is present, the loss of control by the intrinsic musculature allows the soft tissues to be ground between bone and walking surface beneath, so that tissue necrosis takes place. The necrotic material slowly tracks to the surface, breaks through the skin and an ulcer is formed. The distribution of these ulcers is described in a later paper and is recognized to be that of the points of maximum walking pressure.

THE NATURAL HISTORY OF PLANTAR ULCER

The course of the lesion from its earliest beginning to frank ulceration can be readily observed whenever patients are submitted to regular weekly inspection. The duration and the ultimate permanent disability depend on two factors – the extent of pyogenic infection of the deep tissues, and the degree of collapse of the architecture of the foot which takes place.

In limited cases, the lesion may consist of a chronic ulcer with bouts of acute pyogenic infection; in more advanced cases, the attachment of the ligaments of the foot may be involved and collapse takes place. When collapse of the foot occurs, the primary ulcer may heal spontaneously and it is then often followed by a new ulcer at the prominent area of the sole. This latter ulcer may then persist indefinitely and even produce total destruction of the foot.

The natural history may therefore be considered in four phases:

1. Primary tissue damage
2. Primary plantar ulcer
3. Collapse of foot
4. Appearance of secondary ulceration.

1. *Primary Tissue Damage*

The sequence of events leading to ulceration passes through two stages that have been described as the *pre-ulcerative* state and the *necrosis blister.*

The *pre-ulcerative* state consists of localized swelling, deep tenderness, and evidence of deep oedema, such as spreading of adjacent toes. The disability is not severe enough to make a patient seek treatment, so that it will not be observed apart from regular foot-inspection. The sites affected are the pads over the metatarsal heads, the base of the fifth metatarsal, the heel, the interphalangeal joint of the big toe (and sometimes of the second toe) and the pulps of the toes. The time from the earliest symptom until the appearance of the next stage (the necrosis blister) is four to six days. This is the most favourable time for treatment and the lesion will resolve in a few days either by bed-rest or by the use of footwear with rigid sole and soft insole. Untreated, it passes inevitably into the next stage.

The *necrosis blister* is observed when the necrotic material from the deep tissue approaches the skin-surface. It is seen characteristically as a fluid collection, sometimes bloodstained, at the border of the hard skin of the sole. This means that it is seen either at the lateral or medial borders of the foot, or in the fold between the toes and the metatarsal pads. The sites are indicated by the edge of the shape left by a footstep in sand. The blister is largest at the heel, where it may contain 6–8 cc of sterile necrotic fluid. It is smallest at the anterior border of the metatarsal pads, under the toes where it may be mistaken for a banal or fungal infection. When the necrosis blister tracks to the surface directly through the hard skin of the sole, as it sometimes does, the swelling is hardly observed until the necrotic material is subcuticular; in this case the resulting lesion is collar-stud in shape and will give rise to a collar-stud type of ulcer. The necrosis blister rapidly becomes an ulcer if untreated; under treatment, it resolves in 10 days.

Both the pre-ulcerative state and the necrosis-blister are illustrated in the new edition of Cochrane and Davey.

2. *Primary Plantar Ulcer*

The sites of primary plantar ulcer are those of the previous necrosis blister, but it is not uncommon for ulcers over adjacent metatarsal heads to fuse and form a long ulcer across the metatarsal pads. The multiple ulcer on adjacent toe-tips is really one ulcer spread over several toes.

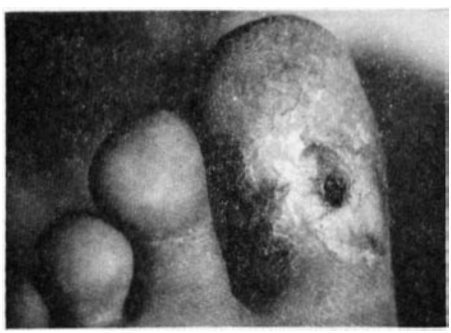
The importance of the ulcer, insofar as it concerns the future of the foot, can be considered as follows:

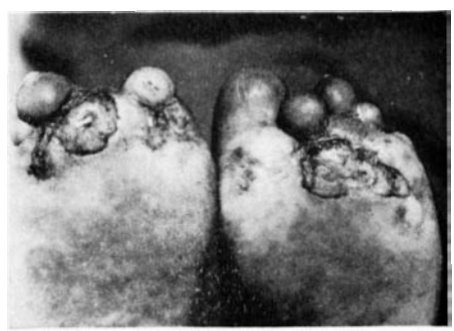
The most dangerous ulcer is that over the base of the fifth metatarsal.

The next is that of the heel, then that over the first metatarsal head.

The least dangerous to the foot are the toe-tip ulcers.

The reasons for this will be seen in the section on collapse of the foot.





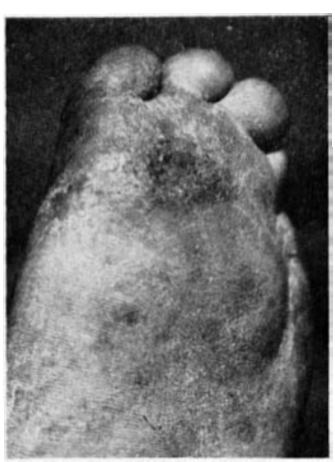


FIG. 1*a* (*top left*)

The Healing Process during Treatment with Footwear with Rigid Soles

(a) Stage 1. Diminution of exudate.

This ulcer, under the interphalangeal joint, was 2 cm. in diameter with considerable exudate, 10 days previously. It is now dry and epithelium is growing in rapidly.

FIG. 1*b* (*top right*)

(b) Stage of Epithelial Cover.

These four ulcers epithelialized under a petroleum jelly dressing within two weeks, using footwear with rigid soles. Epithelial cover proceeds rapidly if footwear is worn consistently.

FIG. 1*c* (*left*)

(c) Stage of Consolidation.

Tissue repair progresses under the epithelial cover, until the superficial scar resembles the surrounding skin. This ulcer healed within four weeks, after being open for nearly two years. Note the deformity caused by a previous metatarsectomy which made it difficult to fit footwear.

The stages by which the ulcer progresses have been described in several papers, so that the present occasion is taken to demonstrate the pathology

in reverse, so to speak. This can be observed by watching the healing that occurs when an ulcer is treated without any local application, except that the patient wears footwear with rigid sole. The following stages are recognized:

(i) Cessation of exudate (Fig. 1a).

The first sign of healing is a lessening of the exudate and this will be obvious within 24 hours, and is independent of pyogenic infection. It represents the beginning of deep-tissue repair.

(ii) Stage of epithelial cover (Fig. 1b).

As soon as exudate begins to lessen, epithelium rapidly grows in from the edges of the ulcer. Epithelial cover is replaced with surprising rapidity and defects of 2–3 cms can be completely covered within a week.

(iii) Stage of Consolidation (Fig. 1c).

When epithelialization is complete, the risk of secondary pyogenic infection ceases, but the 'ulcer' will rapidly recur if footwear is abandoned. Treatment – which in effect means continuation of the use of rigid-soled footwear – must be continued so that the scar consolidates in the deep tissues; at the same time, the superficial tissues and skin begin to take on the appearance of normal sole. This may be so effective that the site of ulceration may be difficult to recognize.

It is obvious from the above that a satisfactory method of treatment for uncomplicated plantar ulcer is the use of footwear with rigid sole. This is, in fact, what the writer uses in all such cases, the ulcer being covered with simple vaseline dressing to prevent soiling of the footwear.

3. *Collapse of the Foot*

The shape of the foot, which is commonly referred to as its arches, is maintained for the most part by the shape of the bones and the very strong

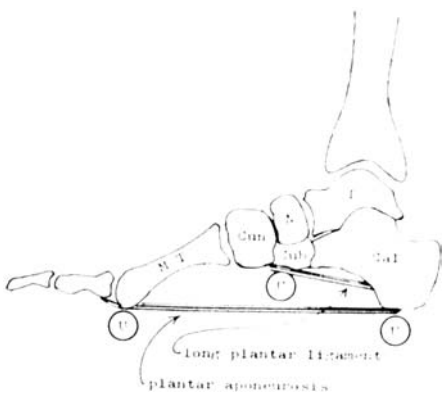


FIG. 2

Plantar Ulcers and the Ligaments of the Foot

Note that the common sites of ulceration overlie the points of attachment of the important plantar ligaments of the foot. The *heel ulcer* may involve the attachment of both the plantar aponeurosis and the long plantar ligament. The *mid-lateral ulcer* overlies the anterior attachment of the long plantar ligament; it is also in close proximity to the series of tarso-metatarsal joints and to those of the tarsal joints leading from the cubo-calcaneal region. This ulcer is therefore the most potentially dangerous of all plantar ulcers. The *ulcers over the metatarsal heads* do not seriously jeopardise the anterior attachment of the plantar aponeurosis because of the multiple attachments of the latter.

ligaments especially the plantar aponeurosis and the plantar ligaments, long and short (Fig. 2).

Muscular force, applied through tendons, is used mainly to prevent undue strain on these ligaments, but even complete muscular palsy does not cause collapse of the foot. The important ligaments are attached as follows: the plantar aponeurosis stretches from the calcaneum forward to the heads of the metatarsal and bases of the phalanges, and it will be observed that there is a multiple attachment in front.

The important long plantar ligament passes laterally from the calcaneum to the under-surface of the cuboid and adjacent bones. It will be seen at once that plantar ulcers lie over the attachment of these ligaments, and it will be recalled that the breakthrough of a necrosis blister from the deep tissues to the skin will expose these ligamentous attachments almost immediately to the risk of pyogenic infection.

The further steps in the natural history of a plantar ulcer that is not healing then proceeds as follows: osteoporosis with direct infection of bone where plantar ligaments are attached – partial detachment of underlying ligament – collapse of arch – projection of abnormal bony prominence in sole of foot – friction-pressure ulceration over new bony prominence.

It should be observed that collapse may follow the osteoporosis that follows longstanding disuse atrophy of a foot that has borne an ulcer for years; this is so whether or not there is direct pyrogenic infection of the bony attachment of the ligament.

The secondary ulcers that may occur depend on the type of collapse that has taken place and it is helpful to describe lateral collapse, medial collapse and total collapse of the arches of the foot. These may occur without resulting in secondary ulceration, but as the occurrence of this special type of ulceration emphasises the pathology, the types of collapse are described in the next section.

4. *Secondary Ulceration*

As the foot is progressively weakened by long infection or osteoporosis, the arches are progressively flattened by the weight of the body. Clinically, this event is noted by the projection, in the sole, of bony prominences that do not normally occur. The skin overlying these prominences may remain intact, but often superficial ulceration, due to friction-pressure, follows and then remains as a chronic or permanent lesion on the presenting surface of the sole of the foot. This ulcer has been called 'static ulcer', in distinction from the 'dynamic ulcer' which occurs at the beginning of the present clinical history of plantar ulcer.

The *lateral arch* depends largely on the integrity of the long plantar ligament and when it collapses it allows the following bony prominences to appear in the sole – the tuberosity of the cuboid and the anterior tubercle (keel) of the calcaneum. These two fall roughly in a line from the lateral tuberosity at the posterior part of the calcaneum at the heel, to

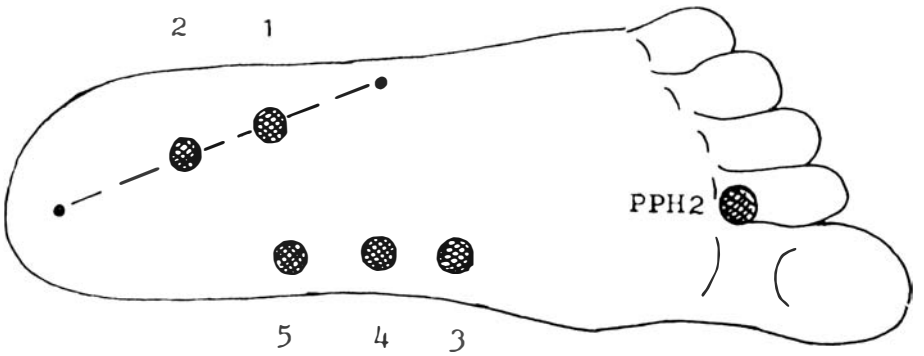


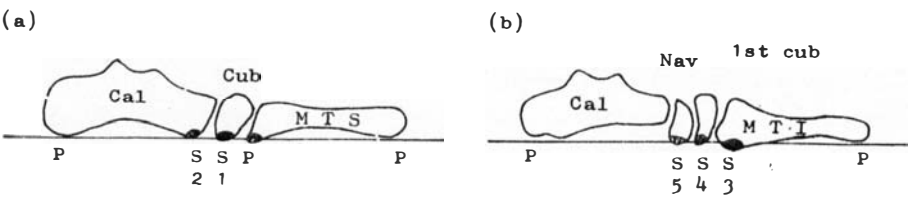
FIG. 3

Plantar Ulceration in Collapse of the Longitudinal Arches of the Foot

The diagram lists the sites of plantar ulceration which represent collapse of the longitudinal arches as follows:

1. Overlying the tuberosity of the cuboid.
2. Overlying the anterior tubercle (keel) of the calcaneum.
3. Overlying the base of the first metatarsal.
4. Overlying the medial cuneiform.
5. Overlying the tuberosity of the navicular.

PPH2 (Proximal Phalangeal Head 2) represents the ulcer that sometimes occurs over this bony prominence when the second toe attemps to take over the walking function of the big toe. This occurs when there is failure of the intrinsic musculature of the big toe.



(a) Collapse of lateral arch.
P represents primary plantar ulceration.
S represents secondary ulceration following collapse of the arch.
(b) Collapse of medial arch.
The numerals refer to the bony points on the diagram of the sole of the foot.

the tuberosity on the base of the fifth metatarsal (Fig. 3). An ulcer due to these prominences will lie along this line (Fig. 4).

The *medial arch* is much longer than the lateral and is supported by many structures; but the plantar aponeurosis and the many ligaments under the tarsal joints are mainly responsible for its shape. Detachment of one or more slips of the anterior attachment of the plantar aponeurosis is not likely to allow collapse of the arch, because the others remain intact. In fact, ulceration over one or more metatarsal heads does not usually lead to collapse of the foot.

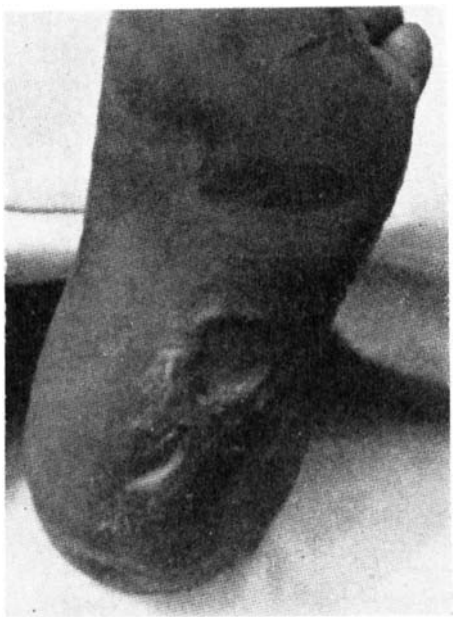


FIG. 4
Collapse of the lateral arch results in ulcers along a line from the base of the fifth metatarsal to the lateral half of the heel. In this case, the calcaneum is partly destroyed and the ulcers overlie the tuberosity of the cuboid and the keel (anterior tubercle) of the calcaneum.

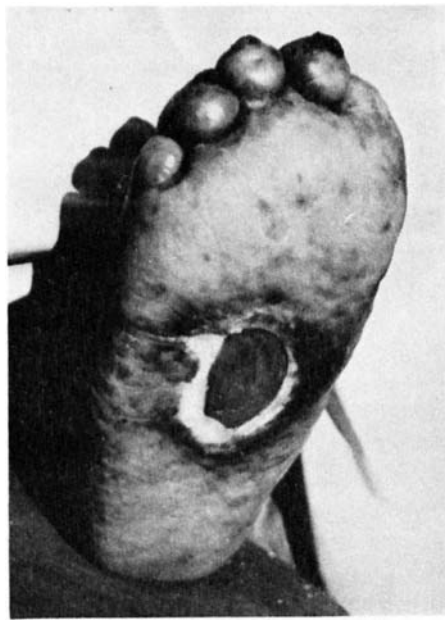


FIG. 5
Secondary Plantar Ulcer
This lesion, sometimes called friction-pressure ulcer or 'static' ulcer, to distinguish it from the 'dynamic' ulcer caused by walking on a mobile foot, occurs on the prominence of a foot that is totally collapsed.

The arches have all given way and the keel of the calcaneum underlies the ulceration. This is often called a 'boat-shaped' foot.

However, ulceration of the heel often leads to weakening and even detachment of a major part of the posterior attachment of the plantar aponeurosis. Collapse of the medial arch then allows new bony prominences to appear in the sole, and these again are in a roughly linear order proceeding medially forwards from the tuberosity of the calcaneum; these are the tuberosity of the navicular, the medial cuneiform and the base of the first metatarsal bone (Fig. 3). The overlying ulcer, if it occurs, demonstrates this clearly.

When both arches, lateral and medial, collapse, both linear ulcers may occur together. However, it is more usual for total collapse of the foot to follow a different course.

Total collapse of the foot allows the foot to take up, passively, the shape of a rocker which closely approximates to that which is carved on to the sole of wooden rockers. It is often called the 'boat-shaped foot'. The

prominence in the sole is the anterior end of the calcaneum and is mainly the keel of the bone.

An ulcer then appears to be directly central in the sole (Fig. 5). If ulceration does not occur, the collapse of the foot is total and if it is painless it can produce a surprisingly good rocker to the foot. It is often observed that as the rocker develops, any existing true ('dynamic') plantar ulcer will heal spontaneously. In effect, the body has achieved what we endeavour to do by using special footwear – it produces a state in which the strain of walking no longer falls on the front of the foot at its maximum pressure; in consequence, primary ulcers heal spontaneously.

DISCUSSION

The description of the natural history of plantar ulceration in leprosy shows that a small lesion of the foot may, if untreated, progress inexorably to total collapse of the foot. If this latter condition is also crowned by a chronic secondary ulceration, the foot may be totally lost as far as function and use is concerned. In addition, it is clear that the cause of the initial ulceration is a mechanical upset that can be controlled. In fact, medical knowledge of the lesion is such that every disabled foot in leprosy should indicate lack of sufficient care on the part of the patient or of the medical attendant. With the co-operation of both, leprosy should run its course to final arrest by therapy, without any major deformity or disability of the foot other than the loss of anesthesia (which may be inevitable) and some motor palsy (which can be compensated).

SUMMARY

1. The major factor in the etiology of plantar ulcer is mechanical.
2. The natural history of the lesion is described in detail from its earliest beginning to final destruction of the foot.
3. It is emphasized that plantar ulceration can be prevented, can be arrested at whatever stage it has reached, and should never be allowed to produce the deformed and defective feet that are still only too common in areas where the disease of leprosy is endemic.

References

ANDERSEN, J. G. (1961). *Leprosy Review*, **32**, 16.
PRICE, E. W. (1959). *Leprosy Review*, **30**, 242.
PRICE, E. W. (1961). *Leprosy Review*, **32**, 108.
ROSS, W. F. (1962). *Leprosy Review*, **33**, 25.
ZAMBUDIO, L. (1963). *Dermatologia*, **7**, 3.

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The Problem of Plantar Ulcer

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Ulceration of the feet undoubtedly causes more unpleasantness for patient and medical attendant than any other complication of leprosy. Many papers have been written on the subject, and many types of treatment have been recommended; nevertheless foot ulcer remains a major problem.

The purpose of this symposium is to summarise recent understanding of the condition, and to show that present knowledge makes it possible to cure foot ulcer, to prevent its recurrence, and, better, to recognize the danger before ulceration actually takes place.

Foot ulcers cannot be understood unless it is clearly recognized that there are several types of ulcer due to different causes, and needing different treatment. Much of the frustration that is caused by these lesions is due to confusion about classification. The term also includes ulcers around the edge of the thick plantar skin, that is, on the side of the foot.

CLASSIFICATION

The following types of ulcer occur on the sole of the foot in leprosy:

A. *Infected Wounds and Burns* (Fig. 1)

These are caused by thorns, sharp stones, or projecting nails inside footwear. In all cases, banal pyogenic infection may occur and endanger the

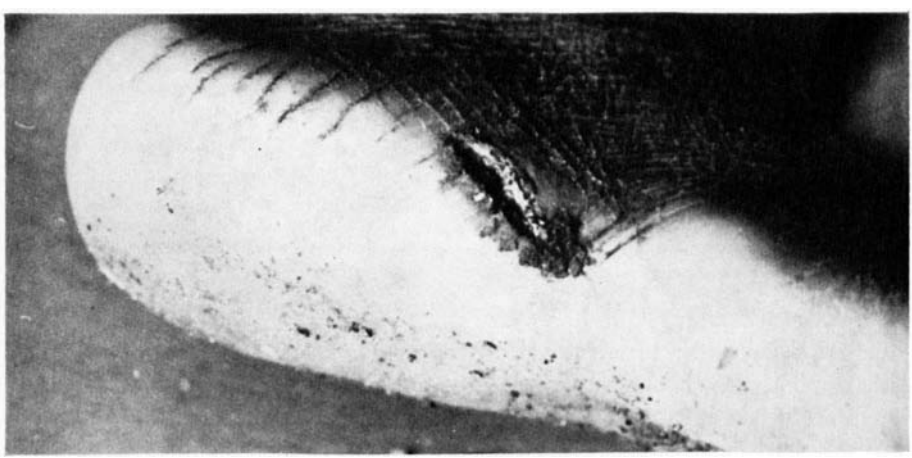

FIG. 1

Foot Ulcer due to Infected Wound

This ulcer is due to infection of a cut. It closely resembles the lesion which occurs when necrotic material from the deep tissues of the sole reach the skin surface at the margin of the thick plantar skin. This ulcer is a bit posterior for a true mid-lateral lesion and too far forward for a true heel lesion.

skin or deep tissues of the foot. These do not differ from similar lesions in non-leprosy patients, but may progress further because of loss of sensation; the patient does not seek early treatment, and will continue to walk on an infected foot.

B. *Plantar Ulcers*

This term is used for a special type of lesion that occurs in any neuropathic foot whatever the cause – diabetic neuritis, tabes, syringomyelia, etc. It is described in detail in the following papers. Sometimes it is called 'neuropathic ulcer', 'trophic ulcer', or 'pressure ulcer' but the above term is commonly used in modern literature. Since the lesion is related to walking it does not occur elsewhere than on sole of the foot, and it does not occur in neuropathic feet where walking is impossible.

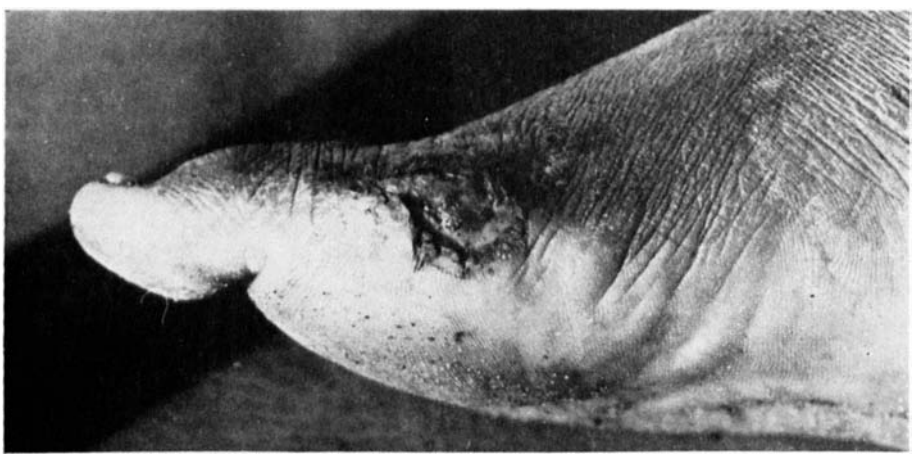

FIG. 2

Friction Ulcers

This ulcer was caused by friction of a sandal strap but it closely ressembles the lesion that occurs when necrotic material from beneath the head of the first metatarsal bone finds its way to the surface at the margin of the thick plantar skin.

C. *Friction Ulcers*

Ulceration is commonly due to friction damage. On the *dorsum of the foot*, and usually over the toes or metatarsal heads, ulcer is commonly due to the rubbing of a sandal strap, or the toe-piece of unsuitable footwear (Fig. 2). On the *sole of the foot*, ulcers occur on the prominent part of the deformed foot and are due to a combination of friction and pressure (see previous paper).

Toe-tip ulcers occur in drop-foot where the toes are scraped along the ground with each step. The sole ulcers due to friction-pressure are described in detail in the section on Complications.

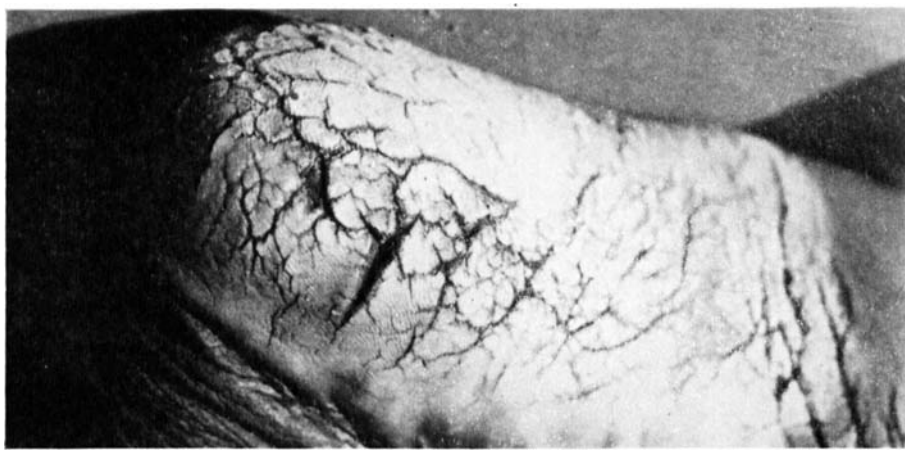

FIG. 3
Cracked Soles
This lesion, of unknown etiology, is common in Africa. It is therefore not infrequent in leprosy patients, and in this case an infected crack at the heel can cause confusion with the ulcer caused by the breakdown of the skin where necrotic material from the deep surface of the calcaneum reaches the surface. When both lesions occur, the necrotic material may make use of the crack to reach the surface.

In addition to the above, ulceration may occur between the toes, due to fungus infection, or in deep cracks round the sides of the heel – this latter of unknown cause (Fig. 3). These ulcers may occur in any patient, leprosy or not, and are not further considered here.

A careful study of the following pages will show that foot ulcer should rapidly become a thing of the past, and the associations of 'ulcer shed', smell, flies, lysol, and large quantities of cotton wool and bandages, be nothing but unpleasant memories. Certainly, few things give more satisfaction to a patient and to the medical attendant, than the permanent healing of a foot ulcer.

SPECIAL DIFFICULTIES

The common occurrence of plantar ulcer in leprosy is responsible for special problems that can be considered under four headings – medical, practical, organizational and psychological.

A. *The Medical Problem*

Why does plantar ulcer occur? Why does it heal by any of at least 40 different methods? Why does it relapse so easily? Can it not be prevented? Happily, we now know the answer to these questions in sufficient detail to make treatment a success. The most important item is the decision as to the type of ulcer; a mistake here will frustrate successful treatment. The problem ulcer is the so-called 'plantar ulcer'. Plantar ulcer will be seen to occur because of the stresses of walking on a foot with reduced nerve

supply; it heals rapidly with any method that reduces walking, intentionally or not; it relapses easily because walking is resumed after the cessation of treatment; its recurrence can be controlled by the use of rigid-soled footwear; and its occurrence can be prevented by regular foot-inspection.

It is not possible to lay down fixed rules for every possible ulcer, so that detailed knowledge of causation, complications and treatment is needed, if failure is to be avoided.

Finally, it is important to know when permanent healing cannot be obtained because of long-standing and severe bone-infection and deformity. Much time can be saved if an inevitable amputation is performed early; on the contrary, it is a serious mistake to amputate a foot that could have been rehabilitated.

B. *Practical Problems*

It is not possible to treat plantar ulcer with satisfaction without some type of rigid-soled footwear. The cheapest is made by local craftsmen, and in most parts of the world wood has proved the best material. In case of difficulty, the wooden soles can be obtained from clog-sole manufacturers. Details of rigid-soled footwear are given later.

An unsolved problem is what to do in areas where there is heavy rainfall and mud, or where the cultivation of rice entails work in water. In these circumstances any type of footwear is a handicap, and it may be that the use of the rigid gait of the bare foot – which can be taught to patients – is the only ultimate solution.

A further practical problem is the provision of adequate below-knee 'legs' for amputation cases. The modern patella-bearing plastic limb is proving a likely solution, but the fitting has to be exact in a patient who is anaesthetic at knee-level, and skill is necessary to achieve this. Nevertheless, the making of such limbs is not beyond the ability of a capable craftsman with sufficient training, and these limbs should be available in every area where a serious attempt is made to solve the 'problem of plantar ulcers'.

C. *The Problem of Organization*

Difficulties of organization arise because of the number of cases that seek treatment in many areas – often a hundred or more a day. The application of systematic treatment with limited staff and material may be well-nigh impossible in these circumstances, and a plan of action must be prepared and followed with diligence to avoid disappointment. In all such situations, the first step is to initiate measures for prevention of new ulcers; otherwise new ulcers appear as fast as the old ones are treated. A suitable plan is outlined here.

(i). Before treatment is begun, *regular foot inspection* must be organized under the direct control of the clinic – monthly at first, but later weekly, of all patients who live nearby. At first inspection is heavy work, but becomes easier as the number of ulcers diminish. The inspector watches particularly for the signs of the pre-ulcerative state, or the necrosis blister,

also for early ulceration which patients tend to neglect, or be unaware of. Absentees must be dealt with firmly.

(ii). At the same time, *a workshop* is organized for rigid-soled footwear. The simple flat sole with rockers may be used at first, but patients will prefer the shaped sole, a pair of which can be made in about three hours by a competent technician.

Many ulcers will heal by the use of footwear alone, and this results in considerable saving in plaster of Paris casts.

(iii). When inspection and sandal-making are begun, *the treatment of ulcers* can be undertaken. To begin with – and when the patients do not yet have confidence in the method – plaster-casts are the best initial treatment; the oedema and pyogenic infection are controlled, and then a plaster-cast will certainly heal the ulcer, if it is maintained for long enough. Later, all uncomplicated ulcers, and many complicated ones, will be treated with footwear alone. Details of treatment are given in a later paper.

If the above scheme is pursued with vigour, it should be possible to close any existing ulcer-shed within three months, and treat new cases in the clinic.

D. *The Psychological Problem*

During effective medical care, there are always a number of cases that do not respond to therapy, or in whom ulcers recur even when the patient says that he wears the footwear all the time. In fact, nothing brings into sharper focus the psychological problems of leprosy than the 'resistant' cases in a foot clinic.

The situation commonly occurs when the patient is aware that the existence of a chronic ulcer will ensure his admission to hospital or a settlement, and that he can rely on food and lodging as long as ulceration continues.

The failure of an uncomplicated ulcer to heal within five weeks, is an indication either that the patient is not wearing his sandals, or that he is walking an inordinate distance daily. In the writer's clinic, patients take advantage of healed feet to walk to a local market three miles away, daily, in order to beg. Newly healed ulcers will not accept this abuse.

Other patients will leave off their footwear to ensure that ulceration continues; one such was found to be sand-papering the healing surface regularly to discourage epithelialization!

It is the writer's practice to discharge from treatment all uncomplicated ulcers that do not heal in six weeks, but a healed ulcer is observed weekly for two months before the final discharge.

Further pyschological problems include the inability to face community life in a village or town, after the protection of a settlement – a condition that has been called 'social atrophy'. An even deeper problem is the aversion that a patient may develop to his own foot when ulceration is prolonged, deformity is considerable and the smell is unpleasant. These

patients will not accept their foot even when it is seen to be healed over a period of months. In such cases, it is surprising how rapidly the mental outlook improves after amputation. This is even more evident, when cheap, but efficient, below-knee limbs are available.

CONCLUSION

Few things improve the morale of patients – and, one might add, of the medical attendant – than the successful treatment of plantar ulcers. This symposium is composed to demonstrate that this is now possible, and that the protection of the anaesthetic foot can be achieved and maintained, with a minimum of equipment and expense, so long as the principles of foot care are practised with determination.

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Surgery and the Prevention of Plantar Ulcers

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INTRODUCTION

Ulceration of the extremities in patients with nerve damage is almost invariably the result of unregarded trauma (RICHARDS). In the foot the large majority of ulcers occur on the weight-bearing portions of the sole and are due to the trauma of walking. This is very much increased when the neuropathy is associated with established deformity. BRAND and his colleagues have shown that for example in cases of foot-drop the pressure under the head of the 5th metatarsal may be increased to 2 or 3 times that in normal individuals. CLAWSON and SEDDON are of the opinion that deformity is a much more important cause of ulceration than anaesthesia. This opinion is entirely in accord with our experience and we find that in practically all cases the site of an ulcer is determined by the presence of static or dynamic deformity. In many individuals the points of increased

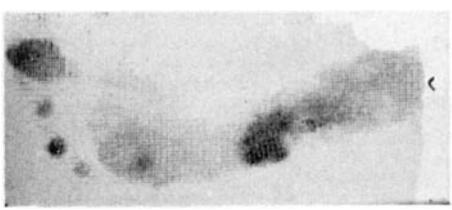


FIG. 1. Walking footprint of a patient with gross inversion.

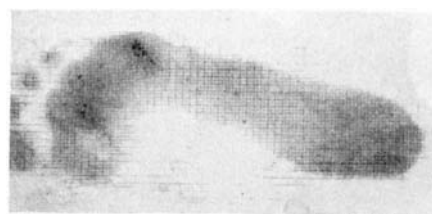


FIG. 2. Walking footprint of a patient who subsequently developed ulceration under the 2nd metatarsal head.

trauma on these anaesthetic and deformed feet is obvious on inspection as for example in an inverted foot it is quite clear that the maximum trauma is occurring at the lateral border of the foot (Fig. 1). In other cases points of maximum trauma are not so obvious but can be discovered by means of a walking footprint. In many instances the use of this device enables the surgeon to predict the point at which ulceration will occur unless preventive measures are taken. Some of our patients have not accepted the necessity for such measures and we have seen ulcers develop at the expected sites (Fig. 2). It is not always appreciated that a dynamic deformity, due to muscle imbalance, is just as important a factor in causation of ulceration as is fixed deformity. Dynamic deformity cannot always be detected by inspection of the foot but are quite easily revealed by the walking footprint. Further, if the patient has been walking in shoes the pattern of wear on the insole is often a valuable indicator of the site of danger points.

There is no doubt that footwear for these deformed feet is of very great value (PRICE, ROSS, and WARD) but it seems reasonable to say that in addition to providing suitable footwear, deformity should be corrected surgically wherever it is possible. This has the great advantage that when once the deformity has been corrected we find that ulceration can be prevented in many cases by relatively simple and inexpensive footwear. In addition many cases of gross deformity for whom footwear alone is not effective can be helped by a combination of judicious surgery and suitable footwear. Surgery is not to be undertaken lightly and should be part only of a co-ordinated plan to obtain a weight-bearing foot.

METHODS

At this institution 502 patients with plantar ulcers were admitted for treatment during the years 1961–62. Following the healing of the ulcers 58 of the patients were subjected to 76 operations designed to prevent recurrence of the ulceration (from these 10 below-knee amputations and 4 amputations of the great toe are not included in this study). Forty-three of the cases have been followed-up and re-examined in June and July this year. The followed-up cases had had 53 operations. Account has also been taken of 69 similar procedures done in the years 1963–64 and of 33 operations for the correction of foot-drop and 13 for correction of inversion of the foot carried out on leprosy patients who have never had ulceration. One of the cases of foot-drop developed metatarsal disintegration following foot-drop correction (Fig. 3).

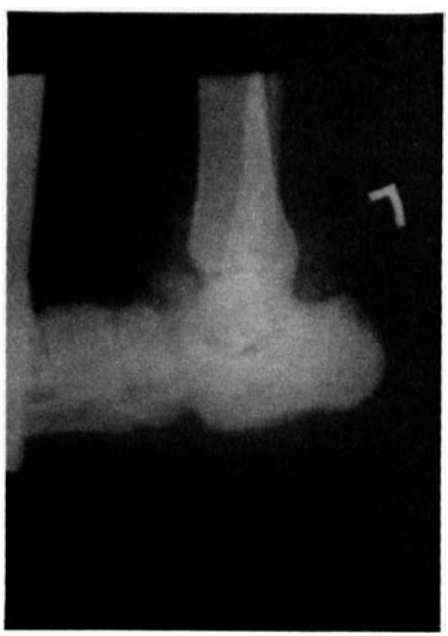


FIG. 3. X-ray showing disintegration of mid-tarsal bones after tibialis posterior transplantation.

In all cases an attempt has been made to ascertain the time at which ulceration of the foot first occurred and to discover in broad outline the subsequent history of the foot. Records of all patients who have been treated here from January 1959 onwards are available and have been consulted. The methods used for pre- and post-operative assessment of patients have been described in Chapter 29 of *Leprosy in Theory and Practice*, 1964, 2nd edition, by R. G. COCHRANE and T. F. DAVEY. These include tests for anaesthesia and paralysis, the taking of a walking foot-print and a recording of the general texture of the skin of the sole and the presence or absence of haematomata, scars, callosities, fungus infection and open

wounds. Clawing of the toes and other deformities are also noted and records made of the range of movement in the ankle joint and of abnormalities detected clinically in the tarsal and mid-tarsal joints. An X-ray plant has been available for the past 6 months and all cases included in the study had standard anterior-posterior, lateral and oblique views taken of the foot (HARRIS and BEATH).

RESULTS

See Table I.

This analysis is concerned only with surgery and no attempt has been made to discuss why footwear failed to control ulcers in the cases who had it.

During 1961/62 approximately 1 in 8 of the patients admitted for ulcer treatment had surgery subsequently. This proportion has now risen but no patient is subjected to surgery if it is felt that footwear alone has a good chance of preventing re-ulceration.

In 33 feet the operations have been completely successful. In 15 there has been failure or partial failure post operatively and in one the foot became gangrenous and had to be amputated.

Nine of the failures were undoubtedly due to inadequate excision of bone, resulting in weight bearing on scarred skin post operatively, 6 including the case (44) which became gangrenous were due to wrong choice of operation and one patient (29) should not have been operated upon at all.

(a) Cases with tibialis posterior transplant (11). Cases (1–10).
Seven out of the 11 cases of tibialis posterior transplant have been completely ulcer free since the operation. The 4 ulcers which have occurred have all been small. One was due to failure to balance up the foot as a result of using a procedure involving tendon anastomosis which has now been given up (case 5), and one was due to failure to line up the stumps accurately during forefoot amputation (case 8). The ulcer in case (3) could perhaps have been avoided if a subtaloid arthrodesis had been done as well as the tibialis posterior transplant, and the feet in case (10) are so badly scarred that the fact that the patient has only had one ulcer is a pleasant surprise.

(b) Cases with triple arthrodesis (7). (Cases 8, 11, 13–18.)
Two of these cases should have had amputation as a primary procedure (cases 11 and 13) as the feet were so scarred that it was impossible to relieve the scarred tissue of weight bearing. In two cases the bone resection was inadequate (cases 17 and 18) and three cases have been completely successful (cases 14, 15 and 16).

(c) Cases with wedge resection of the ankle joint (4 patients).
Two of these cases were failures. Case (12) has subsequently had an amputation and case (30) was recommended for an amputation in 1959 and refused. Cases (28) and (20) have been completely ulcer free since the operation.

(d) Cases with forefoot amputation (9).
Six out of the 9 cases are ulcer free, *i.e.* cases (4, 23a, 25, 26 and 27a and b).

Two of the failures (case 8 and 24) are due to inadequate excision of bone with the result that scarred tissue continues to bear weight and 1 (case 23b) is due to surgical intervention in the presence of infection.

(e) Cases with metatarsal head resection (4).
Two of the cases (42 and 43) have not re-ulcerated and two (40 and 41) have. The two failures were due to failure to line up the metatarsal stumps at operation, with the result that the more prominent stumps bore too much weight.

TABLE I

Case No.	*Duration of Ulcers minimum yr.*	*Principal Sites of Ulcers*	*Shoes Pre-op.*	*Operation*	*Date*	*Shoes Post-op.*	*Date of Ulcers Post-op.*	*Site of Post-op. Ulcer*	*Comments*	*Results*
1	5	M.H.3, mid-sole	—	Tibialis Posterior Transplant	8:62	Micro-cellular rubber shoes	—	—		S
2	5	M.H.3, toe-tips	—	,,	5:61	,,	—	—		S
3	5+	M. Heads mid-lat.	Wood shoes	,,	12:62	,,	63	Mid-lat.	Small dry ulcer. Foot not deteriorating	S
	5+	M. Heads mid-lat.	,,	,,	4:1:63	,,	—	—		S
4	5+	M.H.1, M.H.2 mid-sole	,,	Fore-foot amputation	3:61	,,	—	—		S
				+ tib-post. transplant	12:62					
5	4	P.P.H., M.H1.	M.C.R.	Tib.-post. transplant	1:62	,,	63	M.H.2	Inversion accentuated post-op. Tendon anastomosis done	F
6	4	M.H.5	,,	,,	7:61	,,	—	—		S
7	3	M.H.5 lat. sole	,,	,,	4:62	,,	—	—		S
8	3+	M.H. 1–5	,,	Fore-foot amputation	3:62	,,	64	M.H.3	18/12 post-op. small ulcer due to inadequate excision of M.H.3	F
				Tib.-post transplant	11:62					
				Sub-taloid arthro'	5:62					
9	3+	Mid sole M.H.3 heel	Clogs	Tib.-post transplant	6:62	,,	—	—		S

TABLE I—*continued*

Case No.	*Duration of Ulcers minimum yr.*	*Principal Sites of Ulcers*	*Shoes Pre-op.*	*Operation*	*Date*	*Shoes Post-op.*	*Date of Ulcers Post-op.*	*Site of Post-op. Ulcer*	*Comments*	*Results*
10	10+	M. Heads Toes	Clogs	Tib.-post transplant	5:61	M.C.R. shoes	—	—	These feet are very short	S
	10+	,,	,,	,,	5:61	,,	64	Mid-lat.	Small dry ulcer with gross M.H. damage	S
11	5+	M. Heads Toes Mid-lat.	Clogs and M.C.R.	Triple arthrodesis	6:61	,,	61	Mid-lat.	This foot was grossly scarred and the procedures failed to relieve scar of weight bearing	F
12	5+	M. Heads Toes Mid-lat.	M.C.R.	Wedge osteotomy	9:62	,,	62	Mid-lat.	(Same patient as 11) This foot was grossly scarred and the procedures failed to relieve scar of weight bearing	F
13	5+	Heel and mid-lat.	Wood shoe	Triple arthrodesis	9:62	,,	63	Mid-lat.	,,	F
14	3	Mid-lat.	—	Triple arthrodesis + tib.post. transplant	11:62 8:63	,,	—	—	11/12 follow up	S
15	5+	,,	M.C.R.	Triple arthrodesis	12:62	,,	64	Lat. border	Ulcer not on weight bearing surface. Due to shoe strap	S
16	5	M. Heads Toes	—	,,	4:62	,,	—	—	Anterior wedge to correct equinus	S
17	5+	Mid-lat.	Clogs	,,	12:62	,,	63	Mid-lat.	Inadequate lateral wedge	F
18	5	M.H.2 and 3 mid-lat.	—	,,	10:62	,,	63	,,	,,	F
19	4	M.H.1 and 5	—	MH1 excised Toe 2–5 claw corrected	61 8:62	,,	— 63	— Toe tips	Ulcers due to burns	S

TABLE I—*continued*

Case No.	*Duration of Ulcers minimum yr.*	*Principal Sites of Ulcers*	*Shoes Pre-op.*	*Operation*	*Date*	*Shoes Post-op.*	*Date of Ulcers Post-op.*	*Site of Post-op. Ulcer*	*Comments*	*Results*
20	3	P.P.H. and M.H.1	M.C.R.	Claw toe correction 1–5	4:61	M.C.R.	—	—		S
21	2	M.H.5	,,	Claw toe correction 1–4 Excision M.H.5	12:62	,,	—	—	Slight decrease in depth of anaesthesia by June 64	S
22	5	M.H.4	—	DuVries M.H.4	10:62	,,	—	—		S
23	4	M.H.2, 3, 4 and 5	—	Fore foot amputation	7:61	,,	—	—		S
	4	M.H.2, 3, 4	—	,,	7:61	,,	62	Dorsum	Operations done in presence of ulcers. Small portion of wound did not heal satisfactorily	F
24	5	M.H.2, 3	Wood shoe	,,	6:62	,,	10:63	M.H.3	Amputation too conservative	F
25	4	Mid M.H.	Clog	Fore foot amputation	12:62	,,	—	—		S
26	2+	M. Heads	,,	,,	2:62	,,	—	—		S
27	11	M.H.2, 3 4	,,	,,	9:62	,,	—	—		S
	11	M.H.1, 2, 3, 4	,,	,,	9:62	,,	—	—		S
28	5+	Mid-lat., M. Heads and toes	,,	Wedge osteotomy ankle	7:62	,,	—	—	Grossly deformed foot	S
29	5+	Forefoot	,,	Wedge resection	8:62	,,	—	—	Equinus remains fixed at 100°	S
30	10+	Mid-lat.	Wood shoe	,,	9:62	,,	5:63	Lt. mid-lat.	Amputation recommended in 1959 and refused by patient	F
31	5+	Heel	—	Ankle fusion for Châcot joint	6:62	,,	—	—	No ulcers but fusion has completely broken down	F

TABLE I—*continued*

Case No.	*Duration of Ulcers minimum yr.*	*Principal Sites of Ulcers*	*Shoes Pre-op.*	*Operation*	*Date*	*Shoes Post-op.*	*Date of Ulcers Post-op.*	*Site of Post-op. Ulcer*	*Comments*	*Results*
32	2	Toe tips	Clog	I.P. arthrodesis	7:62	M.C.R.	—	—		S
33	3	M.H.2, 3, 4, 5	—	I.P. arthrodesis Ex. to M.H. heads	7:62	,,	—	—		S
34	2	Toe tips M.H.1	—	I.P. arthrodesis Hallux 2–5 extensors to M. Heads	5:62	,,	—	—		S
35	10	M. Heads	—	Extensors to heads 1 and 2	4:62	,,	—	—		S
36	9	M.H.2	Clog	Excision P.P. toe 2	3:62	,,	—	—	Previous division of extensor tendons to relieve clawing failed	S
37	6	Heel	—	Split heel	10:62	Special shoe	63	Heel	Pre-op. scar fixed to calcaneum	F
38	5	M.H.2, 3, 4, 5	Clog	Tendo-Achilles lengthening	10:62	M.C.R.	3:64	M.H.1 and 3		F
39	3	M.H.5	M.C.R.	Tib. ant. transplant	7:62	,,	—	—	M.H.5 excised previously but this failed to prevent reulceration	S
40	5	M.H.1, 2 and 4	Wood shoe	Excision metatarsal heads	12:62	,,	10:63	M.H.4	Excision of metatarsal inadequate	F
41	8	Mid M. heads	,,	,,	7:62	,,	4:64	M.H.2	Excision H.2 inadequate	F
42	4	,,	Clog	,,	12:62	,,	—	—		S
43	5	,,	,,	,,	6:62	,,	—	—		S
44	10+	Mid-lat. M. Heads Toes	Wood shoe	Astragalectomy	12:62	—	—	—	Foot became gangrenous and was amputated	F

(f) Claw toe corrections.

i. Interphalangeal joint arthrodesis (3).
One case (32) had arthrodesis alone, one (33) had arthrodesis combined with transplantation of the long extensor tendons into the metatarsal heads, and one (34) had arthrodesis of the hallux combined with transplant of the long extensors. All were successful.

ii. Extensor tendon transposition (3).
In addition to cases (33 and 34) which had extensor transplant plus arthrodesis, one case (32) had the extensors of first and second toes transposed into the metatarsal heads. No further ulcers have occurred.

iii. Excision of proximal phalanges.
In case (36) the P.P. of toe 2 was excised, and the patient has had no further ulcers.

iv. Flexor tendon transposition (3).
None of these cases has had an ulcer post-operatively; in case (19) the transplant was combined with excision of the 1st M.H., and in case (21) with excision of M.H.5.

v. Duvries operation (Fig. 20)
The single case (22) was successful.

(g) Split heel (1). Case (37).
Failed. The patient has subsequently had a long leg amputation using the dorsal skin of the foot to cover the stump, the stump is not suitable for weight bearing.

(h) Tendo achilles lengthening (1). Case (38).
Failed. The patient has no equinus nor foot drop, but the forefoot is very badly scarred, he is now ulcer free in a shoe with a metatarsal bar.

(i) Tibialis anterior transposition (1). Case (39).
This patient had peroneal palsy only, with the foot inverted. Transplant of the tibialis anterior to the lateral side of the foot has provided an adequate correction.

(j) Astragalectomy (1). Case (47).
This operation was attempted through an anterior incision. The foot became gangrenous. The procedure has been abandoned in favour of lateral wedge resection.

CONCLUSIONS AND RECOMMENDATIONS

I. *Pre-operative Considerations*

No corrective surgery should be undertaken in the presence of infection or of infected wounds. The first step is, therefore, to heal any ulcers and clear up fungus infection. Surgery has a relatively small part to play in the treatment of ulcers and the application of a plaster of Paris cast after carrying out wound toilet remains the basic method of treatment. In our experience wounds heal more quickly if plaster of Paris casting can be supplemented by bed rest, but this is not essential. It is prudent to wait for one month after all wounds have healed before undertaking surgery of the fore-foot and at least 3 months before operations on the mid-foot and hind-foot are carried out. If it occurs, operative infection is at the worst disastrous leading to gangrene of the extremity and at the best, delays healing much longer than the 4 weeks or 12 weeks recommended above. As a rule there is no urgency about operations for the correction of foot-drop and in these cases it is reasonable to wait until at least 6 months have passed after the foot-drop has occurred as with suitable medical care many

cases of early foot-drop recover. Surgery for deformity causing ulceration is a matter of urgency and should be carried out whatever the stage of the patient's general treatment. If reactions occur they can be treated in the usual manner.

II. *General Surgical Technique*

Surgery of the foot does not as a rule require such delicate techniques as surgery of the hand but, of course, if good results are to be achieved, just as much care and attention to detail is required in foot surgery as in hand surgery. A bloodless field is essential and a pneumatic tourniquet should be used. A pressure of 280 mm. of mercury is adequate and should not be exceeded and it is probably wise not to keep the tourniquet applied for more than 1 hour at a time. This is because the operation may precipitate neuritis and in addition, many of these operations are taking place in feet potentially infected and the shorter the period of haemostatis the better. If at the time of operation, when incisions are being made through previously ulcerated tissue, as for example when metatarsal heads are being excised, the tissues are found to be oedematous or show other signs of deep infection, the wounds should be left unsutured. These wounds may subsequently be sutured as a delayed procedure or suturing omitted altogether. Such open wounds heal well if properly immobilized but if infected tissue is sutured sloughing invariably occurs. Pressure bandages should not be used post-operatively as these may lead to superficial skin necrosis or even deep necrosis in certain cases. All operated limbs should be immobilized in padded non-weight bearing plaster of Paris casts. The toes should be left open to inspection in every case and the limb elevated for 48 hours post-operatively. After 48 hours if there is no oedema or pain ambulation with crutches may be permitted.

III. *Post-operative Care*

In most neuropathic limbs response to trauma is good and healing takes place at the normal rate so that skin sutures can be removed after the normal period of 10 days and parts subjected to tendon transplantation and arthrodesis immobilized for 4 to 6 weeks for tendon transplants and 10 to 16 weeks for arthrodeses. In patients with neuropathic bone changes, however, fusion is extremely difficult to achieve and surgery is not recommended as a method of dealing with this problem. All cases must be provided with suitable footwear immediately post-operatively. The deformities amendable to surgery are only one factor of many in the etiology of ulcers and unless facilities are available for the provision of footwear surgery is better not undertaken.

IV. *Common Deformities and their Corrections*

1. Deformities due to paralysis of the lateral popliteal nerve. This nerve supplies all the muscles which dorsiflex and evert the foot and which dorsiflex the toes. It also supplies one of the muscles which can invert the

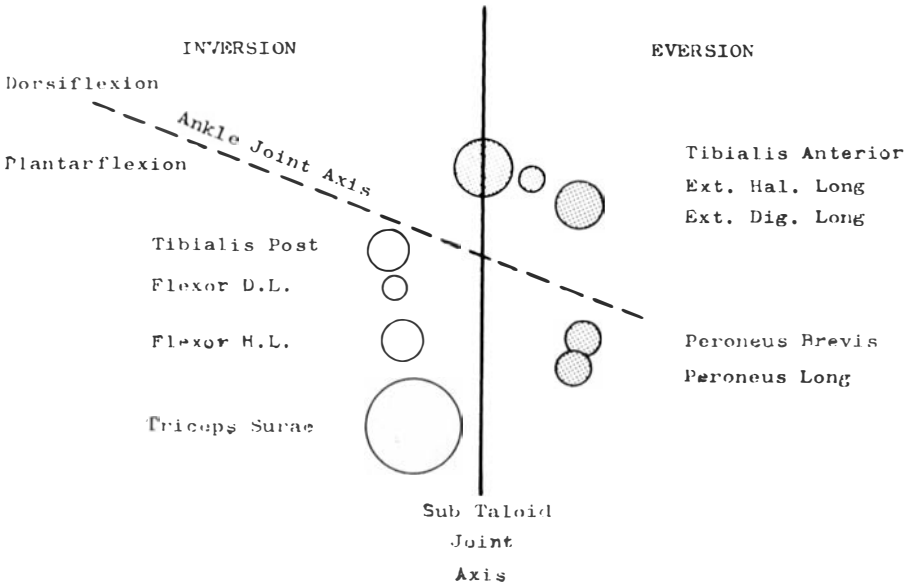


FIG. 4. Muscular control of the ankle joints. The muscles are represented by circles that have an area proportional to the physiologic cross section of the muscle. The position of the circles represents measurements of their lever arms with respect to the joint axes. Muscles marked are innervated by the lateral popliteal nerve. (From Elftman H. 'The Transverse Tarsal Joint and its Control'. *Clinical Orthopaedics*, **16**, 41–44.)

foot and a plantar flexor of the first ray (Fig. 4). In leprosy these muscles may be completely paralysed or not paralysed at all or different muscles in the group may be affected to different degrees. Two stable patterns of paralysis are seen (a) complete paralysis, and (b) paralysis of lateral compartment muscles only. The former is by far the most common. We have seen 78 cases of complete foot-drop, 18 of them bi-lateral and only 6 cases of lateral compartment paralysis, 2 of them bi-lateral. None of the cases of lateral compartment paralysis escaped destructive ulceration. Sixty-four of the legs with complete foot-drop were of over 6 months duration when first seen. In only 1 of these did recovery occur and this patient had had the paralysis for 9 months. Thirty-seven of the feet showed gross secondary deformity of various sorts. These included 9 cases with neuropathic changes in the mid-tarsus, 9 cases with gross damage to the toes, and 23 cases with gross damage to toes and metatarsal heads; 6 of the latter had fixed equinous deformity also. Twenty-six of the late cases with complete foot-drop had minimal secondary deformity. Fifteen of them had never had ulceration and these included one bi-lateral case who claimed to have had the foot-drop for 20 years. All except 4 of the cases had an associated anaesthesia of the sole of the foot. Of the 11 cases who had had ulcers 4 had ulceration of the tips of the toes, 4 ulceration under the proximal phalanx of the hallux; one ulceration under the 5th metatarsal head and one ulceration under the 5th metatarsal base, and 1 case had ulceration under the middle metatarsal head. Of the 23 legs

seen within 6 months of the foot-drop developing 14 recovered under medical treatment.

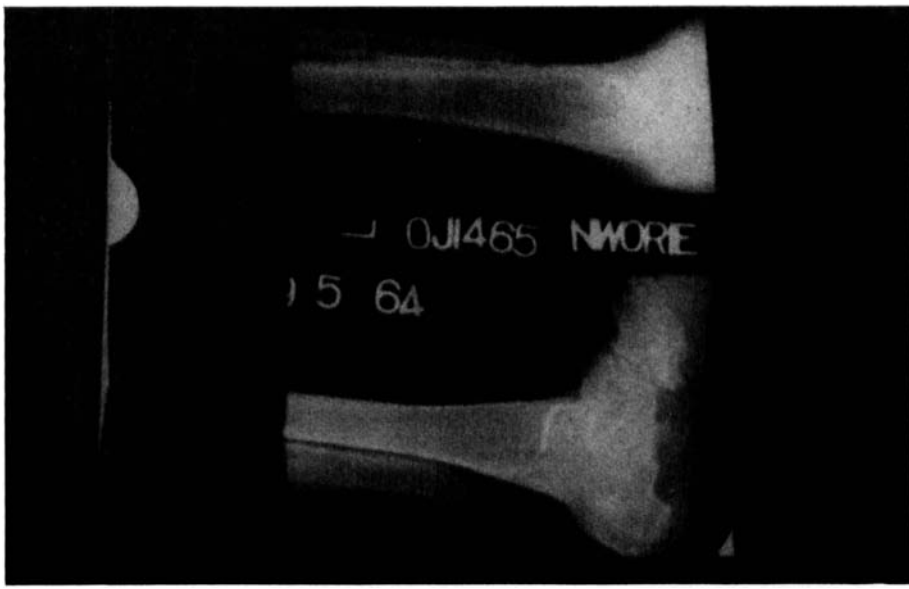


FIG. 5. X-ray of a patient who has had wedge resection of the ankle joint (case 28).

There is, therefore, sufficient evidence to say that if left to themselves feet with paralysis in the lateral compartment are in danger of developing gross ulceration sooner or later and the inversion deformity must be corrected. In early cases the inversion may be corrected by tibialis posterior transplantation but if there is any degree of fixed inversion subtaloid arthrodesis followed by tibialis posterior transplantation is required. Late cases commonly need lateral wedge resection or amputation (Fig. 5).

More than half the cases of complete foot-drop also develop gross ulceration in time. Many of these can be corrected by tibialis posterior transplantation and tendon achilles lengthening alone. Those with fixed inversion require subtaloid arthrodesis also, as do the cases with a fixed equinous (Fig. 6).

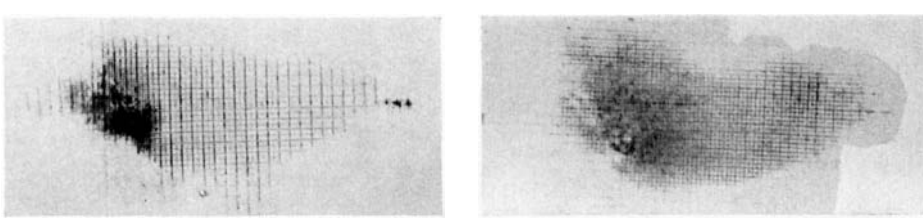


FIG. 6. Pre- and post-operative footprint of a patient with a fixed equinous showing changes after triple arthrodesis and tendo-achilles lengthening.

2. Deformities due to paralysis of the posterior-tibial nerve. This nerve carries the motor fibres for the four layers of intrinsic muscles on the plantar aspect of the foot. Some of these muscles flex the proximal phalanges of the toes and paralysis of them results in clawing. Some degree of clawing is almost universal in patients with feet that have been shod since childhood and gives rise to increased pressure under the metatarsal heads, but it is extremely uncommon in habitually unshod feet. In a personal series of walking footprints taken from 60 normal adults who habitually walk bare-footed no case of clawing was found and the only cases with any localization of pressure under metatarsal heads were those with a congenital retraction of the 5th toe (Fig. 7). DUCHENNE pointed out that the principal action of the toe extensors is on the proximal phalanges whereas the flexors act the most strongly on the two distal phalanges. Balance is maintained by the action of the intrinsics which are flexors of the proximal phalanges and extensors of the others. When the intrinsics are weak or paralysed the proximal phalanges are hyper-extended and the others flexed (LELIÉVRE) (Fig. 8a). HICKS drew attention to the fact that the plantar fascia is attached at its anterior end to the bases of the proximal phalanges with a result that when the proximal phalanges are hyperextended increased tension is set up in the fascia; the plantar fascia is completely inelastic so that the result is to shorten the distance between the metatarsal heads and the Os Calcis (Fig. 9). The plantar fascia forms the hypothenuse of a triangle of which one of the other two sides is formed by

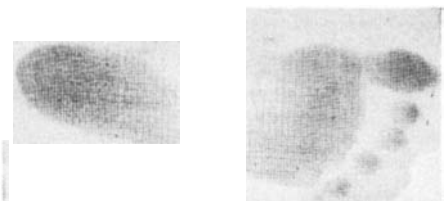


FIG. 7. Footprint of a normal habitually unshod foot.

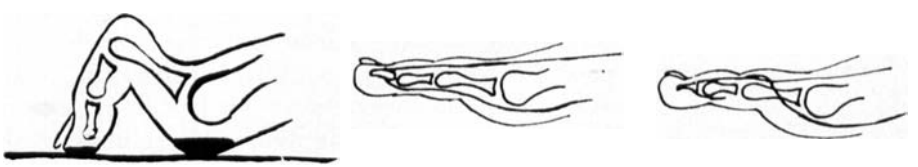


FIG. 8a. Diagram of a claw toe (after Leliévre).
FIG. 8b. Diagram to show operation for claw toe correction.

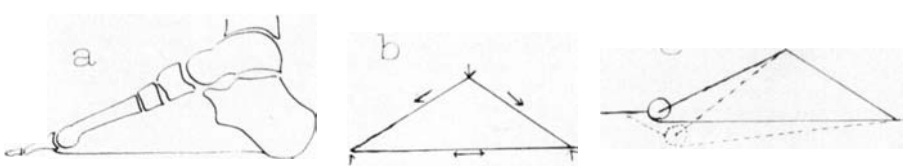


FIG. 9. (a) The plantar fascia. (b) The foot as a triangle of forces. (c) The windlass effect.

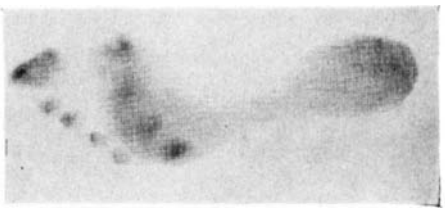
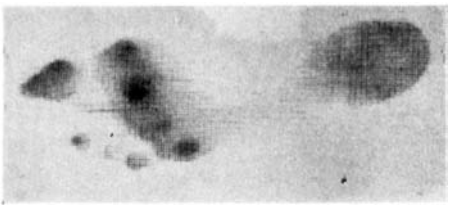


FIG. 10 a. Walking footprint of one of us (W.F.R.).



b. Walking footprint of the same foot after the second toe had been strapped into a position of dorsi-flexion.

the Os Calcis and the Talus and the other by the Navicular, the Cuniform and the Metatarsal bones. The result of shortening the hypotenuse is a decrease in the angle between the other two sides. The position of the apex of the triangle is fixed by virtue of the body weight pressing down on it. The position of the posterior angle is also fixed for the same reason so that the only movement which can take place is a downward movement of the anterior angle which represents the metatarsal heads. That this takes place can be confirmed by holding a finger under the metatarsal heads of a normal foot and strongly dorsiflexing one of the toes (Fig. 10). Besides resulting in increased weight bearing under metatarsal heads clawing also places the tissues of the fore-foot under tension. This considerably reduces their resilience and their resistance to trauma. The increased trauma due to clawing (Fig. 8a) results in ulceration in many cases and the clawing should be corrected if possible before ulceration occurs. The choice of operative procedure depends on five factors.

(1) The degree of clawing.
(2) The range of movement in the interphalangeal joints.
(3) The degree of damage already existing in the fore-foot.
(4) The presence, or absence, of other deformities.
(5) The particular toe or toes which exhibit clawing.

OPERATIVE PROCEDURES

(1) *For Mobile Claw Toes*

In all cases with mobile toes and relatively minor skin damage an active correction by transposition of the flexor digitorum longus on to the dorsum of the proximal phalanx is adequate (Fig. 8b). This operation is not suitable for correction of clawing of the hallux and in all cases if this digit is clawed it should be treated by fusion of the I.P. Joint (Fig. 11).

(2) *Rigid Claw Toes*

(a) With the fore-foot relatively intact. In these cases the brunt of the trauma has fallen on to the tips of the toes and the fore-foot is relatively undamaged. Fusion of the I.P. joints of the toes is recommended.

(b) With the fore-foot scarred. In cases where the scarring is limited to the skin and there is little or no damage to the subcutaneous tissue the

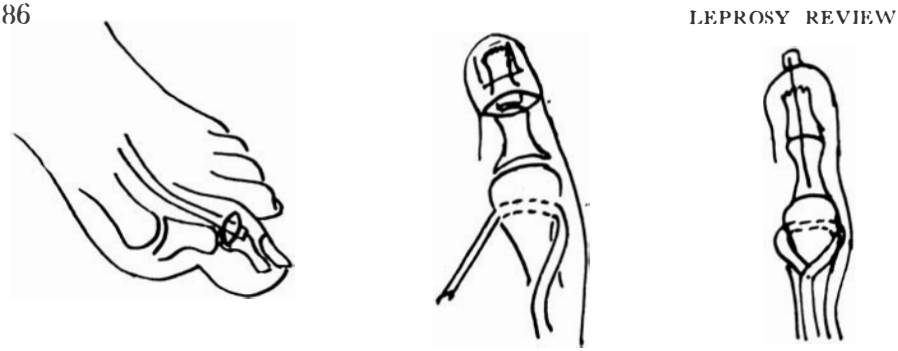


FIG. 11. Jones' correction of clawing of the great toe (redrawn from Duvries' surgery of the foot).

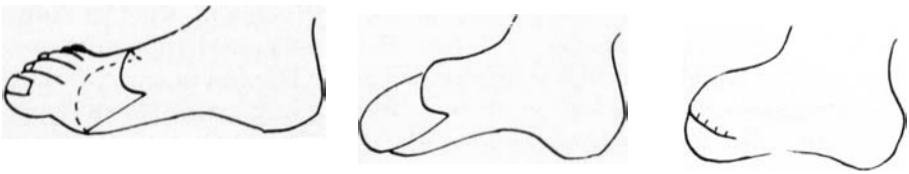


FIG. 12. Forefoot amputation redrawn from Lewin 'The Foot and Ankle'.

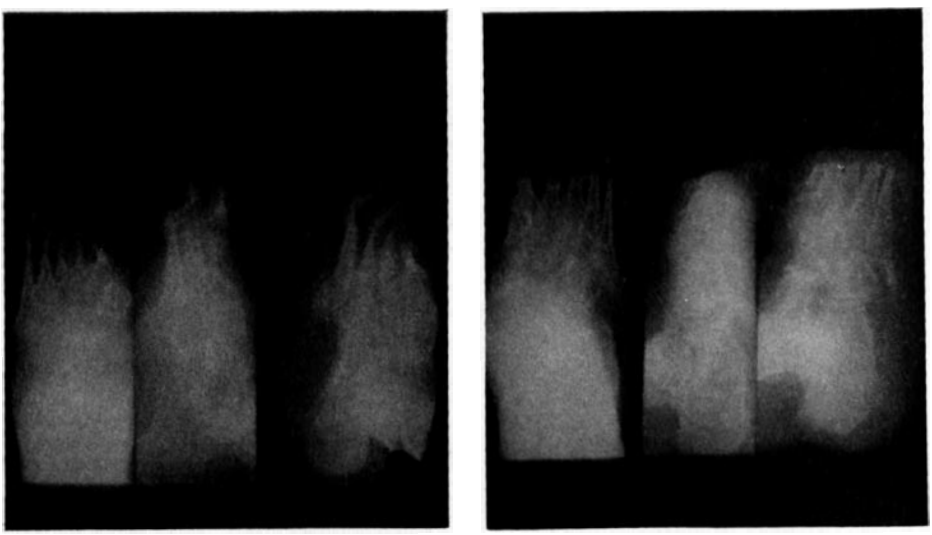


FIG. 13. X-ray to show pre- and post-operative findings in a case of fore-foot amputation (case 4).

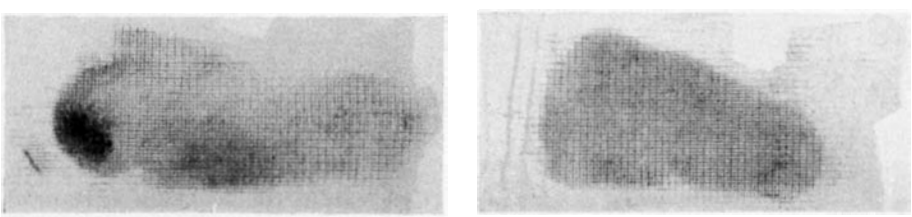


FIG. 14. Pre- and post-operative footprints in a case of fore-foot amputation.

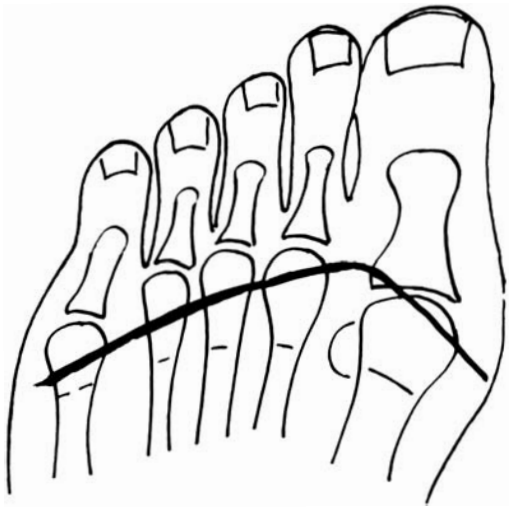


FIG. 15. Resection of metatarsal heads – Hoffmann's operation (after Clayton).

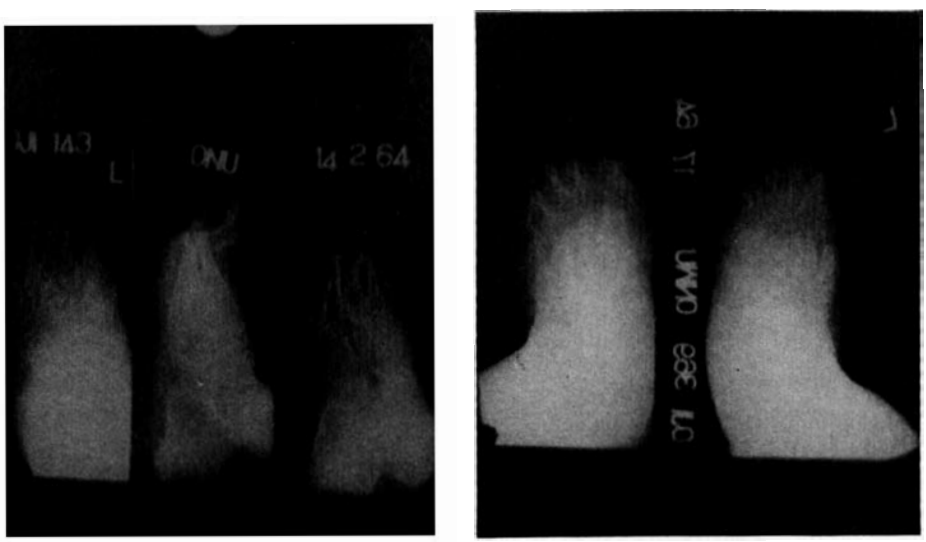


FIG. 16. X-ray to show pre- and post-operative findings in a case submitted to Hoffman's operation.

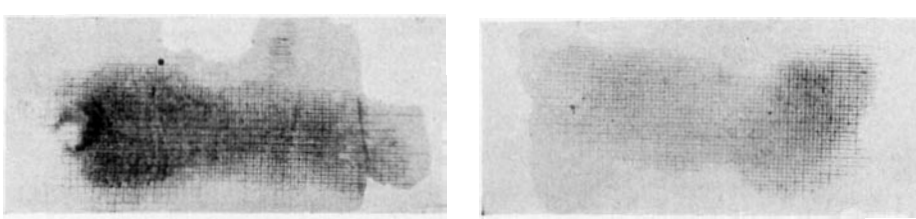


FIG. 17. Pre- and post-operative footprints in a case of Hoffmann's operation (same case as was used for Fig. 16).

procedures described above together with the provision of resilient soled footwear will be adequate. Wherever there is more severe scarring with loss of subcutaneous tissue and damage to the underlying metatarsal heads there is no doubt that the most satisfactory procedure, where more than one metatarsal head is involved, is amputation of the fore-foot proximal to the metatarsal heads (GASS) (Figs. 12, 13 and 14). This operation gives a short well padded foot which is easy to fit with shoes and which is most unlikely to re-ulcerate. Indeed, most Leproseria contain a few patients in whom this has happened 'spontaneously' as a result of recurring ulceration and who, once the toes have disappeared and the metatarsal heads have been absorbed remain ulcer free. Unfortunately many patients are reluctant to submit to fore-foot amputation but we have found that a useful substitute is resection of all the metatarsal heads through a dorsal transverse incision (Figs. 15, 16 and 17). Patients like to retain their toes for aesthetic and, no doubt, deep psychological reasons but from a functional point of view the toes in these cases are useless and from the shoe workshop's point of view they are nothing but a nuisance. Nevertheless, the result of the operation if properly done in terms of freedom from subsequent ulcers is very gratifying.

(c) Proximal phalanges grossly hyperextended, fore-foot relatively intact. In these cases excision of the proximal phalanges of toes 2 to 5 through a plantar incision is the procedure of choice (Fig. 18).

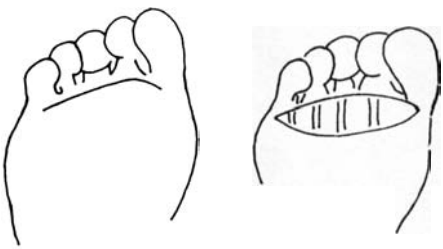


FIG. 18. Incision for excision of proximal phalanges.

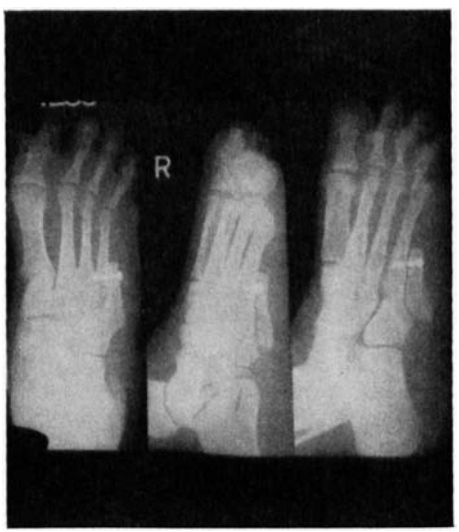


FIG. 19. X-ray to show post-operative findings in a case of excision of 5th metatarsal head.

(3) *Ulceration and Scarring limited to one Toe only*

(a) Hallux. The 1st metatarsal head alone should almost never be removed. If it is quite impossible to prevent re-ulceration under the hallux with footwear on account of the poor quality of the scar then all the metatarsal heads should be excised.

(b) The 5th Metatarsal Head. This metatarsal head can be removed without seriously damaging the foot but it is recommended that the metatarsal should be cut off quite short or else re-ulceration is likely at the tip of the metatarsal stump (Fig. 19).

(c) Heads 2–4. Where one of the other metatarsal heads is involved DUVRIES' operation which involves removal of part of the head and implantation of the long extensor tendon into the neck of the metatarsal has been found very satisfactory (Figs. 20 and 21).

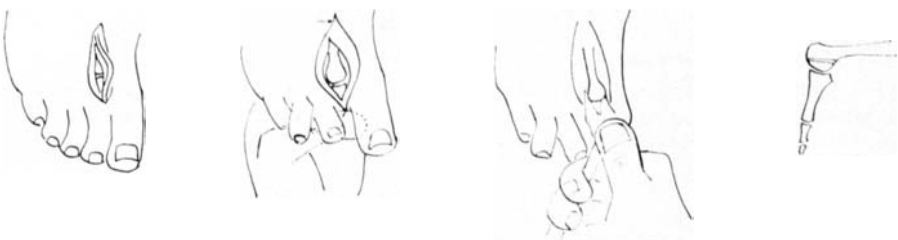


FIG. 20. Duvries' operation for partial removal of metatarsal head (from surgery of the foot by Duvries).

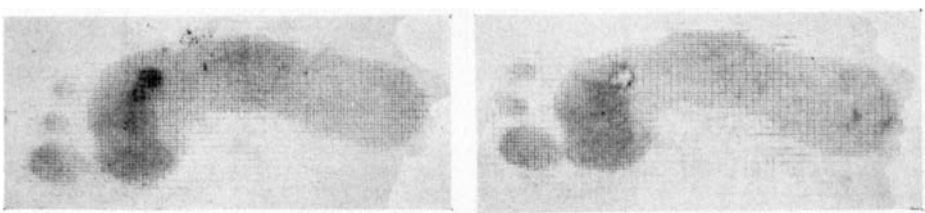


FIG. 21. Pre- and post-operative footprints in a case of Duvries' operation (case 22).

(4) *Ulceration not Associated with Specific Deformities*

(a) Proximal Phalanx of Hallux. A high proportion of normal individuals' weight bear on the proximal phalanx of the hallux (Fig. 8) and in some leprosy patients this results in ulceration and scarring. In my experience the only way to prevent re-ulceration in all cases, except those in which the skin only is involved, and these are very rare for usually the long flexor tendon is destroyed at the time of the 1st ulcer, is to excise the proximal phalanx leaving its base intact. A more satisfactory procedure is to amputate the toe, again, leaving the base of the proximal phalanx in position.

(b) Heel Ulcers. Fortunately the majority of heel ulcers are not associated with calcaneal involvement and will remained healed if the patient is provided with resilient soled footwear. If X-rays are taken many patients with heel ulcers show a calcaneal spur but unless it is clear that this spur is related to the site of the ulcer, and in most cases it is not, it should be left alone. If the spur is related to the site of the ulcer then it may be removed through a heel flap. This operation needs to be done

very carefully and the flap must be dissected subperiostally in order to conserve the structure of the heel pad. In the few cases in which there is gross osteomyelitis of the calcaneum or in which a thin scar becomes fixed to the calcaneum amputation must be resorted to. In selected cases use can be made of the dorsal skin of the foot to form a flap to cover the end of the stump.

NOTES ON OPERATIVE TECHNIQUES

Complete and slightly different accounts of these procedures can be found in the references given.

(1) *Tibialis Posterior Transposition.* This is the procedure of choice for correction of foot-drop and the inversion associated with it. It should be noted that it is the inversion component of the foot-drop which is most dangerous. The aim of the operation, therefore, must be primarily correction of the inversion. The correction of the foot-drop is almost

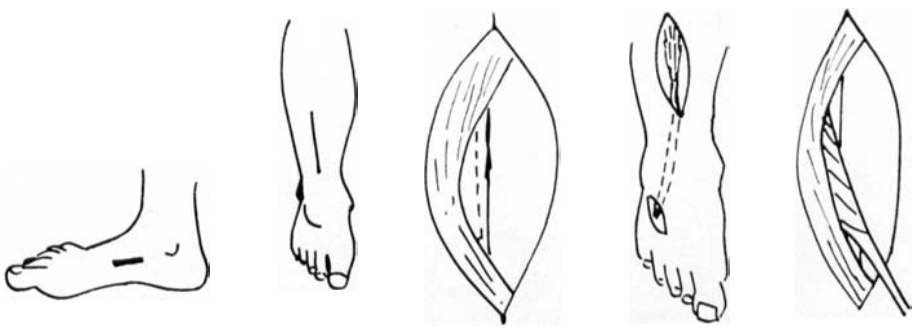


FIG. 22. Anterior transposition of the tibialis posterior, interosseous route. (Modified from Leléivre.)

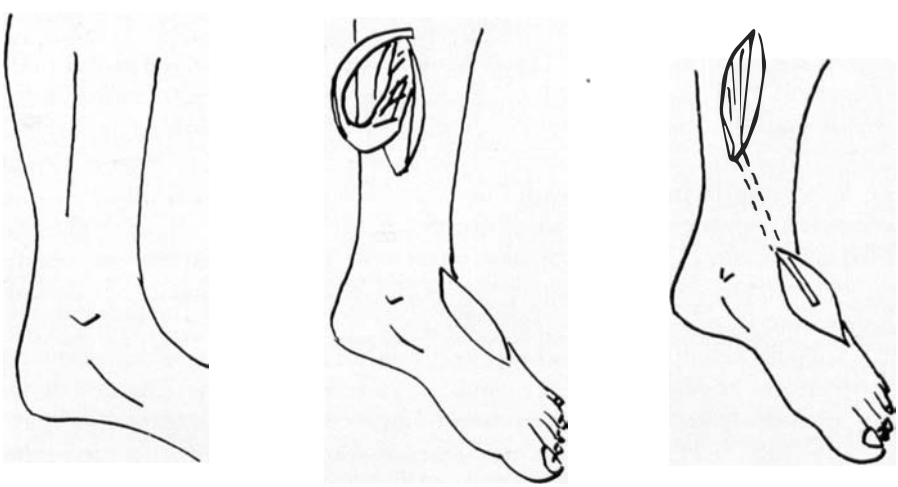


FIG. 23. Anterior transposition of the tibialis posterior, circumtibial route. (Modified from Lewin.)

incidental. Fortunately the Tibialis Posterior is never affected in leprosy and is always available for transposition. The basis of the procedure is to divide the Tibialis Posterior tendon at or near its insertion in the tubercle of the navicular and to re-route the tendon so that it crosses the ankle in front of the axis of the joint and to re-insert it on to the dorsum of the foot. (Figs. 22, 23.) The muscle in its normal site is a plantar flexor and inverter of the foot (Fig. 4) and in its new position is used as an everter and dorsiflexor. Considerable re-education is, therefore, necessary and this must begin pre-operatively. The patient must be taught to isolate the muscle and learn to contract it without contracting the calf muscles at the same time. Re-education is not easy pre-operatively. Post-operatively it may prove impossible. Provided the patient can isolate the muscle exercises designed to strengthen it are usually not necessary. The range of movement of the ankle joint should also be measured pre-operatively. If with the knee flexed the foot cannot be dorsiflexed to at least 75° a tendo-achilles lengthening will be required. Experience has shown that for patients with simple unheeled shoes the best range of movement of the ankle is from 85° to 95°. As approximately 10° of dorsiflexion is lost during the post-operative period in the average case it is necessary to set the foot up dorsiflexed at an angle of 75° during the operation.

Method of Distal Fixation of the Tendon

The standard method of fixing the free end of the Tibialis Posterior tendon on to the dorsum of the foot is by bone insertion. In some cases of leprosy this has been followed by disintegration of the tarsal bones and in order to avoid this complication various forms of tendon anastomosis have been tried. None of these procedures, in my hands at least, gives as good a result as bone insertion and in patients with unstable subtaloid joints the results of procedures involving tendon anastomoses are bad. In addition tendon anastomoses are much more difficult to perform than bone insertion and finally mid-tarsal disintegration has been seen following such a procedure (Fig. 3). Mid-tarsal disintegration is also seen in leprosy patients with neuropathic feet who have never had surgery (PATTERSON and JOB) and it seems likely that the coincidence of operative procedures and mid-foot bone destruction is purely a fortuitous one. Bone insertion is, therefore, the procedure of choice.

Re-routing the Tibialis Posterior

Two routes are available for the Tibialis Posterior to pass from the calf to the dorsum of the foot. The tendon may be taken either between the tibia and fibula (Fig. 22), through the interosseous space or by a circumtibial path round the medial border of the tibia (Fig. 23). In the first instance, the tibialis posterior is taken through a longitudinal incision made over the distal 1″ or 1½″ of the tendon. A longitudinal incision is then made over the anterior compartment extending through the middle third of the leg. The Tibialis Anterior muscle is exposed and retracted laterally together with the anterior tibial vessels and nerves. A long incision is then made in the interosseous membrane and the post-Tibial muscle will then herniate or partially herniate through the cut into the anterior tibial compartment. The tendon is then identified and withdrawn. The tendon can also be withdrawn on the lateral side of the Tibialis Anterior but in this case care must be taken not to injure the Anterior Tibial Artery and Vein. From the anterior compartment the tendon is passed subcutaneously on to the dorsum of the foot for its attachment as far lateral and distal as is convenient. I have yet to see a foot in which inversion has been over-corrected by this procedure. Before passing the tendon subcutaneously it is useful to estimate the point at which its distal end will be when it has been re-routed and site the incision on the dorsum of the foot accordingly.

Secondly, the circum-tibial route (Fig. 23). The Tibialis Posterior is taken from its distal insertion as before and a longitudinal incision about 4″ long is made just behind the medial border of the Tibia with its distal end 3″ from the medial malleolus. The Tibialia Posterior tendon is identified, withdrawn and passed subcutaneously across the front of the ankle to the lateral border of the foot.

Method of Insertion

The best method of insertion is to make a hole in one of the lateral bones of the foot in such a position that when the foot is fully doriflexed and everted and the tendon is taut the end of the tendon will snug down into the hole. The tendon can be fixed in position best by a pull out wire.

(2) *Claw Toe Correction.* Toes 2–5. Through a long dorsi-lateral incision which curves at its base onto the dorsum of the toe the Flexor tendon sheath is exposed and opened. The Long Flexor tendon is taken near its insertion and withdrawn (Fig. 10). The skin is then retracted laterally so as to expose the Extensor tendon. The Long Flexor is threaded through the Extensor on the dorsum of the proximal phalanx and is then taken across the proximal inter-phalangeal joint and again threaded through the Long Flexor. It is sutured in this position with the I.P. Joint as near straight as possible with the tendon under full tension and with the ankle plantar flexed. Post-operatively the foot is immobilized in a below-knee plaster of Paris case with the ankle in neutral position for four weeks. This operation may sound a rather drastic procedure for claw toe correction but in fact we have yet to see a toe over-corrected by this method and have found that to anastomose the Long Flexor to the lateral band of the dorsal expansion is not adequate.

(3) *Excision of proximal phalanges.* Proximal phalanges of toes 2–5 can be quite easily excised through a plantar incision across the bases of these four toes (Fig. 18). The incision opens onto the middle of the proximal phalanx and from here the phalanges can be dissected out sub-periostially without interfering with the sub-cutaneous tissue at all. Using this incision it is often possible to determine by inspection that the base of the proximal phalanx is in some cases more closely related to the site of ulceration than is the metatarsal head.

(4) *Metatarsal Head Resection.* The object of this procedure is to shorten the metatarsals so that their proximal ends are behind the scarred area of the foot. The metatarsal heads can also be resected through a plantar incision at the base of the toes but we have found that the increased dissection necessary to remove the metatarsal heads sometimes leads to damage to the subcutaneous tissue under them. It is, therefore, recommended that metatarsal heads should be removed through a dorsal incision (Fig. 15). A slightly curved incision is made across the fore-foot over the metatarsal heads. And the flap so formed retracted. Longitudinal incisions are then made over each metatarsal head in turn. And the head and necks dissected out sub-periostially. It is easiest to begin at the 2nd metatarsal head and then proceed to the 1st and to the others in turn. The metatarsals must be shortened sufficiently to ensure that the new end of the metatarsal is proximal to the scarred area on the fore-foot. Care must be taken to see that the metatarsals are lined up in an even curve at the end of the procedure and that the underside of the cut bone is bevelled and that no fragment of loose bone remains (Fig. 16). Bases of the proximal phalanges can also be removed at the same time but this is not usually necessary. The skin is closed and the foot immobilized in a plaster of Paris cast for three weeks post-operatively.

(5) *Fore-foot Amputation.* In this procedure the object is to shorten the metatarsals so that their distal end comes to lie proximal to the scarred area of the fore-foot. In addition it is, of course, necessary for the suture line to lie on the dorsum of the foot so that in making the incisions the plantar flap should be approximately 1½" longer than the dorsal flap (Fig. 12, 13).

(6) *Duvries' Operation* (Fig. 20). This is the procedure of choice for dealing with the problem of one prominent metatarsal head. A longitudinal incision is made over the top of the head. The Long Extensor tendon is divided over the middle portion of the proximal phalanx and withdrawn. The incision is deepened and the metatarsal phalangeal joint opened and the head freed until by flexing the toe the metatarsal head can be

delivered into the wound. The underside of the head is then trimmed off flat. A hole is drilled in the dorsum of the head from side to side and the end of the Long Flexor tendon threaded through it and sutured to itself under full tension. After closing the skin incision the foot is immobilized in plaster of Paris with the ankle at 90° for four weeks.

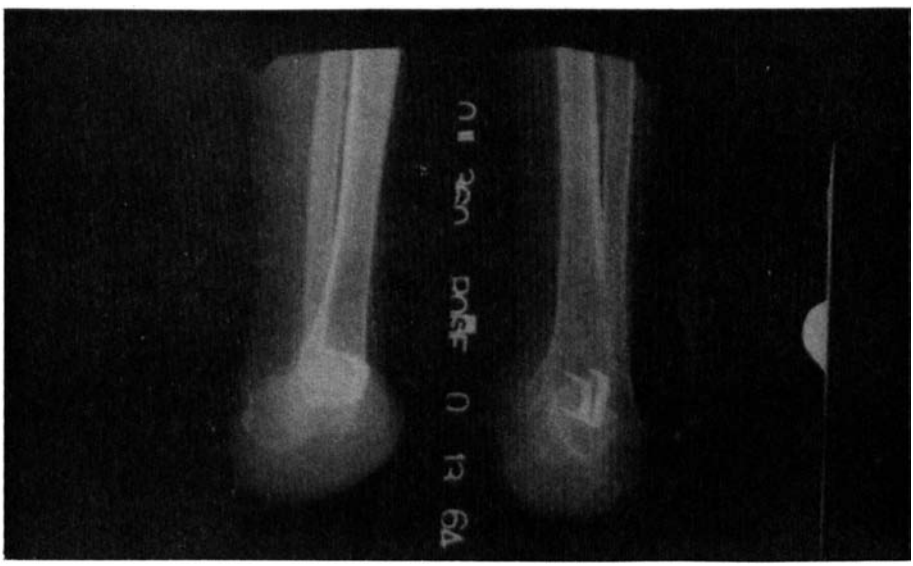


FIG. 24. X-ray in a case subjected to James' amputation.

(7) *Hind-foot Amputations.* The major areas of damage in leprosy are the fore-foot, the mid-lateral foot and the heel. So that mid-foot amputations have little if any part in the surgery of leprosy. But in cases where there is gross damage to the mid-foot it is sometimes possible to obtain an end bearing stump by hind-foot amputation. The most satisfactory stump is that given by a James' amputation in which the calcaneum is fused to the tibial mortice after excision of the talus (Fig. 24). This procedure avoids any interference with the heel pad and in cases where the heel is unscarred it is a worthwhile operation. More frequently a Symes' amputation is the only one feasible as there is often insufficient undamaged tissue to cover the calcaneum. The most important points in carrying out a Symes are (1) sub-periostial dissection of the heel pad off the calcaneum, and (2) on no account should dog-ears be trimmed. These will, in most cases, spontaneously absorb and if they do not can be excised once the wound is thoroughly healed. If the dog-ears are removed at the time of the initial operation sloughing of the heel flap is likely. For this operation to be worth while it is not necessary to have a completely intact heel pad but by varying the initial incisions and so excising scar tissue a useful if unconventionally shaped stump can be produced even from a partially scarred heel.

(8) *Long Leg Amputation.* When an amputation has to be performed in our view the leg should be left as long as possible. There are three reasons for this. Firstly, we have found it easier to fit satisfactory prosthesis to long stumps than to short ones. Secondly, a long stump enables the patient to make brief journeys on his stump, for example, at night, without having to search for his prosthesis, and thirdly, it is quite easy to shorten a stump if necessary. If the skin on the lower third of the leg is very poor or the circulation inadequate or there is some other complication such as deep venous thrombosis then, of course, amputation at a higher level is necessary.

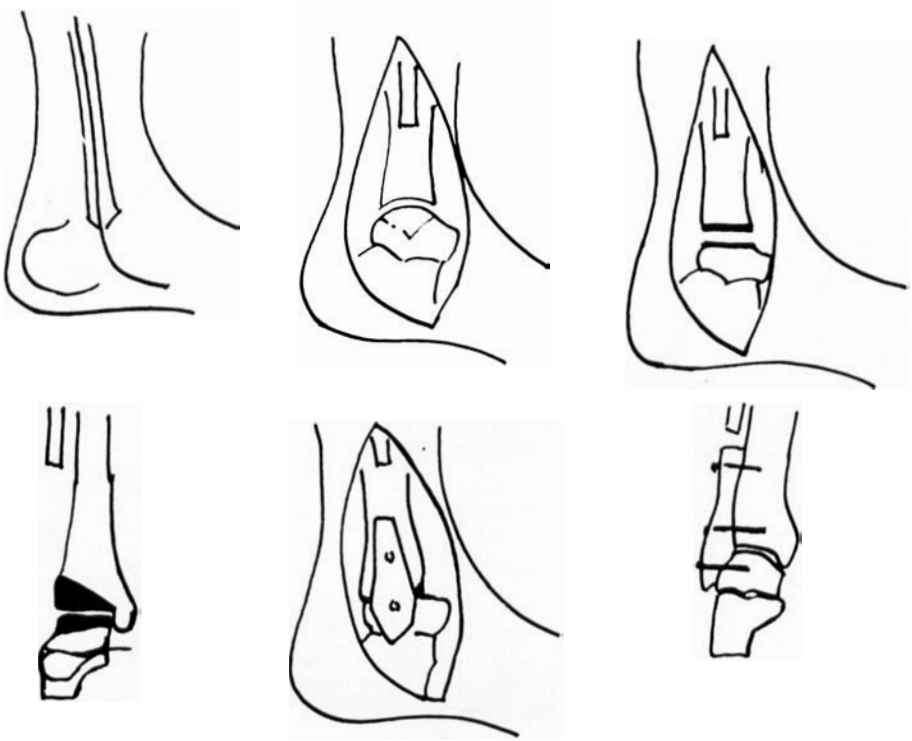


FIG. 25. Wedge resection of the ankle joint. (Modified from Leliévre.)

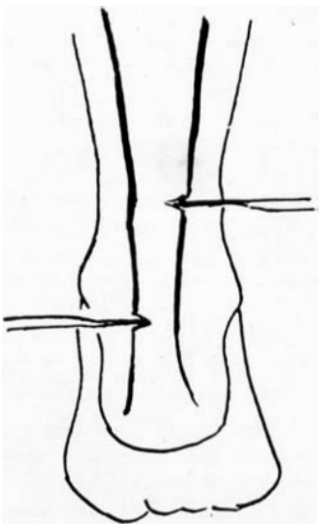


FIG. 26. Double subcutaneous tenotomy of tendo-achilles (after Lewin: The Foot and Ankle).

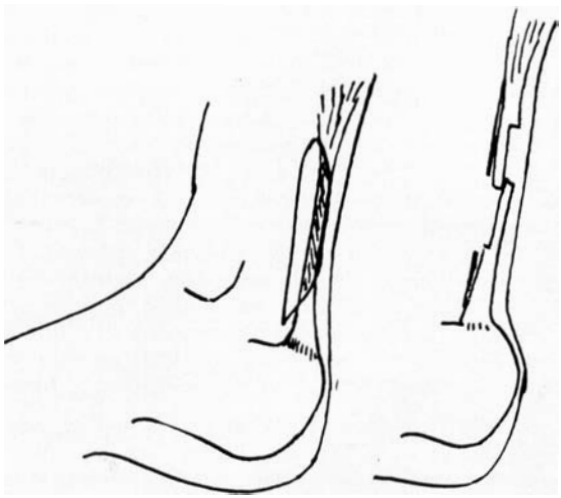


FIG. 27. Open operation for tendo-achilles lengthening (Brand).

(9) *Sub-Taloid Arthrodesis.* The purpose of this procedure in leprosy patients is to correct eversion. It is not recommended as a treatment for neuropathic changes in the mid-foot or hind-foot for in these cases fusion is extremely difficult to achieve and our view at present is that feet showing neuropathic changes in the bones are best left alone surgically and treated by means of prolonged bed rest. A formal triple arthrodesis is well described in standard text-books. We have not found internal fixation necessary and 12–16 weeks immobilization post-operatively is quite adequate.

(10) *Wedge Resection.* A J-shaped incision is made along the line of the tibia with the crook of the J extending forwards just below the tip of the lateral malleolus (Figs. 5 and 25). The lower third of the fibula is dissected free sub-periostially and removed thus exposing the ankle joint. An adequate wedge of the joint is then taken so as to correct whatever deformities are present. This is usually eversion and some equinous. If it is possible to close the wound without tension the segment of fibula may be used as an onlay graft but this is not essential. After closing the wound the limb is immobilized in a below-knee plaster of Paris cast for 12–16 weeks.

(11) *T.A. Lengthening.* If a minor degree of lengthening is needed this can simply be done by subcutaneous tenotomy. More extensive lengthening requires open operation (Figs. 26 and 27).

SUMMARY

This paper presents a series of 43 patients operated on to prevent recurrence of ulceration, in 1961–62. Based on this, and subsequent experience, operative proceedures for dealing with specific problems are recommended and details of operative techniques which have an application to foot problems in leprosy are described.

FOOTNOTE

The M.C.R. sandals are made with a micro-cellular rubber called Sorbocel (R.B.N.F.S.) obtainable through Bata shoe company.

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References

1. BAUMAN, J. H., GIRLING, J. P., and BRAND, P. W. Plantar Pressure and Trophic Ulceration. An Evaluation of Footwear. *J. Bone and Joint Surgery*, 45B, 652–673.

2. CLAWSON, D. K., and SEDDON, H. J. The Late Consequences of Sciatic Nerve Injury. *Ibid.* 42B, 213.

3. HARRIS, R. I., and BEATH, T. Canadian Army Foot Survey National Research Council of Canada.

4. HICKS, J. H. The Mechanics of the Foot. II. The Plantar Aponeurosis and the Arch. *J. Anat.*, **88,** 25–30.

5. PATERSON, D. E., and JOB, C. K. Chapter XXVI, *Leprosy in Theory and Practice.* COCHRANE and DAVEY (2nd Edition). John Wright & Sons, Bristol.

6. RICHARDS, R. L. Vasomotor and Nutritional Disturbances after Injury of Peripheral Nerves. Peripheral Nerve Injuries. Medical Research Council, pp. 25–56.

7. PRICE, E. W. Studies in Plantar Ulcers in Leprosy. *Leprosy Review*, **30** and **31.**

8. ROSS, W. F. Footwear and the Prevention of Ulcers in Leprosy. *Leprosy Review*, **33,** 202–206.
9. WARD, D. J. Footwear in Leprosy. *Leprosy Review*, **32,** 94.
10. GASS, H. H. (1938). 'A Study of the Results of Certain Surgical Procedures in Leprosy' Proceedings of Cairo Conference (1938).

Standard Texts

1. COCHRANE, R. G., and DAVEY, T. F. *Leprosy in Theory and Practice* (2nd Edition), Chapters XXVII, XXIX and XXX. John Wright & Sons, Bristol.
2. DUVRIES, H. L. *Surgery of the Foot.* The C.V. Mosby Company, St. Louis.
3. LEWIN, P. *The Foot and Ankle.* Henry Kimpton, London.
4. LELIÉVRE, J. *Pathologie du Pied.* Maison et Cie, Paris.

Special Texts

1. Foot-drop and Claw Toe Correction.

FRITSCHI, E. P., and BRAND, P. W. The Place of Reconstructive Surgery in the Prevention of Foot Ulceration in Leprosy. *International Journal of Leprosy*, vol. 25, 1.

2. Metatarsal Head Resection.

CLAYTON, M. L. Surgery of the forefoot in Rheumatoid Arthritis. *Clinical Orthopaedics*, **16,** 136–140.
HOFFMANN, P. An Operation for Severe Grades of Contracted or Clawed Toes. *Am. J. Orthop. Surg.*, **9,** 441, 1911.

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Clinical and Immunological Results obtained with the *Marianum* Antigen after more than Ten Years of Therapeutic Use

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In 1952 the experimental study of the Marianum antigen was begun at the St. Michel Leprosarium, Nden by DR. M. BLANC. His first report was on 457 patients in 1954. The results being satisfactory the antigen was applied in therapy for all new patients of the leprosarium. Since that time we estimate that about 1,500 patients have derived benefit from this therapy.

The present paper comprises a detailed report on the results obtained with combined treatment with sulphone and Marianum antigen over ten years.

The Mitsuda test is carried out on every patient before the treatment is begun, and the test repeated every year. Every six months bacteriological examinations are made. Using the customary technique, the patients every month receive one intradermal injection of 0.10 ml. of the Marianum antigen. This goes on for six months and is followed by a rest period of three months and then another course of injections is begun. The therapy is really a combined one with sulphone or other anti-leprosy drug, such as Ciba 1906, or Sultirene.

The results mentioned in this paper are expressed as such:

'Arrested' – no clinical nor bacteriological activity for one year;

'Much improved' — the clinical improvement is great, and the bacteriological examinations are negative for one year;

'Improved' — there is clinical improvement and bacteriological findings are still undecided;

'Stationary';

'Worse'.

The reactions to the Mitsuda are described in the usual way:

Negative (—), Slightly positive (+), Positive (++),
Highly positive (+++).

1.—*Results with* 266 *Actually Inmate Patients of the Leprosarium*

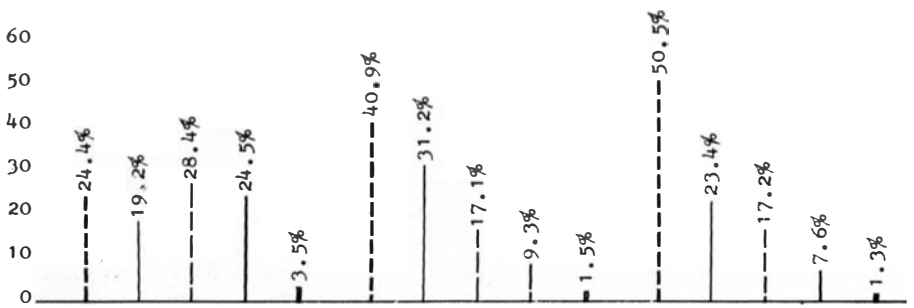
These consisted of 57 Lepromatous,
64 Tuberculoid,
145 Indeterminate,

and their classification of leprosy was seen and adjusted by DR. MARTINEZ, of WHO in March 1961

The present clinical results are:

	L.	*T.*	*I.*	
'Arrested'	14	26	73	= 113
'Much improved' ..	11	20	34	= 65
'Improved	16	11	25	= 52
'Stationary'	14	6	11	= 31
'Worse'	2	1	2	= 5

In relation with the type of case percentages are: (1)



a—arrested. b—much improved. c—improved. d—stationary. e—worse

Results can be summarized as follows:

Lepromatous = 72.1 per cent have benefited of the treatment, against 28 per cent without effect;

Tuberculoids = 89.2 per cent have benefited of the treatment, against 10.8 per cent without effect;

Indeterminates = 90.9 per cent have benefited of the treatment, against 8.8 per cent without effect.

For each of the above groups, the results are shown in relation with the number of injections received.

To achieve this result, we point out that in the three first groups of 'Arrested', 'Much improved,' and 'Improved', higher percentages are obtained from a large number of injections (between 30 and 40).

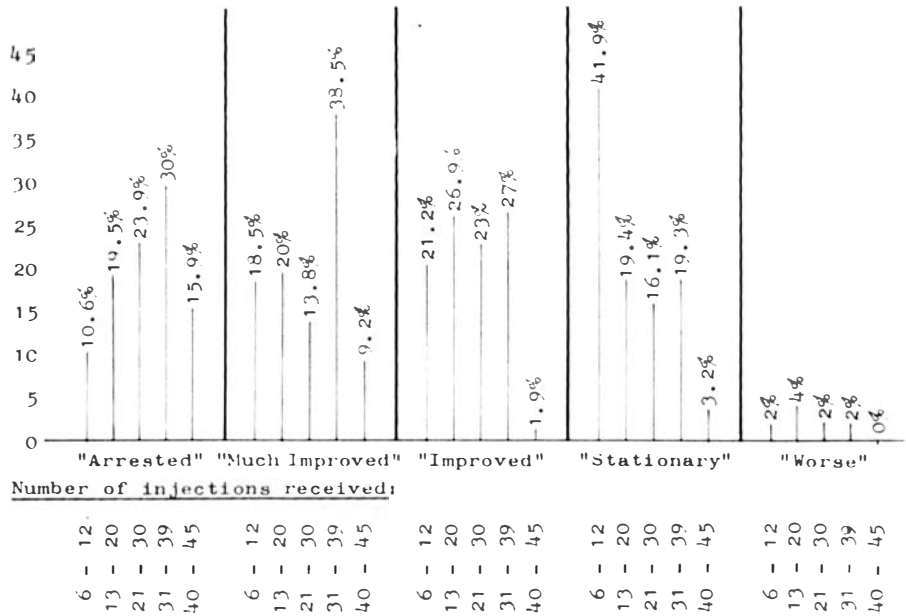
Immunological results – The regular giving of the Mitsuda test at the beginning of treatment and once a year enabled the study of the action of the Marianum antigen according to the type of the disease. The results mentioned hereto were checked at the end of 1962 or beginning of 1963.

To summarize; those who were positive before treatment (83 patients) remained positive in 97.5 per cent. Among the 117 negative before treatment, 101 cases became and remained positive, giving a percentage of 86.3 –. This figure corresponds with the clinical results where the number of 'Arrested' and 'Improved' cases gives 86.5 per cent. For most of the cases, positivation of the Mitsuda test is observed at the end of the

Summary of the Mitsuda modifications (I)

	Before	*After*	*Before*	*After*			*Before*	*After*		
	++	++	+	—	+	++	—	—	+	++
Lepromatous 57	5	5 = 100%	6			4 = 66.6%	46	3	2	39c = 84.8% 2v
Tuberculoid 64	37	37 = 100%	24		4	20 = 83.4%	3			3 = 100%
Indeterminate 145	41	39c = 95.1%	36	3	5	28 = 77.7%	68	4	2	59c = 86.8% 3v
Total	83	81c = 97.5%	66			52c = 78.7%	117			101c = 86.3%

(I) c = constant v = variable



second course of Marianum antigen, which consists of 12 injections, and maintained with a prolonged treatment.

2° – Results with 53 patients of Sangmelima Leprosarium, who had previously the Marianum antigen: these patients were four lepromatous, 10 tuberculoid, and 39 indeterminate.

The results were that 34 per cent were 'arrested', 39.5 per cent 'much improved', and 30.1 per cent improved.

The Mitsuda changes to positivation were 67 to 77 per cent to definite positive (++), and 28 negatives to positive (++) were noted. These results had been obtained on six to 12 injections in 47 patients, and 12 to 24 injections in six patients. A greater percentage of positive Mitsuda test might have been obtained with more courses of Marianum injections.

3° – Results with 30 patients, inmates of Nden Leprosarium, previously contacts of the patients. Of these contacts there were 11 children and 19 adults, of whom six children born at the leprosarium had received the Anti-Lepreux Vaccination (prepared with Mycobacterium marianum).

The group includes eight tuberculoid and 22 indeterminate.

The general results are 23.3 per cent 'arrested', 36.6 per cent 'much improved', and 40 per cent 'improved'. For the six children previously vaccinated four had no clinical nor bacteriological activity, one had no clinical activity, but variable bacteriology, and one still suspicious activity of patches.

The changes in the Mitsuda were that all six were positive (++) before, remained positive (++). Of seven who were mildly positive before (+), six became definitely positive (++), and of 17 who were negative, 13 became definitely positive (++).

4° –Considering results with 124 patients who received this treatment but left the leprosarium a short time ago, studying their condition at departure and assuming that their lack of returns signifies that their leprosy condition has not changed. The group includes: 18 lepromatous patients, 29 tuberculoid, and 77 indeterminate.

The clinical changes were 29 per cent much improved, 27.4 per cent improved, 40.4 per cent stationary and 3.2 per cent worse. About half of these patients had been treated for one to six years.

The changes in the Mitsuda were that of 29 who were positive before, 27 remained positive (++). Of 32 who were mildly positive before (+), 29 became positive (++), and of 63 who were negative before, 56 became positive (++); therefore percentages vary between 88 to 92.7 per cent in the various groups of the three main types.

5° – An extra study of the Mitsuda reaction was made in 412 patients who had received benefit from the Marianum therapy, but who left the leprosarium at an early date. This group included 75 lepromatous, 94 tuberculoid, and 243 indeterminate.

Results of the positivation of Mitsuda test summarize as follows:

	Before	*After*	
Lepromatous:	71 —	61 ++	= 85.9 per cent
(75)	4 +	4 ++	= 100.0 per cent
Tuberculoid:	93 ++	85 ++	= 91.1 per cent
(94)	1 —	1 —	
Indeterminate:	155 —	133 ++	= 85.8 per cent
(243)		25 —	= 14.2 per cent
	88 ++	82 ++	= 93.1 per cent
		6 —	= 6.82 per cent
Total:	227 —	194 ++	= 85.4 per cent
		33 —	= 14.5 per cent
	185 ++	171 ++	= 92.4 per cent
		14 —	= 7.5 per cent

6° – *Results obtained with the Anti-Lepreux Vaccin (prepared with Mycobacterium marianum) on 'contacts' living at the leprosarium.* There were 92 children living in permanent contact with their parents and other inpatients of the leprosarium. These were all examined, as all of them had one or more courses of the Anti-Lepreux Vaccin; since 1960, on the suggestion of DR. GAY PRIETO, they also had been given prophylactic sulphone. Study was made of 73 children and the others discarded, as they were only a few months old, and had had too short a prophylactic treatment, although none showed any sign of leprosy. Most of the children in the group studied had been born at the leprosarium; 13 only were aged 7, 8, 10 or 12 years at the time of their admission with their parents. Although they already had a few years contact with their parents, they showed no difference from the others in respect to changes in the Mitsuda reaction.

After six to 30 injections of Anti-Lepreux Vaccin this group showed considerable modification of the Mitsuda:

Before	*After*	
65 —	54 ++	= 83.0 per cent
	5 +	
	6 —	
1 +	1 +	
7 ++	7 ++	

Eighty-eight of these children show no clinical sign of leprosy; four only have a suspicious patch with no clinical or bacteriological activity.

Twenty-four adults 'contacts' have also been examined. Contrarily to the children we notice that 11 of them had a Mitsuda (++) before vaccination, which was a sign of acquired immunity. Eight were slightly (+) and five (–). On these 13 'contacts' the positivation of Mitsuda is 76.9 per cent.

In comparison, data from Bafia Leprosarium was studied concerning child contacts, and it was found that over nine years the percentage of infection was 4.75 per cent.

The children were given B.C.G. a few weeks after birth, as well as prophylactic sulphone (Disulone). As is seen in Section 3 of this paper, six children vaccinated with the Anti-Leprosy Vaccin out of 30 contacts developed Leprosy. In fact out of 225 child contacts observed over 10 years to whom the Anti-Leprosy Vaccin was given, the percentage of infection was 2.6 per cent.

SUMMARY

This paper reports on the records of 885 patients treated with the Marianum antigen. Of 349 inpatients of the leprosarium, 86.5 per cent received benefit from this therapy as 'arrested' or 'improved'; 12.1 per cent remained 'stationary', and 1.4 per cent 'worse'.

Concomitant study of the Mitsuda reaction showed that in 349 inpatients a previously negative Mitsuda became positive in 81.7 per cent (there were originally 159 negative Mitsudas) and remains positive to date; 10 per cent attained a positive but unstable Mitsuda. Most of the conversion of the Mitsuda was noted after 12 injections of Marianum antigen, and remained constant.

Of the 536 patients studied, there were 357 with a negative Mitsuda. These became positive in a percentage of 85.9 per cent.

In contrast with the general opinion of leprologists that the first few years of sulphone therapy results only in a low percentage of positive Mitsuda, the present study has shown that even in 1½ years, a high percentage is obtained. This means at the end of the second course of injections of Marianum antigen.

The authors think that this antigen is a good adjuvant in therapy, which reinforces the resistance of the patient, and permits a better and safer response to chemotherapy. The absence of relapse over 10 year period is most striking, and is attested by MISS M. TH. PROST, Directress of St. Michel Leprosarium.

From the point of view of prophylaxis, the authors draw attention to the low percentage of infection among permanent contacts, and cite the 92 children, who might have been considered to have a high susceptibility to infectious diseases.

References

M. BLANC and M. TH. PROST (1953). Present therapy of leprosy at St. Michel Leprosarium, Nden (Cameroun). St. Med. and Anat. Clin. de Lille, séance du 26/11/52. *Journée des Sciences Médicales de Lille*, No. 3–71è, An. 15/3/53, p. 129 à 139.

M. BLANC and M. TH. PROST (1953). Our projects and our realisations regarding the prophylaxis of leprosy. St. Med. and Anat. Clin. de Lille, séance du 26/11/52. *Journal des Sciences Médicales de Lille*, No. 3–71è, An. 15/5/53, p. 139 à 142.

M. BLANC and M. TH. PROST (1953). New orientation with regard to the prophylaxis of leprosy in Cameroun. Congrès International de la Lèpre, Madrid (1953).

M. BLANC, M. TH. PROST, and SR M. SUZANNE (1953). The influence of a Mycobacterium isolated from leprosy tissue, strain Chauviré, on the Mitsuda reaction. *Bull. Soc. Path. Exot.*, 8/7/53, No. 6, p. 1009 à 1014.

M. BLANC, M. TH. PROST, LEMAIRE, E. KUNA, et J. M. NKOA (1953). Immunological study of the antigen Chauviré. *Ann. Biol. Clin.*, No. 10–12, Oct. Déc. 54, p. 1 à 6; *Journal Soc. Méd. de Lille*, 1953, No. 12, pp. 384–390.

M. BLANC, M. TH. PROST, MICHEL, LEMAIRE, E. KUNA, J. ESSELÉ, and J. M. NKOA (1954). Clinical and therapeutic study of an antigen prepared with the Mycobacterium marianum, and its application on 457 leprosy patients. *Ann. Biol.*, No. 10–12, Oct. Déc. 1954, p. 1 à 12; *Intern. Journal of Leprosy*, January–March 1955, vol. 23, No. 1, p. 23 à 31.

M. BLANC (1955). Study of the reactions caused by the antigen Marianum. Sté Méd. and Anatomo-clinique de Lille, séance du 16 Mars 1955. *Journal des Sciences Méd. de Lille*, No. 5–73è, Ann. Mai 1955.

M. BLANC and M. TH. PROST (1955). Clinical research on the immunological and therapeutic properties of the antigen Marianum. *Rev. Colon. de Médecine and Chirurgie*, No. 241, 27e. Ann. 15/12/55, pp. 226–236.

M. BLANC (1956). New orientation of the anti-leprosy campaign in Cameroun. Congresso Internationale per la difesa e la riabilirazione sociale del 'Lebbroso' Roma, p. 418 à 426, vol. II, 16–18, Aprile 1956.

M. BLANC and M. TH. PROST (1956). Leprosy survey in the Schools of the district of Dja and Lobo – Prophylaxy with the anti-lépreux Vaccin sm. sm. Congresso Internationale per la difesa e la riabilitazione sociale del 'Lebbroso' Roma, 16–18, Aprile 1956, Atti e memorie V. II, p. 138 à 149.

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Letter to the Editor

Apartado-aereo 1708
Cali-Colombia
August 25, 1964

Sir,

I wish to put forward for discussion the hypo-iodic basis of leprosy. Incidentally I have records of the use of a thyroid-depressant drug, Tapazole or Methimazole, in the therapy of more than 100 leprosy patients, and at present am studying them from the point of view of high dosage, and hope to report this soon in detail. In the meantime, it suffices to say that lepromatous patients have an exceptionally good tolerance for very high doses of Tapazole.

With regard to the wider question of epidemiology, I have inquired of S. G. BROWNE of Uzuakoli, E. Nigeria, with wide experience of leprosy in the Congo, and of GLYN GRIFFITHS of N. Rhodesia, and their kind replies have been on the same lines. I quote here the remarks of S. G. BROWNE: 'With regard to your thesis that the incidence of leprosy tends to be low in goitrous districts, the facts are quite against it in Congo. There was no adverse relation between goitre and leprosy. The gross figures for incidence in such districts, and the individual patients with both diseases, give no support whatever for your ideas. The populations concerned are Hamite as well as Bantu, lived on varied diets, mainly millet, or mainly maize, with cassava and plantain as their source of carbohydrate. They are facultative vegetarians as well as meat-eaters.'

'There are other foci of goitre in the ex-Belgian Congo, but in each of these the incidence of leprosy is lower than in the Wamba-Pawa district first mentioned. In these districts also the incidence of goitre would seem to have no bearing on the incidence of leprosy and *vice versa*.'

While having every respect for this opinion and for the work of BROWNE and GRIFFITHS, I feel that reports from other epidemiological areas are needed to fill out the picture, such as in marine areas and in hypo-iodic areas such as the mountain location of the new leprosy centre contemplated in India by Japan. I also feel that direct evidence should be obtained of the iodine status in water, soil, and air of important areas in Africa such as of great lakes and rivers, as well as marine areas.

I should be grateful if leprologists everywhere would write in to the Editor of *Leprosy Review*, to DR. ROBERT COCHRANE, London, or to me concerning facts and figures and opinions.

ARTHUR O'BYRNE

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