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PRINCIPAL CONTENTS

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Contents

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Editorial

A paper by OLITZKI and GERSHON on Maintenance of Cytopathic Activity of *Mycobacterium Leprae* in Eagle's Medium Supplemented by Mycobacterial Extracts, in Israel J. Med. Sci. 1, (5), 1965.

Because of its great importance and interest to the whole world of leprosy, we draw attention to the Abstract of this paper published in this issue page 127. There has been quite a large amount of notice taken of it in the general press in which it is claimed that it is now possible to reproduce the bacillus of leprosy in the test tube. We have been fortunate in being able to publish at once a comment by Dr R. J. W. Rees of the National Institute of Medical Research, Mill Hill, London N.W.7, and this follows immediately.

COMMENTS ON THE PAPER BY *Olitzki* AND *Gershon* BY DR R. J. W. REES.

In spite of the importance of the successful transmission of leprosy to experimental animals, first demonstrated by Shepard, the full impact of modern scientific techniques cannot be applied to the study of leprosy until the causative organism is grown *in vitro*. Therefore every claim of the successful cultivation of *M.leprae* must be investigated with the greatest vigour, but also with the greatest care bearing in mind the multitude of past claims, none of which have withstood critical analysis nor have been substantiated by other workers. Olitzki and Gershon claim to have maintained the viability and the multiplication of *M.leprae* in a complex medium (Eagle's medium) used in tissue culture with the addition of an extract prepared from an unnamed strain of atypical *mycobacterium*. They tried the addition of a mycobacterium extract because many years previously Twort had conclusively demonstrated that another mycobacterium, Johne's bacillus, could only be grown *in vitro* with the addition of an extract of mycobacterium (*M.phlei*). The present claim is based on *M.leprae* obtained from only one patient inoculated into Eagle's medium plus the mycobacterial extract and maintained for 5 months. From this culture up to 3 subcultures were made which were maintained for periods of 1–3½ months. In all cultures increased opacity was noted after periods of 30 days, even with inocula diluted to 10^{-6}. In addition to these observations the author showed that suspensions of *M.leprae* isolated directly from a patient were toxic for, and produced death of, mouse monocytes in tissue culture and that suspensions of bacilli obtained from the cultures throughout all passages produced a similar toxic effect on the monocyte cultures. They concluded that the persisting cytotoxic effect was evidence of persisting viability of *M.leprae* in the primary culture and in the sub-cultures. Unfortunately the data presented, as a preliminary communication, did not include any direct counts on the number of acid-fast bacilli at the beginning and end of each culture period nor did it include a description of the stained bacilli recovered from the cultures and whether the bacilli appeared healthy. Although the suspension of *M.leprae* used in these experiments failed to grow on Loewenstein's medium no mention is made of checking this point with the acid-fast bacilli recovered from the cultures after several months. This latter check is of the greatest importance in order to exclude the possibility that the cultures were contaminated with a recognised and cultivable strain of mycobacterium.

Therefore with the limited data presented it is impossible to be sure that the authors have cultured *M.leprae*. However, a sufficient number of definitive tests are now available to identify *M.leprae* and it is essential that these should be applied to the bacilli isolated from the cultures of Olitzki and Gershon before making a definite claim of having cultured *M.leprae* The crucial tests include the behaviour of the organism in the foot pads of mice where it is now possible to make a direct bacteriological and pathological comparison with *M.leprae*, to exclude carefully the possibility that the organism grows in ordinary bacteriological media and to prepare a lepromin from the organisms

and test it in patients with lepromatous and tuberculoid type leprosy (a test already proposed by the authors). Furthermore it will be essential to apply the same cultural methods to suspensions of *M.leprae* isolated from other patients with leprosy. It is hoped that the authors will actively pursue their important studies and also undertake the essential tests suggested, and where necessary provide cultures of their organisms to others working in the field of leprosy research.

2 NOTICES:

We once more draw your attention to the following:

(a) The new subscription to *Leprosy Review* is **£2 per annum from 1st January, 1966.**

(b) The Editorial office of *Leprosy Review* will be at **6, Hillcrest Avenue, Pinner, Middlesex, England, from 1st April, 1966,** with Dr Ross Innes continuing as Editor.

(c) Mr Stanley Stein wishes to give subscribers of Leprosy Review a complimentary year's subscription to his publication. Send your request to the Editor, STAR, U.S. Public Health Service Hospital, Carville, Lousiana 70721, USA.

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Onset and Pattern of Deformity in Leprosy

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I INTRODUCTION

Leprosy, one of the oldest afflictions of man, is still the object of ignorance and prejudice. Its social stigma originates from fear, primitive, rational or religious as Edwards (1964) pointed out, and the opprobrium attached to it is largely emotional. Ever since the discovery of Hansen's bacillus (*Mycobacterium leprae*) in 1872, no acepted method of *in vitro* cultivation has been reported. The epidemiology of the disease is still largely unknown; its mortality is low; its classification disputed; the evolution of its clinical features and its pathology not fully understood.

The long duration of leprosy, its uncertain incubation period, the normal life-span of its various clinical types without reaction, do confer on the disease a rather special status among chronic infections. In fact, as Skinsnes (1965) rightly observed, 'Leprosy is unique. It is unique in the peculiarly intense reaction it has called forth in diverse societies. It is also unique in the complex of immunity and pathology which gives it its identity'.

No other disease indeed entails such human and social consequences in the community where it is prevalent, as expressed by so much distress and unhappiness to the patients and their families, whose numbers run into millions. Because of the slow action of anti-leprosy therapy, hence the long duration of treatment, the non-discovery as yet of new and more effective drugs and/or immunizing agents to provide a short-cut to leprosy control, results are not spectacular, and indeed cannot be so in a disease with such characteristics and disposing of such limited tools.

Yet deformity is the overriding feature of leprosy: it not only throws an ever increasing burden upon society, by reducing the patient's capacity for work, but is also responsible for the dread and special horror with which the disease is still regarded. In both instances, its relentless crippling is associated with a progressing loss of function, and this does carry more weight when, as is tragically the case, resources are limited and facilities for the care of the disabled are wanting in general. Furthermore, the handicapped leprosy patients, in the absence of any other occupation, become mendicants as is commonly seen in some South East Asia countries. Their growing numbers do represent a loss to the community in terms of productive labour.

The problem of deformity in leprosy is of such magnitude and growing importance that it will retain our sole attention here, for apart from the clinical aspects, it is obvious that in many developing countries where the disease is endemic, the loss of man power does hamper the rise of economic level, the standard of living and the education of the community.

Brand (1960) commented realistically as follows: 'The disease of leprosy is not itself directly responsible for most of the deformities that are attributed to it. It often removes the sensation of pain, and so allows the patient to damage and to deform himself. The more closely one studies the natural history of deformity, the more one finds that secondary changes and sometimes almost incidental damage and infection have been the cause of most of the mutilation from which these patients suffer'.

One is thus led to the realization that more light is needed into the problem and, with this in mind, the present study arises out of an experience of some 10 years in the field, in West Africa, Burma and India, covering thousands of patients among whom a detailed study of 700 was made in relation to the onset and the clinical pattern of deformity of the extremities produced by leprosy.

It is difficult to overestimate the significance of deformity as part and parcel of a disease which has come to occupy the unique position that it has throughout the centuries. What is

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the size of the problem? Estimates of the prevalence of leprosy itself vary widely, yet the latest consensus of opinion is that there are about 20 million patients throughout the world. The following tables will give an idea.

How many of these are disabled and/or deformed by the disease? Again, estimates vary and the following tables will provide some figures that would allow an appraisal of the socio-economical implications of leprosy:

TABLE 1

Estimates of the World Prevalence of Leprosy

Author	*Year*	*Estimates*
Chaussinand	1950	5 million cases
International Congress of Leprology, Madrid	1953	9 million
Doull	1956	3 to 12 million
International Congress of Leprology, Tokyo	1958	15 million
Guinto	1960	2 million registered cases
Dharmendra	1960	5 million
Gay Prieto	1961	10 million
International Congress of Leprology, Rio de Janeiro	1963	20 million

TABLE 2

Registered, Estimated and Treated Patients in the World*

Continent	*Registered*	*Leprosy patients* *Estimated*	*Total*	*Treated* *%estimated*
Africa	1,708,855	3,865,000	969,717	24·6
America	179,813	361,531	95,723	25·1
*Asia***	939,729	4,169,268	747,827	17·9
Europe	16,519	51,944	9,186	17·7
Oceania	9,281	36,265	4,235	11·7

* WHO figures produced at the 14th Session of the UNICEF/WHO Joint Committee on Health Policy in April 1965, Geneva, but based on information received so far.
** China (Mainland) not included.

TABLE 3

An Estimate of the Frequency of Disabilities

Total No. of cases	*Percentage Disabilities*	*Author*	*Country*
10,550	32%	Doull (1954)	Philippines
—	25%	Diniz (1959)	Brazil
—	16·4%	Convit (1959)	Venezuela
—	16%	Montestruc (1959)	Martinique
—	25%	Kinnear-Brown (1959)	Uganda
—	11·3%	Gay-Prieto (1959)	Spain
1,294	31·6%	WHO Leprosy Advisory Team (1959)	Northern Nigeria
2,487	12·8%	Guinto	Philippines
400,000+	7·5%	Lechat (1960)	Ex-Belgian Congo
4,025	25%	Mallac (1960)	Gambia
1,000	29·2%	Chodankar (1962)	Bombay
200,000+	10%	Mallac (1962)	Burma
4,961	28·8%	Susman (1963)	Gambia
16,000+	15%	Mallac (1963)	Pogiri, India
18,000+	29·7%	Glyn Griffiths (1964)	Zambia
500,000+	15%	Mallac (1964)	Andhra Pradesh, India

TABLE 4

Estimated Disabilities in the World*
Disabilities

Continent	Degrees 1–5	Degrees 2–5**
Africa	1,106,015	422,297
America	169,844	105,890
Asia (excluding China)	1,683,550	1,018,350
Europe	17,187	8,014
Oceania	11,685	5,270
Total	2,988,281	1,559,821

* WHO figures produced at the 14th Session of the UNICEF/WHO Joint Committee on Health Policy in April, 1965, Geneva, but based on information received so far.
** Classification adopted by the WHO Expert Committee on Leprosy, 1960.

How long does it take for deformity to appear after the earliest stages of leprosy? How much grace have we in which to apply therapeutic precautions? In short, what is the latent period between the first appearance of leprosy and the onset of deformity, and what is the clinical pattern of the development of deformity in leprosy? These questions seem to the author to be the keys to the mastery of the disease in its most dreaded aspect, i.e. deformity and/or mutilation. It was to answer them that the present study was undertaken.

Two problems are interrelated and have to be studied together. The first is the latent period: how long does it take for leprosy to cause deformity? But this question cannot be answered without a proper study of the different patterns of deformity which the disease does produce: what are these patterns, and how do they emerge?

Three terms are used throughout this study which require definition: disability, deformity and mutilation. It is accepted that these emotive words are not the ideal for an objective study, but there does not appear to be any acceptable alternative. By *disability* is meant loss of function; it connotes mainly sensory loss (anaesthesia) which is a severe state of affairs particularly in the hand. By *deformity*, a deviation from the normal shape or size of the part; by *mutilation*, a partial or total absorption of the part. These last two terms imply motor paralysis and/or direct damage to the tissues by the lepra bacilli, both of which being the chief concern of this study in relation to the extremities.

II *Review of the Literature*

Over the past 50 years or so, workers in the field of leprosy have studied the local and general manifestations of the disease in minute details, but it seems to the author that they have ignored two cardinal features, namely the latent period of 'germination' of leprosy leading to deformity and the clinical pattern of the latter, particularly the sequence of involvement of the digits.

From time immemorial leprosy has been feared for its contagiousness. Through the centuries the image of the disease has been the image of its late and neglected stages, i.e. deformity and/or mutilation. It is still an image associated with all that is loathsome and unclean. The late James Doull (1963) put it aptly: 'Although more than 88 years have elapsed since the announcement of the discovery of the leprosy bacillus, an accepted method of *in vitro* cultivation has not been reported, nor has any one succeeded in producing disseminated leprosy in an experimental animal. These are obviously the great handicaps to progress and it is interesting to speculate on why greater progress has not been made. Some of the reasons are as follows: there are inhibitions resulting from the failures of able scientists in the past. New prospects, especially in the study of ricketsia and viruses, appear to offer quicker returns. There is a public demand for the investigation of the diseases of greater magnitude, such as cancer, heart and mental diseases. There is the significant fact that leprosy is uncommon in most countries that have become leaders in scientific endeavours. Add to these the world-wide shortage of scientific personnel and

the increase in demand of the physical sciences and we have a formidable list to be overcome before any comprehensive attack can be made on the basic problem of leprosy'.

Today, with the introduction of the sulphones in 1947, leprosy demands a wider study because it is now curable. With this new intent, attention is being paid to basic research, the various clinical forms of the disease, its bacteriology, its classification, its pathology and immunology. Much has been written on the epidemiology of leprosy, on its reactional phases, on rehabilitation and leprosy control. While all these are of great value, it is the author's opinion that the time scale of this dynamic disease is of equal importance once the cure becomes possible.

Brand (1958) made a classic study of the pathology of deformity. According to him most of the terror surrounding the word 'leper' comes from the idea of gross deformity and open sores. Although a great deal of work needs to be done before one can claim to understand the pathology of deformity, Brand suggested as a beginning that it be divided into Primary and Secondary deformity.

The neurological patterns of leprosy in terms of disorders of sensation, nerve involvement, trophic, vascular and ocular changes have been studied by Wayson and Badger (1929), Bresani-Silva (1956–7) and Argenta (1961). Andersen (1963) elaborated the patterns of anaesthesia and of motor paralysis of neural leprosy. However, these studies have not been concerned with the progression of the disease so much as with different syndromes atdifferent and unrelated stages.

Whereas it was held that neural signs in leprosy are due to an inevitable and irreversible degeneration of the affected nerves, Chatterjee (1955 and 1964) proved the important point that this is not so. Neural signs are due more to vascular than to neural changes and that these are not irreversible. Encouraging results were obtained in both the prevention and correction of polyneuritic signs, as well as in the eradication of their residual ones.

When it comes to the aetiology of bone changes Murdock and Hutter (1932) were of opinion that neural or neuro-vascular factors were the main causes of bone changes in leprosy. More recently, Oberdöffer and Collier (1940) have pointed out that disturbances of nutrition which cause atrophy of both the cortex and cancellous bone can also follow the paralysis of muscles inserted into them, secondary to the neurological lesions. Cuervo *et al* (1944) agreed that the majority of bone lesions are neurotrophic but have shown that in the lepromatous variety they are due to the direct action of *M.leprae* on the osseous tissue. Again, while Faget and Mayoral (1944) agreed that bone changes are neurotrophic lesions to which trauma is contributory, they found that it is long standing leprous neuritis and disturbance of the nutritional function of the affected nerve which is the main cause of bone absorption. Others have drawn attention to the part played by disturbances of circulation, anaesthesia, pressure and changes in acid-base equilibrium brought about by the metabolism of *M.leprae* (Cooney and Crosby 1944, Barondes 1946, Esquerra-Gomez and Acosta 1948, and Barnetson 1951). Da Veiga (1947) regarded osteoporosis as being due primarily to vascular disturbances; Wozonig (1956) that non-specific trophic disturbances secondary to neural involvement are the causes of bone changes in leprosy.

Thus recent studies have largely refuted the classical concept that bone destruction in leprosy was a simple process of anaesthesia and trauma and that those neural or neuro-vascular factors were the main causes of bone changes. Patterson (1958) puts forward a soft tissue chronic non-specific infection as the main cause of bone absorption and Brand (1958) that one of its main causes is the misuse of the finger tips. Early diagnosis should enable the reversible process of trabecular absorption and decalcification to be at worst halted, and at best reversed. None of these observers, so far, have indicated how long is the latent period during which such treatment might be applicable.

Further evidence which emphasizes the importance of early diagnosis in leprosy has come from the study of the role of ischaemia in the pathogenesis of bone changes. Vascular lesions of the type specific to leprosy have been found in more than half of the cases studied by Fite (1941): the organisms were present in endothelial cells of terminal vascular loops, sometimes forming a mass projecting into the lumen. Clearly, at this stage, the pathological process should be reversible if the diagnosis could have been made.

Two approaches to using more specialised methods for establishing the early diagnosis at this potentially reversible stage have been exploited, on the one hand by study of the alteration in peripheral nerve function by means of measurements of chronaxie*; on the other hand, the detection of early vascular changes by oscillometry, skin temperature studies, and arteriography.

In 1942, Freitas and Virgilio utilised electrical stimulation as a means of precise localization of the peripheral nerve lesion in leprosy. This technique was developed by Dubois (1951) and Raedermecker (1952) into a useful means of establishing early diagnosis of minimal nerve lesions: they found alterations in the chronaxie before there were any other clinical signs. Lechat (1960) extended this work further. He found that although there was no relationship between the incidence of osseous changes and the clinically paralysed groups of muscles, there were definite alterations in chronaxie, indicating latent motor weakness in these cases with or without anaesthesia. However, Lechat did find evidence of distal absorption of digits and disorganization of joints in cases without even these alterations in chronaxie. This work would suggest that chronaxiemetry may be useful, but not a completely reliable early screening method in the diagnosis of the onset of deformity.

Barnetson (1950) made a detailed study with oscillometry on neural cases. He showed that local blood vessels had not lost their dilatability but vaso-motor control was defective, though he thought that the neurotrophic changes depended on more factors than loss of vaso-motor control. Gokhale (1959) studied the temperature changes in the feet after blocking the autonomic system, and found that vaso-motor control was defective in the ulcerated feet of the leprosy patients. Bang and Diep (1958) showed by arteriography that there is an ample blood supply even in advanced cases. Patterson (1955) showed narrowing of the digital arteries in extreme deformity. But neither this author nor the subsequent studies of Chardome and Lechat (1957), Lechat (1960) and Basu (1962) have thrown light on the question whether these arterial changes are primary, or secondary to the tissue destruction. On the other hand, measurements of skin temperature (Barnetson 1950, Katsumi 1955, Lechat 1960, Montestruc and Hydronimus 1961) suggest that there is a distinct lesion, amounting to loss of vasodilator control in response to heat, in these patients.

On the plea that arterial changes in the form of an endarteritis causes slowing of the peripheral circulation and prolonged venous return in the affected parts, Lechat (1960) puts forward the hypothesis that absorption of the bone develops when the nervous mechanism controlling vasoregulation of the skin is damaged, the terminal vascular system is no longer able to adapt the flow of the peripheral blood to the demands that follow multiple and constant neuro-traumatisms at the level of the tips.

So far, neurotrophic and vascular factors have been considered in the aetiology of bone changes, but metabolic factors have been studied since the pioneer work of Honeij (1917) who tried to find out whether there was an abnormality of calcium metabolism in leprosy and whether changes in serum calcium concentration was correlated with the degree of mutilation. Honeij reached no definite conclusions. Later groups of workers have reported a hypercalcaemia in cases improving on treatment, with a hypocalcaemia during reactional phases (Badenoch and Byron 1932, Esquerra-Gomez 1948).

Not all workers have agreed with these findings. For example, Wooley (1931) found a hypocalcaemia particularly among the several neural cases; similar findings were obtained by Wooley and Ross (1933), Nishikawa (1932), Ross (1955) and Dhople and Magar (1962).

Still others have found no significant alterations in the serum calcium, or else a complete want of correlation between the stages of the disease and the calcium level, namely Venkatasubramanian (1941). Yet, Lindui and Ross (1957) found no relationship between blood levels of calcium and the evolution of deformity, the studies of Lechat (1960) and those of Languillon and Boissan (1960) confirming these results.

No correlation can be found between the phosphorus levels and the state of the disease (Wooley and Ross 1932, Ross 1955, Lindui and

*By chronaxie or chronaxiemetry is meant the shortest duration of a current necessary for excitation when its strength is twice the rheobase. Readings vary from muscle —1/10 to 1/4 of sigma or thousandth of a second—In the presence of neuritis, nerve or muscle involvement, readings increase 50 to 100 times the normal value or more.

Ross 1957, Lechat 1960, Languillon et al. 1960 and Dhople and Magar 1962). It seems then that calcium and phosphorus estimations are of no practical value in detecting the subsequent progress of the disease.

Similar disappointment has followed the use of serum alkaline phosphatases. Venkatasubramanian (1941) was the first investigator; he thought that the mutilations, more marked in the 'pure' neural form, could be reflected in an increase in alkaline phosphatases, but Ross (1955) found it within normal limits in both the mutilated and the non-mutilated patients. Four years later, Imaeda (1959–60) found that it was raised in the leprosy patients and suggested that phosphatase was related to nerve destruction. Lechat (1960) also found a difference in the phosphatase levels between the mutilated patients and the healthy individuals of his group, but found no correlation with the state of the bone lesions. Dhople and Magar (1962) found that the average values were within normal limits in the early stages of the disease, but were increased in severe tuberculoid cases. As an index to early diagnosis, the alkaline phosphatases prove useless.

Wooley and Ross (1932) were among the first to investigate the role of blood proteins in leprosy. They concluded that in the disease the albumin-globulin ratio was lower and the globulin fraction increased against controls. These findings were confirmed by Muelling et al. (1960), Mille and Papa (1961) and Torsuceva (1962). Lechat (1960) made a serial blood protein analysis in mutilated and non-mutilated patients, and in normal controls. He found that the beta-globulin fraction was lower in the mutilated patients than in the non-mutilated, a difference which was also influenced by age and the duration of the disease. Gamma-globulins were significantly increased in both groups of patients with leprosy as compared with the healthy group, being higher in the mutilated cases.

Radiological investigations have been used in the attempt to chart the progress of leprosy, and to assist in the classification of its various manifestations. Studies (reviewed above) have been made into the aetiology of bone changes. Classification of the end stages of the disease based on radiological appearances is now securely based, thanks to the publications of Kadrenka and Merdje (1938), Erickson and Mayoral (1950), Casacci (1950), Barnetson (1950), Cherlinzoni and Piratsu (1952), Mut Mut (1955), Chardome and Lechat (1955), Patterson (1956), K'ung Ch'ing-teh *et al.* (1957), Negre and Fontan (1957), Zamudio (1960), Languillon and Carayon (1961) and Lechat (1962).

It is clear, however, that sequential studies in which the clinical picture is correlated with the radiological ones, and in which the progress of the disease in its different patterns can be followed in time, are still wanting. In recent years the radiological changes in leprosy in response to treatment have been studied by Erickson and Johansen (1948), Basset and Schneider (1950), Lechat and Chardome (1957), and Languillon and Boissan (1960). From these it is also clear that treatment may, if too late, fail to halt the progress of deformity once it has begun. Although reiterating the importance of early diagnosis, these radiological findings have not been concerned with the way different patterns, having different rates of progression, can emerge early in the course of the illness.

Mathur and Saxena (1965) observed that some cases of deformity, particularly the early ones, treated with intraneural Priscol give satisfactory and promising results on the whole, both in correction and prevention.

Lennox (1964) forwarded a useful classification of leprosy foot deformities, stressing the close relationship of deformity to trophic ulceration.

As a by-product of their general findings in Nigeria, Cameroon, Thailand, Burma and the Philippines, the WHO Leprosy Advisory Team (1965) found out that the longer the period prior to treatment, the greater the proportion of disabilities.

From all the above investigations, no single biochemical or radiological test emerges as an accurate or reliable method which will replace clinical examination in early diagnosis and in the recognition of early deformity, and the role of chronaxiemetry is seen to be supplementary to clinical investigation. Throughout all these studies, however, the theme emerges again and again that many of the early lesions, both of peripheral nerves and small blood vessels are reversible, and that if reversed, bone destruction with deformity and mutilation could be prevented.

For this reason, it seems all the more important

for the physician to know how urgent the treatment is or how long does leprosy take to reach a stage when treatment becomes salvage, not cure.

None of the studies briefly reviewed here have dealt specifically with the onset of deformity of the extremities and with the sequence of events regarding the affected digits, yet the information is fundamental to any large scale attack upon leprosy. Planning and execution of public health measures dealing with the disease must in our generation be directed not only against the curable stages but also against its most dreaded and seemingly inexorable aspect; deformity. Before such plans can be made, the planner must be provided with his basic data.

Part and parcel of the study of the evolution of the clinical picture of leprosy must be data on the onset of deformity and the sequence of events concerning the affected digits. As this review of the literature has shown, there have been a great many studies on the disease as a static entity, many descriptions of the end stages of the disease process. The gap in our knowledge is the *timing*, the dynamic study of leprosy. To fill this gap is the object of the present study.

III *Plan and Description of the Investigations*

There is a delay between the first signs of leprosy and the manifestation of deformity: how long is this delay and how long this period of 'germination' of the disease leading to deformity?

From the foregoing review of the literature, it appears that one out of four or five patients with leprosy will develop some sort of deformity or other: how soon in the case of a male patient still in his teens? How soon if it is a case of Tuberculoid leprosy? How long after the start of the disease when concerning a female in her early thirties, and a lepromatous type? How long after the start of the disease when it is a male in his late thirties of the indeterminate or borderline group? In other words, how long does deformity take to develop as between the clinical forms, the sex and in the different age groups? How long also from one extremity to the corresponding and/or the opposite one(s)?

From the start it was obvious that the problem could only be studied in patients who would be reliable witnesses and who could answer a number of questions. Few patients met these conditions in the parts of Africa where the author started his career, and also in India. In Burma, however, there is a better school attendance in urban and rural areas and more patients were available who could understand and co-operate on this study. It was then in Burma that the necessary informations were obtained: 700 patients with deformity were examined in detail.

TABLE 5

Choice of Out-patients and Their Percentage Examined

	Shwebo	Monywa	Total
Number of villages	17	28	45
Population	18,571	19,073	37,644
Prevalence of leprosy per mille	26	28	
Number of patients examined	473	456	929
Number found with deformity	138	157	295
Percentage of deformity	29·3%	30·4%	—

TABLE 6

Choice of In-patients and Their Percentage Examined

	No. of Patients Examined	No. found with Deformity	Percentage Deformity
St. John Leprosy Asylum Mandalay	432	249	53·3%
Leprosarium (Gvt) Mandalay	104	81	77·8%
Leprosarium (Gvt) Shwebo	41	28	68·2%
Leprosarium (Gvt) Monywa	62	47	75·9%
Total	639	405	

The plan of investigations was as follows: visits to enough villages where the disease was known to be prevalent, with the purpose of examining registered cases at the clinics and in their homes as well as in many leprosaria. A detailed questionnaire and a recording of findings was used for every patient found with deformity of the extremity(ies). Analyses of these results built up a composite picture of its clinical patterns.

From the above two tables, we have the following:

Total No. of Out-patients examined	929
Total No of In-patients examined	639
Grand total of patients examined	1568
Total No. found with deformity	700

IV *Results of the Investigations*

A. *The Onset of Deformity*

— Between Clinical Forms
— Per Sex
— Per Age Group

TABLE 7

The Onset of Deformity Between Clinical Forms

	Lepromatous	Tuberculoid	Indeterminate	Borderline
No. of patients with Deformity Hands &/or Feet (x)	279	321	25	75
Duration of the disease in years (y)	3849	3914	261	1017
Duration of the Deformity in years (z) ..	1299	3013	128	668
Onset of Deformity in years (t)	2550	901	133	349
Average (t = y — z ÷ x)	9·1 yrs	2·8 yrs	5·3 yrs	4·6 yrs

OBSERVATIONS

Deformity occurred an average of 2 years and 9 months after the start of the disease in the tuberculoid cases; an average of 4½ years in the borderline; an average of 5 years and 3 months in the inderterminate and an average of 9 years and 1 month in the lepromatous.

The time of onset of deformity was thus about 3 times as long in the lepromatous than in the tuberculoid; twice as long in the lepromatous than in the borderline; twice as long in the indeterminate as in the tuberculoid.

TABLE 8

The Onset of Deformity per Sex

	Males	*Females*
No. of patients with Deformity (Hands &/or Feet) (x)	443	257
Duration of the disease in years. (y)	5497	3664
Duration of the Deformity in years. (z)	3157	1696
Onset of Deformity in years. (t)	2340	1968
Average (t = y — z ÷ x)	5·05 years	7·6 years

OBSERVATIONS

Men were earlier affected, an average of 5 years after the start of the disease as opposed to an average of 7½ years in the case of women.

TABLE 9

The Onset of Deformity per Age Group

	0–5	6–10	11–20	21–30	31–40	41–50	51+
No. of patients with Deformity in years (x)	—	—	91	277	195	98	39
Duration of the disease in years. (y)	—	—	591	3210	3122	1540	873
Duration of the Deformity in years. (z)	—	—	323	1677	1770	815	520
Onset of Deformity in years. (t)	—	—	268	1352	1352	725	353
Average (t = y — z ÷ x)	—	—	2·7 yrs	5 yrs	6·8 yrs	7·3 yrs	9·05 yrs

OBSERVATIONS

In this study, no deformity was observed in the 0–5 and the 6–10 age groups.

Deformity appeared an average of 2 years and 8 months in the 11–20 age group (13% of the total); an average of $5\frac{1}{2}$ years in the 21–30 age group (39·5%); an average of 6 years and 9 months in the 31–40 age group (27·8%); an average of 7 years and 4 months in the 41–50 age group (14%); and an average of 9 years in the 51 years old onwards (5·5%).

Thus the younger the age group on the whole, the earlier seemed the onset of deformity.

B. *The Clinical Pattern of Deformity*

(i) *Deformity of the Hand:*

Comparative Frequency of Deformity of the Hand between Clinical Forms
Comparative Frequency of Deformity of the Hand per Sex
Comparative Frequency of Deformity of the Hand per Age Group
Pattern of Deformity of the Hand
General Mode of Involvement of the Affected Fingers
Average Interval between Affected Fingers of both Hands.

TABLE 10

Comparative Frequency of Deformity of the Hand between Clinical Forms

	Lepromatous	Tuberculoid	Indeterminate	Borderline
Total No. of examined patients	627	632	186	123
% incidence of the type of the disease ..	39·4%	40%	11·2%	7·7%
Total No. with deformity of the hand(s)	266	308	25	75
% incidence of deformity of the hand(s)	42·2%	48·5%	13·8%	60·9%

OBSERVATIONS

Deformity of the hand(s) was proportionately more frequent among the Borderline cases (60·9% representing 7·7% of the total).

Next among the Tuberculoid, 48·5% (40·4% of the total).

Then among the Lepromatous, 42·2% (39·4% of the total) and finally among the Indeterminate, 7·7% (11·2% of the total).

TABLE 11

Comparative Frequency of Deformity of the Hand per Sex

	Total cases examined	Grouping	Sex Incidence	No. Found with Deformity	%Incidence of Deformity
Males	1568	953	60·8%	410	43·02%
Females		615	39·2%	264	42·7%

OBSERVATIONS

Among the total of 953 males examined, 410 (43%) showed deformity of the hand(s).

Among the total of 615 females examined, 264 (42·7%).

Men were thus hardly more affected than women with regards to the deformity of the hand(s).

TABLE 12

Comparative Frequency of Deformity of the Hand per Age Group

	0–5	6–10–	11–20	21–30	31–40	41–50	51+
No. of patients with Deformity	—	—	91	277	195	98	39
No. of patients with Deformity of the Hand(s)	—	—	87	267	184	97	39
%Deformity of the Hand(s)	—	—	95·6%	96·3%	94·3%	98·9%	100%

OBSERVATIONS

Deformity of the hand appeared in the 11—20 age group.

It was more common in the 21–30 age group and thereafter declined steadily with age.

Irrespective of the age group concerned, the percentage deformity of the hand(s) was proportionately very high.

TABLE 13

Pattern of the Deformity of the Hand

	Right	Left	Bilateral	Total	%Incidence
A2.* Mobile claw-hand(s)	77	68	91	236	29·8%
A3. Contracture	69	95	164	328	41·4%
A4. Partial absorption	44	25	122	191	24·1%
A5. Gross absorption	7	5	24	36	4·5%

* As per the WHO Classification (1960).

OBSERVATIONS

The commonest type of deformity of the hand was contracture (40·4%).

Next, mobile claw-hand (29·8%).

Then partial absorption of the fingers with useful thumb remaining (24·1%) and finally, gross absorption of the fingers (4·5%).

Bilateral involvement was appreciably more common than unilateral involvement:

Bilateral Involvement (Mixed Types)	=	518 (74·3%)
Unilateral ,, ,, ,,	=	156 (22·1%)
No Involvement of the Hand(s)	=	26 (3·6%)
Total	=	700

TABLE 14

General Mode of Involvement of the Affected Fingers

Fingers	*Unilateral Involvement* *Solely*	*In the first place*	*Bilateral Involvement*	*Total*	*% Incidence*
1st	8	6	36	50	7·4%
2nd	10	7	69	86	12·8%
3rd	—	6	45	51	7·6%
4th	1	6	22	29	4·3%
5th	38	23	304	365	54·4%
4th + 5th	—	18	5	23	3·4%
Simultaneous involvement	11	—	34	45	6·6%
Mixed involvement	25	—	—	25	3·7%

OBSERVATIONS

The 5th or little finger was by far the most commonly involved (54·5%).

Next the 2nd or index finger (12·8%); then the 1st finger or thumb (7·4%) and the 3rd finger or middle one.

Finally the simultaneous involvement of fingers (6·6%); the 4th or ring finger (4·3%); the combined 4th and 5th (Pure Ulnar Palsy), 3·4%, and the mixed involvement of fingers (3·7%).

TABLE 15

Average Interval between Affected Fingers of both hands

	Total	Average Interval
From Right to Left	207	2·2 years
From Left to Right	166	2·1 years

OBSERVATIONS

When both hands were effected in turn, it was found that the involvement from right to left was more common than the involvement from left to right.

Practically no difference was detected between their respective average interval, the latter being just over 2 years for both.

B. *The Clinical Pattern of Deformity*

(ii) *Deformity of the Foot:*

Comparative Frequency of Deformity of the Foot between Clinical Forms
Comparative Frequency of Deformity of the Foot per Sex
Comparative Frequency of Deformity of the Foot per Age Group
Pattern of Deformity of the Foot:
General Mode of Involvement of the affected Toes
Average Interval between affected toes in both Feet

TABLE 16

Comparative Frequency of Deformity of the Foot between Clinical Forms

	Lepromatous	Tuberculoid	Indeterminate	Borderline
Total No. of examined patients	627	632	186	123
% incidence of the type of the disease	39·4%	40·03%	11·2%	7·7%
Total No. with deformity of the foot	172	215	19	52
% incidence of deformity of the foot	27·4%	34·01%	10·2%	42·2%

OBSERVATIONS

Deformity of the foot was proportionately more frequent among the Borderline cases (42·2% representing 7·7% of the total).

Next among the Tuberculoid group, 34% (40% of the total).

Then among the Lepromatous, 27·4% (39·4% of the total), and finally the Indeterminate, 10·2% (11·2% of the total).

TABLE 17

Comparative Frequency of Deformity of the Foot per Sex

	Total cases examined	Grouping	Sex incidence	No. found with deformity	%Incidence of deformity
Males	1568	953	60·8%	281	30·1%
Females		615	39·2%	177	25·5%

OBSERVATIONS

Among the total of 953 males examined, 281 (30·1%) showed deformity of the foot.

Among the total of 615 females examined, 177 (27·5%).

Men were thus slightly more affected than women.

TABLE 18

Comparative Frequency of Deformity of the Foot per Age Group

	0–5	6–10	11–20	21–30	31–40	41–50	50+
No. of patients with deformity	—	—	91	277	195	98	39
No. of patients with deformity of the foot	—	—	31	199	136	59	33
% deformity of the foot	—	—	35·06%	68·5%	69·7%	60·2%	84·8%

OBSERVATIONS

Deformity of the foot appeared in the 11–20 age group.

It was more common in the 21–30 age group and thereafter steadily declined with age.

Irrespective of the age group concerned, the percentage deformity of the foot was, like in the hand, proportionately quite high.

TABLE 19

Pattern of Deformity of the Foot

	Right	Left	Bilateral	Total	%Incidence
B2. Perforating ulcers	58	58	326	442	42·6%
B3. Claw-toes &/or foot-drop	83	116	203	402	38·7%
B4. Partial absorption	49	62	60	171	16·5%
B5. Gross absorption	9	6	7	22	2·1%

OBSERVATIONS

The commonest type of deformity of the foot was perforating ulcers (42·6%).

Next, claw-toes (including foot-drop), 38·7%.

Then partial absorption of the foot (16·5%) and gross absorption)2·1%).

Bilateral Involvement (Mixed Types)	=	328 (46·8%)
Unilateral „ „ „	=	130 (17·1%)
No Involvement of the Foot	=	242 (34·5%)
Total	=	700

TABLE 20

General Mode of Involvement of the Affected Toes

TOES	*Unilateral Involvement*		*Bilateral Involvement*	*Total*	*% Incidence*
	Solely	*In the first place*			
1st	13	36	152	201	47.8%
2nd	2	12	50	64	15.3%
3rd	1	6	37	44	10.4%
4th	1	5	32	38	9.5%
5th	2	8	40	50	11.9%
Simultaneous Involvement	3		6	9	2%
Mixed Involvement	14		—	14	3.3%

OBSERVATIONS

The 1st or big toe was by far the most commonly involved (47.8%)
Next the 2nd toe (15.2%); then the 5th (11.9%); the 3rd (10.4%); the 4th (9.5%)
Finally, the mixed involvement of toes (3.3%) and their simultaneous involvement (2%)

B. *The Clinical Pattern of Deformity*

(iii) *The Combined Deformity of Hand and Foot*

Of the 700 cases with deformity, 432 (61.7%) had combined deformity of hand and foot, an unusually high incidence which necessitated further investigation
As would be expected, their comparative frequency between clinical forms, per sex and per age group was identical as in the case of the hand and foot alone
The general pattern of the combined deformity and the average interval between hand and foot and foot and hand will only be considered here.

TABLE 21

General Pattern of the Combined Deformity

Involvement	*Frequency*		*Total*	*% Incidence*
	Males	*Females*		
Right hand + right foot	9	4	13	3%
Right hand + left foot	4	2	6	1.3%
Right hand + both feet	10	3	13	3%
Left hand + left foot	4	2	6	1.3%
Left hand + right foot	5	4	9	2.8%
Left hand + both feet	4	9	13	3%
Both hands + right foot	32	16	48	11.1%
Both hands + left foot	42	20	62	14.3%
Both hands + both feet	157	105	262	60.2%

OBSERVATIONS

The commonest pattern of involvement of clinical importance was both hands and both feet (60.2%)
Next both hands and the left foot (14.3%) and both hands and the right foot (11.1%)
Males and females were almost equally affected

TABLE 22

Average Interval between Hands and Feet and Feet and Hands

	Frequency	*Time Interval*
Hands followed by feet	239	3.3 years
Feet followed by hands	193	3.1 years

OBSERVATIONS

In this series of combined deformity, the average interval between hands and feet was 3 years and 4 months; between feet and hands 3 years and 1 month.

The difference between these various average intervals was, therefore, not statistically significant.

V. DISCUSSION

Leprosy is still a curse rather than a disease amidst a complex emotional background intertwined with prejudice, ignorance and fear. There are an estimated 20 million leprosy sufferers throughout the world and some 3 to 5 million among them are the victims of disabilities which are responsible for the physical, mental and social crippling with which the disease is associated.

This study arose from the need of answering 2 cardinal questions which the literature has ignored over the past 50 years: (*i*) the onset of deformity of the extremities in leprosy and (*ii*) the pattern of the deformity within the context of clinical observations. As a result, an analysis of 700 cases of deformity of the extremities was carried out among a selected group of patients.

As Andersen (1963) rightly pointed out, deformities in leprosy have a triple origin, namely motor paralysis, sensory loss and direct damage to the tissues by the leprosy bacillus, and any one clinical deformity may have two or more primary factors interacting together in their secondary manifestations. Deformity from the point of view of motor paralysis and/or direct damage to the tissues by *M.leprae* is our chief concern here, though it must be stated that sensory loss (anaesthesia) is *per se* quite a severe disability particularly in the hand. In fact, Brand (1964) emphasized that in the United Kingdom, and also in America, a person with total loss of sensation in the hand and without any other disabilities is graded for insurance purposes as being 100% disabled for that particular hand.

Onset of Deformity:

In this study, deformity appeared 2 years and 9 months after the start of leprosy among the Tuberculoid cases; an average of $4\frac{1}{2}$ years among the Borderline; an average of 5 years and 3 months among the Indeterminate and an average of 9 years among the Lepromatous. This means that, although deformity is preventable, although one is aware that measures taken in time would logically ward it off, yet one knows now that one has about 3 years of grace in respect of the Tuberculoid cases (excluding, of course, the polyneuritic or 'pure' neural form); an average of $4\frac{1}{2}$ years with regard to the Borderline; over 5 years as far as the Indeterminate cases are concerned and 9 years or more when dealing with the Lepromatous patient. These relative years of grace do, in fact, invite us more than ever to be on the look-out for the early signs of deformity. This is of paramount importance, particularly in a national leprosy control programme, since the deformity rate will go on increasing if proper coverage by means of early diagnosis and treatment does not take these years of grace also into account. If leprosy control is to be ultimately achieved, measures ignoring the relative years of grace in respect of each type of the disease age and sex, must be studied, to save further loss in manpower, and more distress to millions of human beings. Having now such a yardstick of prognosis at hand, this has a direct bearing on the planning and implementation of rehabilitation programmes at both institutional and field levels: since one of every 4 or 5 patients will develop deformity, one is now in a position to forecast the average delay before its onset.

As for the 2 polar types of leprosy, i.e. Tuberculoid and Lepromatous, it is a universally accepted rule that deformity is comparatively an early manifestation in the former, and a fairly late one in the latter. One can now predict how early or how late this will occur. The onset of deformity can be up to three times longer among the Lepromatous than among the Tuberculoid; that deformity seems to be an earlier manifestation among the Borderline than among the Indeterminate patients; that the onset can be delayed as much as twice as long among the Indeterminate than among the Tuberculoid cases.

It now appears that the onset of deformity is related to sex, and that men are earlier affected than women by an average of $2\frac{1}{2}$ years. One reason for this difference may be that men are engaged in a rougher manual work, hence the greater risk of damage through pressure and trauma on the anaesthetic digits.

As for the effect of age, deformity began to appear an average of 2 years and 8 months after the start of the disease in the 11–20 age group; an average of $5\frac{1}{2}$ years in the 21–30; an average of 6 years and 9 months in the 31–40; an average of 7 years and 4 months in the 41–50; an average of 9 years in the 51 onwards.

Yet, it is very likely that the younger patients who are becoming deformed only show their deformity when they get to a later age group. Therefore, the later age group totals include those who are becoming deformed in the earlier age groups. Taking this into account, it seems that, nevertheless, *the younger the age group on the whole, the earlier tends to be the onset of deformity*. On the other hand, the present study shows that deformity practically never appears before the age of 5 and that it is a pretty rare occurrence under the age of 10.

The clinical pattern of Deformity:

In this study, 8.4% of the patients (i.e. 59 out of 700) stated that the disease began and persisted with neural disorders. This is the polyneuritic or 'pure' neural form of leprosy which belongs to the broad Tuberculoid group and which is generally in a minority. However, there is a need to differentiate it from other amyotrophic diseases, for as Freitas and Couceira (1939) pointed out there is sometimes difficulty in distinguishing the 'pure' form of neural leprosy from the muscular atrophy of the Aran-Duschene type. Similar views were expressed by Pessin (1948). Kolb (1946), on the other hand, stressed the importance of differentiating neural leprosy from syringomyelia, a somewhat difficult task in the early stages of both diseases (Pessin, 1948; Darma and Baruch, 1951; Lucas, 1956).

89.2% of the patients with deformity of the extremities had noticed skin signs before neural disorders; 96.9% had a disturbance of sensation before motor or trophic symptons. However, the patients' own impressions may be unreliable particularly in respect of the recognition of the early stages of loss of sensation or the early stages of motor weakness. Bresani-Silva (1956–7) observed that 54.7% of his series started with neural symptoms and in 85% the disturbance of sensation appeared before the motor and trophic ones.

Comparative Frequency of Deformity between Clinical Forms:

It was observed that the deformities either of the hand or of the foot or of both hand and foot in the same individual, were more frequent in the Borderline type, and progressively less so in the Tuberculoid, Lepromatous and Indeterminate types in that order. Proportionately then, the highest incidence of deformity was seen among the Borderline, and the lowest among the Indeterminate cases. However, it must be stated that the frequency of deformity varies from one geographical region to another. Possibly this may be related to the geographical variations in the clinical types of leprosy. More information on a world-wide basis is required on this topic. Gay Prieto (1961) stated that the WHO Leprosy Advisory Team found in Northern Nigeria a deformity rate of 87.8% among the Lepromatous, 34.6% among the Indeterminate and 22.18% among Tuberculoid cases. In the Cameroons, the figures were 81.09%, 43.08% and 26.3% respectively. In both places, the number of Borderline cases were too small to permit any conclusions to be drawn. Yet Hargraves (1963) found that the percentage of disabilities held for both the Lepromatous and the Tuberculoid of his group. Jonquieres and Capurro (1963) found a low incidence of neuro-

logical phenomenon leading to disabilities among the Borderline of their series.

Comparative Frequency of Deformity and Sex:

It was found in the present study that deformity of the hand, foot or both hand and foot in the same individual was seen slightly more often in men than in women. This is in accord with the statement of Gay Prieto (1961) to the effect that, as a rule, leprosy affects men more than women and that there is a concomitantly higher rate of disabilities among males. This was also pointed out by Guinto (1960). although both of these workers did not supply data supporting this view. Hargraves (1963) found a slight sex difference in his group. The findings of the WHO Leprosy Advisory Team in Northern Nigeria, Cameroon, Thailand and Burma pointed to a difference which was statistically not significant.

Comparative Frequency of Deformity in each Age Group:

Deformity of the hand or foot alone or of both hand and foot in the same individual followed a definite pattern: it began to appear in the 11–20 age group, was more common in the 21–30 age group and became progressively less thereafter. These findings do not mean that older leprosy patients have less deformity than younger ones, but that patients contract the disease in their teens or in early adult life. Gay Prieto (1961) stated that the frequency of disabilities increases with age, but cited no statistical evidence in support of his statement. On the other hand, Martinez Dominguez and Bechelli (1963), following the general findings of the WHO Leprosy Advisory Team in Northern Nigeria, Cameroon and Thailand, concluded that the frequency of disabilities increases with old age by reason of the more advanced stage of the disease and the greater proportion of the Lepromatous cases among old people. This is in keeping with the results of the present study which has shown to what an extent disabilities do, in fact, increase with old age as well as decrease with it, on the plea that the majority of Borderline cases do become Lepromatous.

The WHO Leprosy Advisory Team (1965) found in their random sampling surveys that the frequency of disabilities was rather high in children of the 5–14 year age-group, an average of 12.6% in Cameroon, Northern Nigeria, Thailand and Burma. The same team having also found some evidence pointing to the fact that the longer the period prior to treatment, the greater the proportion of disabilities, the overall implications are clear: early treatment may to some extent prevent them, as it remains a sure and cheap way to avoid future socio-economic difficulties and the heavy expenses of rehabilitation. It is also evident that the disability and consequently the deformity rates increase if early diagnosis and early preventive measures are not carried out, hence the need for special attention to the child and young adult populations in countries where leprosy is endemic.

Nature of Deformity:

The sequence of involvement of the digits was the same in the hand as in the foot (in the WHO classification A3, A2, A4, and A5; B3, B2, B4 and B5). Proportionately, claw-hand was almost as common as perforating ulcer. Partial absorption of fingers was more common than partial absorption of toes, actually in a ratio of 3:2. The total absorption of fingers was twice as common as the total absorption of toes. This implies that, in this series, the overall incidence of hand involvement was one and a half times as much as the foot involvement (both representing 96.2% and 65.4% of the total number of cases respectively).

In this study, the author found that it was more common for both limbs to be involved than one alone. The ratio bilateral involvement to unilateral involvement in the hand was 3:1 and in the foot $2\frac{1}{2}$:1.

Our finding that the hands were more frequently involved than the feet agrees with the one of Bresani-Silva (1956–7). This is not surprising in view of their more active functional role. What is alarming, though, is the extent to which the affected hands are neglected in leprosy. Fingers with contraction were usually allowed to develop contracture (from the reversible to the irreversible stage). Bilateral involvement was the common finding. Both of them in fact indicate sad neglect, both of them lend urgency to our problem.

The general pattern of involvement of the affected digits and their sequence of development showed that the 5th or little finger and 1st or big toe are the first to be affected (both representing 54.4% of the affected fingers and 47.8% of the affected toes respectively): in this series, however, the little finger was twice as often and twice as early involved as the big toe. The next digits of clinical importance were the 2nd or index finger and the 2nd toe (both representing 12.8% of the affected fingers and 15.2% of the affected toe respectively). Unfortunately, no data in the literature are available for comparison, but the inference is obvious: in the mass education of population at risk, emphasis should be laid on the involvement of the little and index fingers and the big and second toes as the earliest manifestation of deformity.

The next question, having indentified the earliest manifestation, is 'How quickly does deformity develop from one limb to the corresponding or opposite one?' In this series, an average of 2 years from hand to hand (whether from right to left or left to right); an average of $2\frac{1}{2}$ years from foot to foot and an average of 3 years from hand to foot and foot to hand.

This study shows for the first time, as far as the author can determine, that deformity of one extremity is seldom an isolated occurrence for an indefinite period of time; on the contrary, figures show that deformity of the corresponding extremity or of the opposite one is likely to follow within 2 years from hand to hand; within $2\frac{1}{2}$ years from foot to foot; within 3 years from upper to lower limbs and vice versa. These 2 to 3 years are ample time for preventive treatment. Seldom does such a neglected disease give such a long warning. Seldom is such a clear warning so sadly disregarded.

Combined Deformity in the same Individual:

As with deformity of upper and lower extremities, the incidence of involvement of both extremities followed similar pattern in respect of the other clinical features studied. No figures can convey the suffering expressed by the finding in this study that 58.% of the patients had combined deformity of the extremities, and 60.2% of them had involvement of both hands and both feet. These figures are a measure of the extent to which leprosy had advanced in these patients at the time this study was made. They are an expression in figures of measureless human suffering. The tragedy is that those results are preventable, and once more, there is a period of approximately 3 years during which effective measures of treatment could have been applied. Three years of neglect resulted in deformity of both hands and both feet in 3 out of every five patients.

The earlier part of this study has shown that for a fairly long period the striking mark of leprosy is the involvement of one digit, the little finger or the big toe. The conclusion seems inescapable: the task of leprology is to seek these early cases.

What other disease of this importance gives the physician such a long time to apply these remedies? The essential tragedy of leprosy is the tragedy of opportunities missed. This study attempts to define the problem, and establish the factual basis upon which a rational therapeutic programme can be planned.

VI SUMMARY

Time of Onset of Deformity of the Extremities in Leprosy:

1. A sudy of 700 cases of deformity of the extremities was carried out among a selected group of patients in Northern Burma.

2. Deformity of the extremities appeared an average of 2 years and 9 months after the start of the disease in the Tuberculoid; an average of $4\frac{1}{2}$ years in the Borderline; an average of 5 years and 3 months in the Indeterminate and an average of 9 years in Lepromatous leprosy. Thus in this study, the onset of deformity was 3 times as long in the Lepromatous as in the Tuberculoid cases. At any rate, even when the march of disease was most rapid there was a latent interval of nearly 3 years during which treatment could have prevented deformity.

3. Men were affected at an earlier stage than women, by an average of some $2\frac{1}{2}$ years. This was related to the fact that men are engaged in a rougher manual work at an earlier age, with thus greater risks of injury and secondary infection of the anaesthetic digits.

4. Deformity of the extremities was not seen under the age of 5 and was a rare occurrence

before the age of 10. The younger the age group, the earlier seems the onset of deformity. Whether affecting hands or feet alone or in combination, deformity of the extremities was first seen between the ages of 11 and 20, and most often appeared between 21 and 30. Thereafter its incidence steadily declined with age.

5. As one in four of five patients with leprosy is likely to develop deformity, it is possible, on the strength of the present study, to predict the average delay to be expected with each clinical type, sex and different age groups. A yardstick of prognosis emerges which has its place in diagnosis, treatment and the planning of rehabilitation programmes, at both institutional and field levels.

Clinical pattern of Deformity of the Extremities in Leprosy:

1. *Frequency of Deformity between clinical Forms:* It was found that deformity of the hands, feet or both followed an identical order of frequency: Borderline, Tuberculoid, Lepromatous and Indeterminate. Thus deformity of the extremities was most common in the Borderline and least common in the Indeterminate cases.

2. *Frequency of Deformity per Sex:* Men were slightly more often affected than women.

3. *Frequency of Deformity per age Group:* As per paragraph 4 above.

4. *Predominant Types of Deformity:* Contracture, mobile claw-hand, partial absorption of fingers and total absorption of fingers in that order. In the foot: perforating ulcers, claw-toes + foot-drop, partial absorption of the toes and total absorption of the toes respectively. In this study, the 2 predominant types of deformity were contracture and perforating ulcers.

5. *Nature of Involvement:* Bilateral involvement was much more common than unilateral involvement in a ratio of 3:1 in the hands and $2\frac{1}{2}$:1 in the feet.

6. *Incidence of Involvement:* Hands were $1\frac{1}{2}$ times more involved than the feet.

7. *Pattern of Affected Digits:* The first digits to be affected were by far the 5th or little finger and the 1st or big toe; next of clinical importance were the 2nd or index fingers and the 2nd toe.

8. *Time Interval between Affected Digits:* An average of 2 years from hand to hand (whether from right to left or left to right); of $2\frac{1}{2}$ years from foot to foot and of 3 years from hand to foot and foot to hand.

VII CONCLUSIONS

In this study, two fundamental aspects of the natural history of the onset of deformity of the extremities in leprosy have been investigated, namely the latent interval between the start of the disease and the appearance of deformity and its pattern of development. Such data have not been collected before as far as the review of the literature can determin. They offer a means of predicting the course of the disease, and would appear to be fundamental to any large-scale planning of anti-leprosy campaigns as well as to institutional work.

Now that the average delay before the onset of deformity can be stated for the various clinical forms, the sex and age of the patient, and now that the march of events from digit to digit and from limb to limb is clear, the necessity for early diagnosis, early treatment and early preventive measures comes to have more weight and more than a touch of urgency, with the aim of warding off deformity is now seen to be within the limits of possibilities, with special attention to the child and young adult populations. What other similar disease offers the physician three years of grace?

This concept should encourage those engaged in treatment. It should enrich with understanding and faith those carrying out preventive measures. These data, once more, emphasize the importance of early diagnosis and early recognition of deformity. Perhaps in no other disease are the rewards of early diagnosis so rich. Perhaps in no other disease are the penalties for neglect so terrible.

Deformity is not an inescapable feature of leprosy work. It is preventable by proper measures. All those experienced in leprology are agreed that most of the disabilities in leprosy can be prevented and that those which cannot be prevented can be corrected by reconstructive surgery. The philosophical acceptance of the inevitable is an attitude which can hardly be tolerated in this day and age, when the present study shows that there is plenty of time for therapy to be carried out.

How much is being done for the prevention of deformity, how much for rehabilitation? The author's experience in West Africa, Burma and India, has been that, whereas commendable efforts are being made to train more leprosy physiotherapists, yet there are no wide-scale programmes to prepare them nor is there a sufficient number of surgeons to cope with the demands of the situation. Hundreds of thousands of leprosy patients are the victims of disabilities, thousands more are becoming deformed, and our study has shown that these are mostly young people who could have been saved from them and still be spared unnecessary suffering.

As the members of the Panel on Physical Medicine and Rehabilitation at the International Congress of Leprology (1963) have pointed out, the greatest barrier to rehabilitation from leprosy has been difficulty of the cure of the disease. This is overcome. The second barrier is public ignorance, prejudice and fear. This will, unfortunately, be the case for years. The third great barrier is the prejudice of physical disabilities which is a matter of grave concern as it goes on after the disease is cured, thus making a return to normal life difficult even when public prejudice is no more. This is preventable.

Reconstructive surgery requires both specially trained personnel as well as special equipment, neither of which fulfills the needs of rehabilitation in countries where leprosy is endemic. The heavy financial implications involved would take years to reach all those suffering from deformity, with the result that money and manpower would be better spent in prevention.

Prevention is not only easier than correction, but is within the reach of every leprologist and para-medical worker with the minimum of training and inexpensive equipment. Time must, therefore, be found to guide and help those patients in whom they recognize the early signs of deformity. Sufficient time can be generally found in both institutions and field work for this purpose.

Yet the irony is that rehabilitation is still thought as something which starts after the disease is cured. This tragic error explains to a large extent the present bleak picture of disabilities in the world. Rehabilitation not only starts as soon as the disease is diagnosed, but persists throughout treatment and after it if necessary; if not, the intervening changes in the patient himself and prejudice among his relatives and friends may evolve into a stage when they become irreversible.

What of the future? The stigma of leprosy will fade when public and patients alike accept the disease as any other disease, but unlike many is eminently curable. It lies when full information will be obtained on the frequency and degree of disabilities world-wide so as to have a true picture of the socio-economic implications of leprosy, hence a better idea will be obtained as to the most effective planning of preventive and corrective measures.

The future lies in the cultivation *in vitro* of *M.leprae*, in the clearer understanding of the pathogenesis of the disease, in research and perhaps in the avoidance of the word 'leper', and in substituting Hansen's disease for Microbacterial Neuropathic Dermatosis (M.N.D.) proposed by Ross Innes (1963) or for Hansenosis suggested by Goldman (1963) instead of leprosy, though, as Skinsnes (1964) rightly remarked, it is questionable whether such a laudable purpose can be achieved in view of the pattern of society's deeply-rooted reactions and hence the wisdom of leaving the word leprosy as such so as to allow a gradual decline of the misconceptions and musunderstandings attached to the disease on its time-honoured scale.

The future lies in a wider study of leprosy with increasing contributions from the allied branches of medicine, for as Loginov (1964) pointed out, there is still a great deal of obscurity in the theory and practice of leprology, and that the majority of the unanswered questions are closely bound up with the present-day problems of medicine and biology.

The future finally lies in the leprosy rehabilitation services when they will be fully integrated with other rehabilitation programmes in general hospitals and other institutions, hence the vital importance to pave the way, however long, for it in countries where the fight against leprosy is to be guided still along proper lines.

The attitude of society and of other medical and scientific disciplines towards the leprosy patient depends not only on education, but upon the belief in the individual worth of every human being. A lot is achieved when curing a

leprosy patient, but by preventing deformity we do prevent the loss of a man's self-respect, of his dignity. 'Homo sum, humani nihil a me alienum puto?' This study attempts to show that this prevention is well within our reach.

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REFERENCES

ANDERSEN, J. G. (1961) *Lep. Rev.* **32**, 16–27.
— (1962) *J. Ch. Med. Ass. Ind.* **36**, 1–6.
— (1962) *Lep. Ind.* **34**, 116–121.
ARGENTA, G. (1961) Ed. Calia di Napoli. Abstracts.
BANEDOCH, A. G. and BYRON, F. R. (1932) *Med. Trop.* & *Hyg.* **26**, 253–258.
BANG, T. V. and DIEP, N. D. (1958) Symposium paper, Int. Cong. Lep. Tokyo.
BARNETSON, J. (1951) *Int. J. Lep.* **19**, 297–307.
— (1950) *Acta. Rad.* **34**, 35–64.
— (1950) *Trans. Roy. Soc. Trop. Med.* & *Hyg.* **43**, 535—538.
— (1950) *Idem.* 539–544.
BASSET, A. and SCHNEIDER, G. (1950) *Bul. Soc. Fr. Derm.* & *Syph.* **57**, 299–302.
BASU, S. P. et al (1961) *The Lic. Ambala.* **10**, 369—378.
BRAND, P. W. (1958) *Leprosy in Theory and Practice*, Chapter XXI.
— (1958) *Symposium paper, Int. Cong. Lep.* Tokyo.
— (1959) *Int. J. Lep.* **27**, 1–7.
— (1963) *Rehab. Lit.* **21**, 238–245.
— (1964) Personal communication.
BRESANI-SILVA, F. (1956) I. Rev. Per. de Salud Pub. *Lima.* **5**, 79–204.
— (1956) II. *Idem.* **5**, 229–340.
— (1956) III. *Idem.* **5**, 379–501.
— (1957) IV. *Idem.* **6**, 1–116.
CARAYON, A. and HUET, R. (1958) *Trop. Dis. Bul.* **1**, 54–55.
CASACCI, A. (1950) *Clin. Orth.* **2**, 37–52.
CHARDOME, J. and LECHAT, M. (1955) *Ann. Soc. Belge Med. Trop.* **35**, 267—278.
— (1958) Symposium paper, Int. Cong. Lep. Tokyo.
CHATTERJEE, S. N. (1955) *Int. J. Lep.* **26**, 1–17.
— (1964) *Idem.* **4**, 384–409.
CHAUSSINAND, R. (1950) *L Lèpre.* Exp. Sc. Fr. Paris.
CHERLINZONI, C. and PIRATSU, E. (1952) *Chir. Organi Mov.* **37**, 116–133.
COONEY, J. P. and CROSBY, E. H. (1944) *Rad.* **42**, 9–14.
CUERVO, J. M. et al (1944) *Rev. Lep. Derm. y Sifil.* Cuba. **1**, 148.
DARMAR, H. and BARUTH, S. (1951) *Contr. Clin. Sc.* Istanbul. **1**, 478-97.
DA VEIGA, S. (1947) *Ann. Inst. Med. Trop.* Lisbon, **4**, 149–60.
DHARMENDRA, C. (1960) *Notes on Lep.* Mins. Hlth. Delhi. 57–65.
DHOPLE, A. M. and MAGAR, N. G. (1962) *Lep. Ind.* **4**, 299–305.
DOULL, J. A. (1959) *Int. J. Lep.* **27**, 202–215.
— (1963) Presidential address, CIBA Fd. Symp. Path. Lep., London
DUBOIS, A. (1951) *Ann. Soc. Belge Med. Trop.* **31**, 525—529.
— and RADERMECKER, M. A. (1953) Int. J. Lep. **21**, 204–217.
EDWARDS, R. H. T. (1964) *Lep. Rev.* **35**, 3–13.
ERICKSON, P. T. and JOHANSEN, F. A. (1948) *Int. J. Lep.* **16**, 147–156.
— and MAYORAL, A. (1950) *Rad.* **54**, 357–364.
ESQUERRA-GOMEZ, G. and ACOSTA, E. (1948) *Rad.* **50**, 619–638.
FAGET, G. H. and MAYORAL, A. (1944) *Rad.* **50**, 619—638.
FITE, G. L. (1941) *Int. J. Lep.* **9**, 193—202.
and VIRGILIO, S. (1942) *Rev. Bras. Lep.* **10**, 273–302.
FREITAS, J. O. and COUCEIRO, A. (1939) *Rev. Bras. Lep.* **7**, 97–120.
GAY PRIETO, J. (1958) *Int. J. Lep.* **26**, 441—446.
— (1961) WHO Series, WPR/LEP/20, 1–10.
GLYN GRIFFITHS (1964) Personal communication.
GOLDMAN, L. (1963) *Lep. Rev.* **35**, 51–52.
GUINTO, R. (1960) Proferred paper at the Scientific Meeting on Rehab. *in Lep.*, Vellore, Ind.
HARGRAVES, J. (1963) *Med. J. Australia* **2**, 225–227.
IMAEDA, T. (1968) I & II. *La Lepro*, **27**, 1–25.
JONQUIERES, E. D. L. and CAPURRO, E. T. (1963) *Leprologia*, **8**, 48–49.
KADRINKA, S. and MERDJO, A. (1938) *Rad.* **7**, 269–286.
KARASEFF, J. (1936) *J. Rad.* & *Electrol.* **20**, 373–382.
KATSUMI, H. (1955) I & II. *La Lepro*, **24**, 78–96.
K'UNG C'HING-TEH et al (1959) *Ch. Med. J.* **79**, 130–137.
LANGUILLON, J. and BOISSAN, R. (1959) *Med. Trop.* **19**, 558–567.
— (1960) *Idem.* **20**, 482–492.
— and CARAYON, A. (1961) *Sup. Presse Med.* **69**, 1–4.
LECHAT, M. (1960) *Etudes des Mutilations Lépreuses.* 17–177.
— (1962) *Int. J. Lep.* **30**, 125–137.
— and CHARDOME, J. (1957P *Ann. Soc. Belge Mec. Trop.* **37**, 907—918.
— et al (1961) *Idem*, **41**, 535—566.
— et al (1961) *J. Chir. Dis. St-Louis*, **13**, 221–227.
LENNOX, W. M. (1964) *Lep. Rev.* **35**, 245–249.
LINDUI and ROSS (1957) cited by Lechat (1960).
LOGINOV, V. K. (1964) *Lep. Rev.* **35**, 14–20.
LUCAS, C. J. (1956) *B.M.J.* **2**, 214.
MALLAC, M. J. (1960) *Lep. Rev.* **31**, 12–18.
— (1962) WHO/SEARO/LEP/II, 1–14.
— (1963) WHO/SEARO/LEP/17, 1–9.
— (1964) WHO/SEARO/LEP/19, 1–16.
MARTINEZ-DOMINGUEZ, V. M. and BECHELLI, L. M. (1963) Proferred paper at the Int. Cong. Lep. Rio.
MATHUR, J. S. and SAXENA, K. N. (1965) *Lep. Rev.* **36**, 77–81.
MILLE, R. and PAPA, F. (1961) *Arch. past. Martinique*, **14**, 24–29.
MINATO, J. (1958) *Int. J. Lep.* **26**, 442–448.

MONTESTRUC, E. (1960) *Arch. Past. Martinique*, **13**, 90–98.
— and HYDROMINUS, J. C. (1961) *Bul. Soc. Path. Exot.* **54**, 988–995.
MUELLING, R. J. et al (1960) *Int. J. Lep.* **28**, 144–154.
MURDOCH, J. R. and HUTTER, H. J. (1932) *Am. J. Roentg.* **28**, 589–598.
MUT MUT, T. (1953) *Int. J. Lep.* **21**, 615–623.
NÉGRE, A. and FONTAN, R. (1957) *Trop. Med. Bul.* **54**, 579–586.
NISHIKAWA, T. (1934) *Int. J. Lep.* **2**, 487–492.
NISHIURA, M. (1955) *La Lepro*, **24**, 204–215.
OBERDORFFER, M. J. and COLLIER, D. R. (1940) *Am. J. Roentg.* **44**, 386–395.
PATERSON, D. E. (1955) *J. Fac. Rad.* **7**, 35–36.
— (1956) *Int. J. Lep.* **10**, 90–97.
— (1958) *Leprosy in Theory and Practice*. Chapter XX.
— (1961) *Int. J. Lep.* **29**, 397–422.
PESSIN, J. (1948) Arch. *Neur. & Pschy.* **60**, 288–296.
RADERMECKER, M. A. (1952) *Acta. Neur. & Pschy. Belge*; **52**, 204–217.
Report of the Panel on Epidemiology & Control (1963) *Int. Cong. Lep. Lep. Rev.* **2**, 17–33.
— Physical Med. & Rehab. (1963) *Idem*. 43–51.
— Ed. & Soc. Aspects (1963) *Idem*. 52–72.
ROSS, SR. H. (1955) *Len. Wood Mem.* **6**, 21–23.
— (1955) *Idem*. 26–47.
SKINSNES, O. K. (1964) I. *Lep. Rev.* **35**, 21–35.
— (1965) III. *Idem*. 175–181.
SUSMAN, I. A. (1963) *Lep. Rev.* **2**, 83–94.
TORSUYEVA, N. N. (1962) *Coll. Sc. Paper on Lep. & Derm.* **16**, 86–89.
VENKATASUBRAMANIAN (1941) cited by Ross (1955).
WAYSON, N. E. and BADGER, L. F. (1929) *Pub. Hlth. Bul. USA.* **189**, 16–42.
W.H.O. Tech. Rep. Series (1960) International Work in Leprosy.
— (1960) Scientific Meeting on Rehabilitation in Leprosy.
— (1960) Disabilities due to Leprosy: Extent of the Problem.
— (1965) A Review of the Jointly Assisted Leprosy Control Projects, UNICEF/WHO Joint Committee on Health.
WOOLEY, J. G. (1931) *Pub. Health Rep. USA.* **46**, 641–658.
— and ROSS, SR. H. (1932) *Idem*. **47**, 380–389.
WOZONIG, H. (1958) *Trop. Dis. Bul.* **54**, 699–706.

Lep. Rev. (1966) **37**, 2, 93/97

Neural Involvement in Leprosy Treatment with Intra-Neural Injections of Prednisolone

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Decompression of the thickened nerves has been known to relieve symptoms distal to the thickening. Surgical splitting of the neural sheath and transposing of the nerve as well as injections of corticosteroids have been used mainly for relieving pain. The writer has made some trials with intra-neural injections of Prednisolone (Benzon) as an auxiliary therapy in patients receiving oral anti-leprosy drugs. Most of these cases of neural involvement had shown a poor response to the sulphones. The writer wishes to present the results which he has obtained as they may be of interest to leprologists.

The trials which concerned a study of trophic changes of the skin, its appendages and muscles, could be carried out only for a short period, because the stock of Prednisolone was limited and could not be replenished.

Identification of the involved nerve(s):

This is fairly easy where the nerve is large, thickened and not embedded in a lot of subcutaneous fat. Pressure on the thickened nerve usually causes a sensation in the terminal area of distribution, thus helping in locating the nerve.

Selection of the correct site of injection is essential for obtaining maximal results and the site is determined according to the area of nervous disturbance; for instance if only the little finger is involved the injection should be given in the ulnar nerve; if all the fingers are involved injection should be in the brachial nerve. If the whole superior extremity including the scapular region is involved the site of injection would be the brachial plexus. The site of (presumably) maximal block in the case of easily palpable nerves can be determined by pressing along the nerve from the proximal towards the distal end and noting the severity of the sensation felt in the area of distribution. Pressure distal to the block causes markedly severe sensation as compared to the point proximal to the block.

When the thickening of the nerve is extensive (whether consecutive or of a beaded type) the most proximal thickening which still produces sensation on pressure is preferred by the writer. Without this sensation to guide the correct positioning of the needle one cannot be sure being in the sheath (see further under technique).

After injecting into the selected thickened segment effort is made to squeeze the drug along the course of the nerve. This can be assisted by positioning the limb to take advantage of gravity.

Technique of injections:

The thick nerve can easily be fixed either transversely or longitudinally between the thumb and forefinger. When the nerve is only slightly thickened or/and the subcutaneous fatty layer is substantial, the writer has the following technique:

After identifying the nerve it is pushed with the index and middle finger till fully stretched. When thus anchored the hypodermic needle is passed along the tip of the index finger into the nerve longitudinally. Confirmation of the correct insertion of the needle in the sheath is provided by a tingling sensation in the distal nerve ending on insertion or wiggling of the needle. This wiggling also moves the nerve and this can be felt by the finger. A much stronger and painful sensation is produced when the Prednisolone is actually injected.

Sometimes, however, there is complete absence of these sensory guides and in such cases the whole procedure is based on intelligent

guesswork. Even in such cases after a few administrations clinical improvement can be observed (vide case A923/65 and Si) and even the tingling (sensory) phenomenon is restored. Gradually the tingling can be induced even by manipulating the nerve more and more proximally (vide case Ta).

Clinical improvements can be evaluated by comparison with the previous condition and/or the condition on the other side. For the skin and its appendages the points observed are:

— return of sensation (to cottonwool, pinprick, pulling out of hair).
— reduction of dryness and scaling; return of normal skin lustre, improvement of sweat and sebum production.
— arrest of loss of hair; resistance to epilation; regrowth of hair.
— normalization of nails.

For the muscles the points are:
— restoration of motor function.
— progressive increase in power.
— anatomical recovery of atrophy.

The *patients* were selected from two sources:

1. from the writer's private practice.
2. from the male out-patients of the National Institute of Health. The records exist of eight patients with functional involvement of the little finger (ulnar nerve) in whom intraneural Prednisolone injections were given for 1 to 3 weeks in a dosage of 5 to 10 mg. with good results in the sweat and return of sensation and in making the skin more lustrous, and improvement in the power of the interossei. The nerve also became thinner and softer:

1. B666/63 37y T-type of Leprosy of 5 years duration. 2 years unsuccessfully treated with sulphones. Very irregularly injected. Increase in pulling power little finger from 1900 Gr.* to 4100 Gr. in 29 weeks.

2. D1498/64 38y Pure neural type of Leprosy of 3½ year duration.
In Institute for ½ year unsuccessfully treated with sulphone (?previous therapy). After fairly regular injections increase of pulling power of little finger from 500 to 1700 Gr. in 22nd week.

3. D1190/64 28y T-type, later borderline type of leprosy of 3 years duration.
For 10 months in Institute treated with sulphones with no improvement (?previous treatment). Fairly regular injections increased pulling power of little finger from 1850 to 4900 Gr. in 21st week.

4. N322/65 28y T-type of leprosy of 8 months duration. After 1 month sulphone therapy in Institute, fairly regular injections given. Increase pulling power of little finger from 700 Gr. to 1700 Gr. in 21st week.

5. P974/63 19y L-type of leprosy of 8 years duration. 2 years' sulphone treatment in Institute gave no improvement. Pulling power of little finger improved from 1650 to 4500 Gr. in 22nd week after fairly regular injections.

6. S1130/62 40y T-type of leprosy of 6 years duration. 7 months' sulphone therapy in Institute (?previous therapy). Fairly regular injections increased pulling power little finger from 900 to 2300 g. in 22nd week.

7. S1480 24y T-type of leprosy of 1½ years duration. No improvement at all after 1½ years' sulphone therapy. After fairly regular injections pulling power of little finger increased from 1000 to 3300 g. in 27th week.
One month after discontinuation of injection pulling power further increased to 3500 Gr. equal to pulling power of the other normal (?), little finger.

8. S756/65 24y T-type of leprosy in reaction. Duration 1 year. One month after sulphone therapy in Institute irregularly injected. Increase in pulling power of little finger from 3050 to 4000 g. in 20th week.

SPECIAL PATIENTS

A. 923/65. male, 32 years, first examined 19/5/65.
Complaints: difficulty in movement of left leg; foot-drop, left, 4 months duration.
Previously unsuccessfully treated with B complex and thiamine by a neurologist.
Clinical findings left leg:
— atrophy of calf.
— skin relatively dry, scaly; touch and pain sensations lost; epilation of the sparse hair easy and painless.
— peroneal nerve thickened and insensitive.
Diagnosis: pure neural form of leprosy.
Therapy: patient placed on routine sulphone treatment.
8/6 no improvement noted.
7·5 mg. Prednisolone (Benzon) injected into the peroneal nerve.
15/6 anaesthesia now changed to paraesthesia specially on dorsum foot; movement of toes im-

*Throughout the paper Gr stands for grammes.

proved. 7.5 mg. Prednisolone injected into peroneal nerve; the injection this time caused a sensation in the calf muscles.

22/6 pressure on peroneal nerve now c used sensation in calf muscles; epilation painful. 7.5 mg. Prednisolone I.N. given.

6/7 epilation difficult and painful; touch (cotton wool) and pinprick sensation returned. Sweating seen on the skin. Pressure on peroneal nerve and I.N. injection of 7.5 mg. Prednisolone now caused tingling in toes also.

27/7 further improvement noted. Skin now lustrous. Still unable to lift foot. 7.5 mg. Prednisolone I.N.

5/10 improvement maintained, footdrop persistent.

Conclusion:

Rapid improvement of the trophic changes seen after Prednisolone injections I.N.

D. female, 31 years, first examined July 1963.

Complaints:

1958: noticed fatigue right forearm and tremor of fingers after work. Noticed a discoloured spot on front right thigh. Unable to write in normal position of hand and forearm; could only write standing with forearm awkwardly pronated.

1961: swinging of right arm while walking painful. Unsuccessful electrotherapy by neurologist.

1963: Leprosy type T diagnosed.

Sulphones not tolerated (drug fever, giant urticaria). Put on Ciba 1906. June 1964 Additional Vadrine therapy started.

Jan. 1965: condition unchanged.

12.5 mg. Prednisolone I.N. into ulnar nerve started at weekly intervals. Decrease of tremor and ability to write in normal position gradually improved.

Feb.–Mar. 150 U Hyaluronidase (Benzon) added to 7.5 mg. Prednisolone I.N. injection. Faster improvement leading to return of normal functions. Ulnar nerve also returned to normal size. End of March I.N. injection stopped.

7/5 complaint of pain in right arm and scapular region. Put on weekly I.N. injections of 10 mg. Prednisolone + 25 U Hyaluronidase subclavicularly into the brachial plexus.

28/5 Scapular pain disappeared. I.N. injections now given in the brachial nerve, 5 cm. below the axilla; repeated weekly until 25 June, when patient left town.

5/6 Complaints of 'cramps' in right leg when walking. Femoral nerve found thickened and tender in the femoral triangle 5 mg. Prednisolone +25 U Hyaluronidase I.N. in femoral nerve; after three weeks 'cramps' disappeared, nerve restored to normal.

17/7 Patient re-examined: both right arm and right leg. N.A.D. 10 mg. Prednisolone+25 U Hyaluronidase I.N. into femoral and ulnar nerve.

2/8 Status quo ante; same injections given.

5/9 During past 5 weeks condition deteriorated. Complaints of 'cramps' in legs after walking long distance and difficulty in rising from sitting/squatting position. Heavy feeling in right forearm; difficulty in writing. Ulnar nerve found to be markedly thickened. Femoral and peroneal nerve moderately thickened. The sciatic nerve could not be palpated satisfactorily. A.P.R. examination was done in order to feel the roots of the sciatic nerve. A considerably thickened nerve was felt at 6–9 o'clock position and pressure on this caused a painful tingling in the toes. Left side N.A.D. As the patient was not expected to come back for quite some time the following injections were given:

ulnar nerve: 10 mg. Prednisolone+50 U Hyaluronidase. Peroneal and femoral nerve each: 7.5 mg. Prednisolone+25 U Hyaluronidase.

4/10 Patient had no complaints, all functions normal, all affected nerves felt thinner and softer. Ulnar and femoral nerves injected as on 5/9. R. sciatic nerve injected just below gluteal fold with 10 mg. Prednisolone+25 U Hyaluronidase.

14/11 Marked improvement on all complaints. All thickened nerves and roots of sciatic nerves thinner and softer.

Remark: The exacerbation noted on 5/9 could perhaps be regarded as a 'rebound phenomenon'.

Ta. male, 38 years, first examined 19/6/65.

History: Jan. 1960 after driving an LHD jeep in rain and cold, developed facial palsy left side with ectropion and drooping corner of mouth. Later an infiltrated red plaque developed around left eye. Diagnosed leprosy T-type with neural involvement. May 1961 placed on Sulphone-chaulmogra (Lab. Roussel) injections.

In 1959 was treated for TB lungs with streptomycin and I.N.H. First examined by writer 19/6/65.

Clinical findings:

— no plaque seen

— ectropion, inability to close eyelids leaving lower 1/3 cornea exposed, lacrimal overflow and some atrophy of orbital and cheek muscles.

— drooping corner mouth.

— thickened infra-orbital branch felt, no sensation on pressure.

— anaesthetic area lateral corner left eye.

Therapy: Sulphone-chaulmogra continued and 10 mg. Prednisolone I.N. given in the thickened nerve by feel alone. After three days marked improvement (22/6).

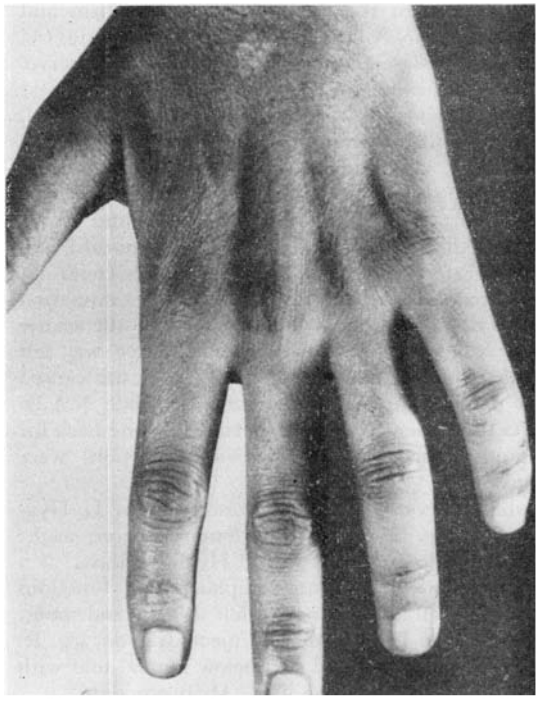


TABLE CASE 7, S 1480/64 4/V/65

Note atrophy of interossei muscles (pulling power of little finger: 1000 Gr.)

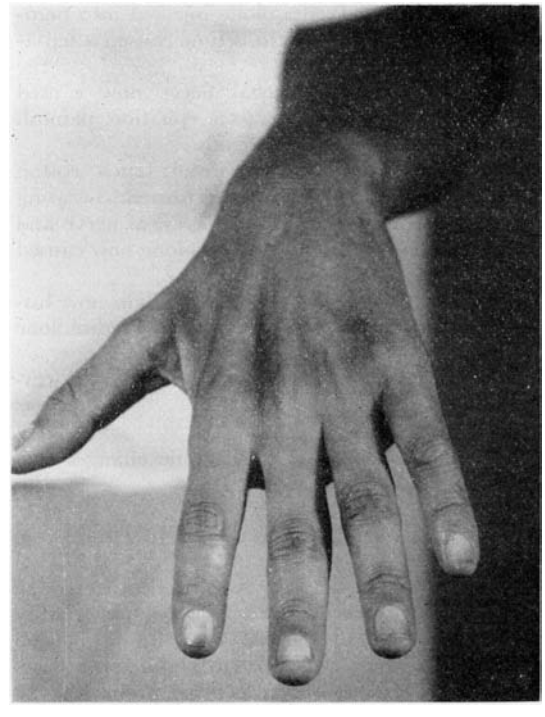
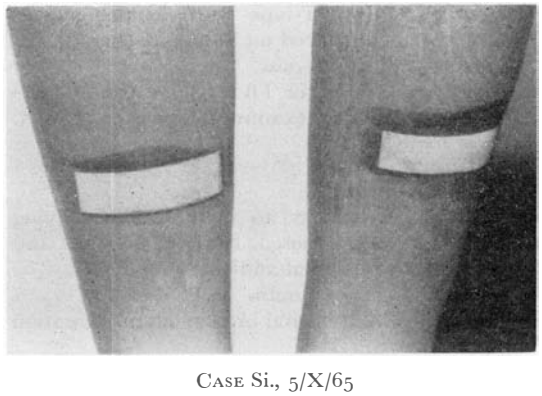


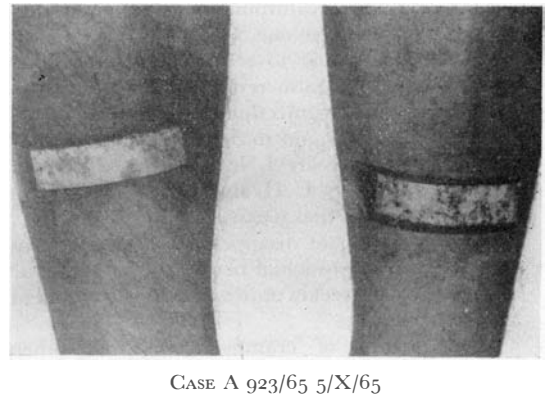
TABLE CASE 7, S 1480/64 5/X/65

Note improvement of ulnar interossei muscles (pulling power of little finger: 3000 Gr.)



CASE Si., 5/X/65

Skin of injected leg more shiny and less scaly; yet sweat-test still negative as other leg.



CASE A 923/65 5/X/65

Rapid improvement of the skin of injected leg:
now lustrous
sweat-test positive

— ability to close eyelid, now only the lower rim of cornea exposed; ectropion reduced; spill-over of tears reduced.
— sensation anaesthetic area returned.
— 7.5 mg. Prednisolone I.N.
29/6 Further reduction in lagophthalmos and other trophic disturbances; drooping of corner mouth reduced. Injection of 7.5 mg. Prednisolone into nerve produced tingling corners eye and mouth.
6/7 Further improvement of above; skin left face more lustrous, 7.5. mg. Prednisolone I.N.
7.5 mg. Prednisolone injection continued at weekly intervals. The return of terminal tingling on insertion of needle into nerve enabled the writer to give the injections into more and more proximal sites until on 20/7 the injection site was 2 cm. in front of ear.
24/8 The injection could be given into the facial nerve between the styloid process and the mandibular angle. Patient himself reported that he no longer needed to protect the eye against a breeze. Can now whistle.
7 & 14/9 injections as 24/8. Progressive improvement.
21/9 Same dosage, given I.N. in foramen ovale, produced big shock on the left face.
Patient failed to turn up after 21/9.

Si. female, 17 years, first examined 31/5/65.
Complaints: foot-drop right side plus red, swollen toes.
Clinical findings:
Infiltrated maculae on face & trunk+trophic changes on skin extremities.
Patient had been on Sulphones for 2½ years without results. This therapy was continued and 7.5 mg. Prednisolone was injected weekly into the vicinity of the peroneal nerve behind the head of the fibula (the nerve itself could not be located with any certainty) I.N. therapy was started on 3/6.
Patient reported improvement soon after injection and her walk continued to improve.
After 2 weeks (17/6) clinical improvement as follows:
— skin less scaly
— toes less red and less swollen
— dragging of the right foot when walking reduced
28/8 skin condition further improved, pull on hair painful.
8/9 Peroneal nerve still could not be identified. Injections contined on 21/9, 5/10 and 12/10: foot drop persisting, Peroneal nerve still could not be identified.

PRELIMINARY CONCLUSIONS

1. Trophic disturbance of the skin and its appendages are favourably influenced even in patients which have failed to respond to the sulphones.
2. The duration of the acute phase (red bluish painful swelling of the fingers/toes) can be shortened.
3. As a result of the shortening of the acute phase loss of digits may be prevented.
4. Muscular power of atrophic muscles can be improved.
5. Lagophthalmos eventually leading to keratitis and blindness can be successfully treated.
6. Sudden and complete withdrawal seems to carry a risk of a rebound.
7. Additional Hyaluronidase appears to be of some benefit.
8. Sebaceous glands seem to restore their function earlier than sweat glands (see picture) as in the Hyfrecator—sparking treatment x).

SUMMARY

Twelve patients with neural involvement were given intra-neural injections of Prednisolone and the results were favourable. It is suggested that this form of therapy is worth being tried out on a large scale.

REFERENCE

* TIO (Tiong Hoo): *Leprosy Review*, 1966, *37*, **1**, pp. 57–60.

ACKNOWLEGEMENTS

I wish to thank Dr R. Wasito, Director of National Institute of Health, Surabaya, for his permission to use the facilities of the Institute. I am also grateful for the kind cooperation of Professor R. Goenawan and Dr R. Sardi. I feel greatly indebted to Dr V. S. Paranjpe for his valuable comments and assistance.

Lep. Rev. (1966) **37**, 2, 99/102

Physiotherapy and Foot Drop Corrections

W. M. LENNOX, F.R.C.S. (Eng.)

Leprosy neuritis affecting the lateral popliteal nerve is the commonest cause of foot drop in tropical countries. Tibialis posterior transfer has proved to be a very satisfactory operation for this condidion, and many of these operations are performed every year. Good results depend upon many factors, but one cause of failure is improper physiotherapy. Since the overall responsibility for cases selected for operation rests with the surgeon, it is intended in this paper to review some facets of physiotherapy for those who may wish to carry out tibialis posterior transfer, and who will be called upon to prescribe physiotherapy afterwards. Surgical technique will not be discussed, but may be reviewed from the references given at the end.

Principles of Physiotherapy for Tendon Transfers

(1) Pre-operatively - Isolation exercises for the muscle to be transferred.

(2) During plaster immobilization: General limb exercises, but not including movements in which the transferred muscle normally participates.

(3) After removal of plaster:
- (a) Unopposed contraction exercises.
- (b) Opposed contraction.
- (c) Protected function.
- (d) Unprotected function.

The principles of physiotherapy in tendon transfer surgery are summarised in the table. In hand surgery, advantage can be taken of muscle synergism to simplify re-education, but after transfer, the basically pro-gravity tibialis posterior has to be educated to an antagonistic function. This is more difficult to achieve than is re-education of a muscle transfered from one function in a synergistic complex to another. However, with the tibialis anterior paralysed, voluntary inversion of the foot is due entirely to tibialis posterior, and this relative isolation is helpful to the therapist. Even so, re-education of this muscle requires close application on the part of all concerned, more so than when dealing with tendon transfers about the hand. As re-education takes place in the brain, young and intelligent patients educate better and more quickly than the elderly and unintelligent.

The management is summarised in the figure. The aims of treatment are:

(a) to teach the patient to isolate the action of the muscle before operation;
(b) to exercise it in isolation after operation;
(c) to reintegrate it into the complex reflex pattern of walking

Pre-operative therapy: The tibialis posterior of the affected leg is exercised by placing the ankle across the opposite knee, and inverting against gravity. This exercise is repeated many times each hour until it can be performed without contraction of any other muscle in the calf. Initially, it is helpful for the physiotherapist to apply manual resistance to the foot, and to palpate the calf. It may take anything from one day to three weeks to isolate this muscle, and operation should not be performed until dissociation is complete. The exercise will not increase the strength of the muscle; any apparent enhancement of power represents increasing voluntary control. Patients who are slow to isolate may be helped by a small weight tied to the foot while they exercise.

During this period patients who have a tight tendoachilles undergo passive dorsiflexion stretching. Established contractures are never improved by this, but it sometimes enables borderline cases to avoid surgical lengthening.

Great stress should be placed upon the achievement of dissociation before operation. Time spent in isolation exercises before operation is an investment saving much time later.

Post-operative management: The leg remains in plaster for the time advised by the surgeon; five weeks is the safest minimum. For the first five or six days after operation the patient remains in bed with the limb elevated, and during this time straight leg raising and knee bending is practised. The weight of the cast forms a convenient load for the muscles, and the exercise maintains their tone, promotes circulation, encourages the absorption of exudates from the operation sites, and helps to minimise adhesions. After a week a walking rocker is added to the cast and the patient is ambulated and discharged from the ward with the injunction to continue the general exercises himself.

Patients who have difficulty in achieving dissociation pre-operatively can be helped to 'remember' by practising isolation exercises on the unoperated foot during their time in plaster.

Immediately after removal of the plaster the angle of the foot at rest, in maximum dorsiflexion, and in plantar flexion, is measured. These measurements are repeated at intervals as a record of progress.

The phase of post-operative isolation exercises now begins. The leg is crossed as before, and the patient attempts to invert, while watching his foot dorsiflex. The physiotherapist palpates the calf to detect contraction of other muscles. If other muscles are felt to contract, then the therapist must sit with the patient and help him 're learn' isolated contractions. After two or three days the patient will be accustomed to dorsiflex by attempting inversion, and the exercise may now be performed against gravity. Between exercise periods a plaster backslab is worn, the foot being held at 75° dorsiflexion, and it is most important that the patient should not at any time remove it except when exercising with his therapist. It is an advantage to have the patient in bed during this period, but failing this, he may non-weight bear on crutches.

Two points require special mention. A minority of patients on coming out of plaster, will be found to have forgotten how to contract their tibialis posterior in isolation. They exhibit a movement consisting of a plantar thrust (calf) followed by a recoil which mimics dorsiflexion. This pattern *must* be stopped before it becomes established. The physiotherapist must sit with the patient and carefully instruct and supervise until the correct movement can be performed. These patients require intensive personal attention, are slow to achieve dissociations, and may relapse if vigilance is relaxed.

The other point concerns the quality of muscle contraction. Many physiotherapists are content for their patients to practise 'Jerky' dorsiflexion contractions of short duration. It is desirable that once the movement of dorsiflexion is established that the muscle should perform sustained contractions in preparation for its function during walking. This type of contraction is also more effective in stretching adhesions. After the first few days, therefore, the patient is encouraged to carry out sustained contractions lasting two or three seconds, and of gradually increasing power. This culminates in the addition of weight to the foot, gradually increasing to about three or four hundred grammes.

Initially, exercise periods are kept short. On the first day two periods of not exceeding five minutes each should be allowed, increasing to three periods on the second day, and gradually lengthening thereafter. Each case must be managed on its merits. The appearance of warmth and/or swelling is an indication to slow down treatment or institute rest: if persistent, some complication (such as stitch sepsis) should be considered.

This initial phase of treatment ends when examination reveals complete dissociation of dorsiflexion from contraction of other calf muscles. Dissociation is decided by careful palpation of the calf during dorsiflexion, and by the following test. The patient dorsiflexes, and by means of a finger the examiner exerts gentle pressure upwards on the ball of the foot, while palpating the calf with his other hand. On the order to relax, softening of the calf combined with further dorsiflexion is evidence that dissociation is incomplete. By the time disociation is complete, favourable cases will have an active range of 20° or more, and active dorsiflexion up to 80° or 75°.

The phase of integration now begins, and partial weight bearing on crutches is introduced. For the first few days the backslab should be retained for wear at night, but after that it can be discarded. Dorsiflexion exercised with weights applied to the foot are advantageously continued throughout. The patient is taught to walk in a heel-to-toe manner, with exaggerated dorsi-

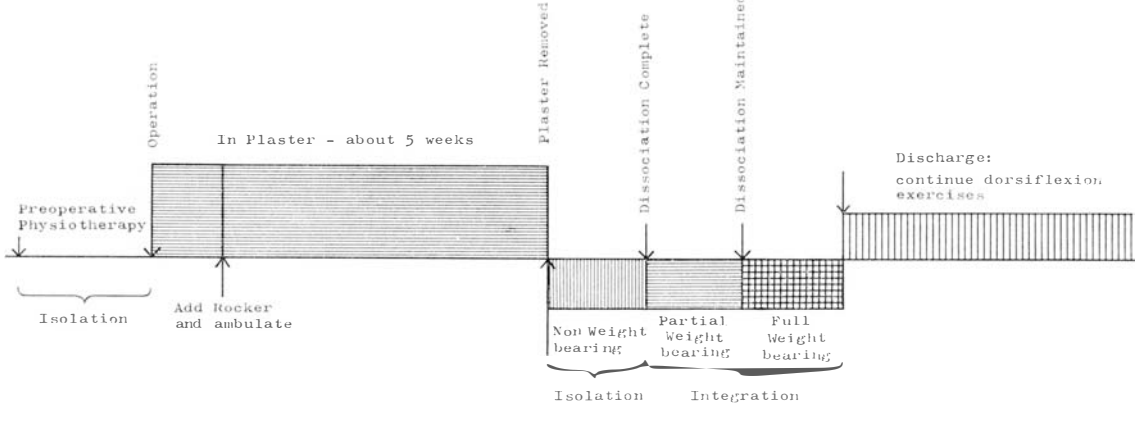
flexion during 'swing through' of the operated foot. This is a time for vigilance on the part of the physiotherapist. Some patients 'forget' their carefully rehearsed dissociated dorsiflexion when faced with the complex pattern of walking, and integration of the new movement has to be deliberately and painstakingly practised. With time the pattern of walking becomes established, dissociation is maintained, and full weight bearing may then commence.

During the phase of weight bearing, the foot will 'drop' by about 5°, and any slight calcaneus deformity will disappear. The late dropping should be allowed for in the tensioning and immobilisation of the transfer at operation. A foot which appears to be too 'high' can be brought down by allowing partial weight bearing a little earlier than would otherwise be indicated and by discarding the back-slab early.

The patient is ready for discharge when, after some days of weight bearing, it is clear that dissociated dorsiflexion has become firmly absorbed into the walking cycle. The elderly should remain under observation for a longer period. It takes them longer to learn the new walking sequence, and there is a danger of relapse into a 'confused' gait if they are discharged too soon.

A warning should be given against adoption of a rigid post operative timetable. It is true that favourable cases can be ready for partial weight bearing within seven days of plaster removal, and can be ready for discharge in three or four weeks, but rigid application of such a schedule will ruin many results. A fault of some therapists is to attempt to force cases through the post operative course in accordance with a preconceived schedule. Experience has shown that patients, who vary in age, intelligence, powers of concentration, and motivation towards rehabilitation very also in their rate of progress after surgery, and no two cases are exactly alike. The technique selected for the operation also has its effect. The time for advancement to the next stage of treatment must be decided in accordance with the criteria outlined above. If, at any time errors in re-education are detected there should be no hesitation in starting treatment again from the beginning. It is better for post operative physiotherapy to occupy three months than for a patient to be hurried into the next stage before he is ready for it. Many of the late failures (i.e. patients presenting with high stepping gait and 'relapsed' drop at follow up) are attributable to hurried re-education and premature discharge from treatment.

Electromyographic studies on tibialis posterior after transfer have shown that at first the muscle discharges during both dorsiflexion and plantar flexion. As re-education proceeds, the discharge during plantar flexion diminishes, while electrical activity during dorsiflexion increases. Even when complete clinical dissociation is seen, slight electrical activity may persist during plantar flexion. This serves to remind that this muscle may always retain a 'memory' of its previous function, and that for some months after operation it retains a tendency to revert to its original pro-gravity function. For this reason it is wise to encourage the patient to practice



isolated dorsiflexion exercises against gravity with the foot weighted for at least six months after discharge, and to examine his gait, dissociation, and range of movements regularly for at least a year. In this way the incidence of late relapses may be minimised.

SUMMARY

The cardinal points in the physiotherapy of foot drop correction may be summarised by the following 'do's and 'don'ts'.

(1) Don't operate until the patient has achieved perfect isolation of the muscle.

(2) Don't start weight bearing until disociation is perfect.

(3) Do look our for (i) trick novements which may have deceived the therapist: (ii) failure to integrate the new movement when weight bearing is started.

(4) Don't hesitate to delay introduction of the next phase, or to order re-education again from the beginning if the patient exhibits 'confusion'.

(5) Don't apply a timetable to the patients' progress.

(6) Do see each patient regularly for at least a year from operation, and urge him to persist with dorsiflexion exercises.

REFERENCES

ANDERSEN, J. (1963). Acta. Ortho. Scand. **33** Fasc. 2, 151

ANDERSEN, J. (1964). *Leprosy Rev.*, **35,** 41.

GUNN, D. R. and MOLESWORTH, B. D. (1957). The Use of Tibialis Posterior as a Dorsiflexor. *Journal of Bone and Joint Surgery* **39-B,** 674, 1957.

SELVAPANDIAN, A. J. and BRAND, P.W. (1959). Transfer of the Tibialis Posterior in Foot Drop Deformities. *The Indian Journal of Surgery*, **21.** 151, 1959.

WATKINS, M. B., JONES, J. B., RYDER, C. T., Jr., BROWN, T. B., Jr. Transplantation of the Posterior Tibial Tendon. *Journal of Bone and Joint Surgery*, **36-A,** 1181. 1954

LENNOX, W. M. (1964). The Surgical & Physiotherapeutic Management of Foot Drop. *Proc. Orthopaedic Section, Assoc. of Surgeons of India*. December 1964.

MENDES, M., *Further Reading on Physiotherapy*; Physiotherapy in Leprosy. John Wright & Son, Bristol 1964.

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Experimental Moulded Soles and Shoe Lasts

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For a period of 6 years rigid soled shoes have been recommended for the prevention of reulceration of the deformed and anaesthetic feet of leprosy patients (Price 1960 and Ward 1962). The dramatic reduction of plantar pressures given by a flat rigid soled shoe was demonstrated by Baumann *et al* (1963). At the Christian Medical College Hospital, Vellore, S. India, and the Schieffelin Leprosy Research Institute, there are many cases of deformed feet that have been kept free of ulcers by the use of these shoes. But it has long been a worry that these shoes cause instability at the tarsal, especially on rough ground. A study of tarsal disintegration (Harris, Brand) has given rise to the thought that this instability may be a factor related with later tarsal disintegration, especially when the shoes have been used immediately after plaster of Paris immobilization. Apart from the instability caused by the rigid soled shoe the weight and the bulk of the shoe makes it unpopular with the patients.

With these factors in mind it was decided to try and develop a new design of shoe that would prevent reulceration but not have the disadvantages of the rigid soled shoe. To decrease the instability the foot should be as near the ground as possible and this would also help eliminate the bulk of the shoe. It was felt that an accurately moulded insole would help distribute the walking plantar pressures as well as give support to the tarsal area of the foot. The mould would have to be in the shape of the walking foot and not of the foot when the patient was standing or sitting. For this reason the standard orthopaedic method of making a moulded sole would not be suitable. Various materials were experimented with to find one that would be inexpensive, easy to handle and would give the correct moulding. Brand found a method of using rubber latex and cork dust which was suitable for our needs. A system of construction has been developed and this is now being used to make footwear for a selected number of deformed feet. This paper explains and gives details of the method used. The shoes described are meant to be used for the deformed, badly scarred or shortened feet that can not be kept free of ulcers in the standard microcellular rubber sandal (Price 1960 and Ward 1962). At this stage it is too early to say that this is the answer to our problem and the footwear is still in the experimental stages even though the results have so far been encouraging. A method is also described for making accurate shoe lasts for deformed feet. Again the number that have so far been made is small, but with this method it is possible to obtain an accurately fitting upper, an important factor when the foot must be held firmly in place on a moulded sole.

For the foot that is not badly deformed but has an uneven or badly scarred plantar surface moulding is going to help distribute the weight more evenly over the plantar aspect. For these cases measurements needed for an orthopaedic shoe are taken (British Standard 1961). The shoes are made on a standard last that has been adapted to suit the measurements and minor deformities of the foot. The bottom of the last is built up with layers of leather, corksheeting or

* Sponsored by the Swiss Emmaus Association.

cardboard to a depth of 15 mm to allow room for the insole. The shoe is then made on the last in the usual way.

At the fitting of the shoe a mixture of coarse cork dust and rubber latex is made up in volume; six of cork to one of latex. Raw rubber latex can be used but it is better to use pre-vulcanized latex when possible, as it makes a stronger mould. When an open design of sandal is being used, strips of X-ray film are placed down the insides of the upper to prevent the cork from being squeezed out of the gaps. The cork and latex is spread in the shoe to a depth of about 20 mm, and left loose and not pressed down. A slightly greater amount is put in the area of the medial arch or other non-weightbearing areas of the foot depending on the deformity. The microcellular rubber insole 15 to 20 Shore; 7 mm thick is put in over the top of the cork (Fig. I). It will be found necessary to press the

FIG. I

The foot in the sandal ready for forming the mould. It will be noticed that at the foot especially the heel is too far out of the sandal. After 15 minutes of walking the foot will have settled down correctly inside the sandal. The X-ray film across the medial arch is preventing the cork from being squeezed out. The black microcellular rubber can just be seen between the cork and the foot.

rubber down a little to allow room for the foot to slide into the shoe. A design of upper that opens right down to the toes eliminates the danger that the sole will be incorrectly pressed at this stage forming wrong moulding. The patient is asked to stand and then walk about for five minutes. The moulding is checked to see that it is high enough up into the arch. This is done without removing the shoe, an open design of sandal makes this job easy; with a closed shoe it is nearly impossible and the checking has to be done at the finishing. The patient should wear the shoes for at least half an hour, half of this time should be spent in gentle walking on an even surface. The shoe is then removed and any cork that has come up over the sides of the microcellular rubber is removed. In a sandal where the cork can be seen in the gaps, the X-ray film is removed and the cork is painted with the sole dye or a strip of leather is stuck onto it. It will be found that the cork presses up under the toes and onto the front of the toes. In sandals this cork can be removed at the finishing stage. When a closed shoe is being used a wad of cotton-wool should be placed in the toes of the shoes before the cork is put in. This piece of wool can then be removed at the finishing stage (Brand).

Shoe Lasts for badly deformed and shortened feet

It is very difficult and time consuming to carve correctly shaped wooden lasts for deformed feet, to get over this difficulty we make the lasts from a negative plaster cast, out of polyester resin and cork dust.

Plaster cast, materials needed:

- Sheet of 20 mm thick 15 Shore microcellular rubber 20 x 30 cm.
- Pieces of 3 mm thick sponge rubber
- Rubber or plastic tube 8 mm diameter, length 30 cm.
- Plaster scissors
- Indelible pencil
- Two 4 inch quick setting plaster bandages
- Vaseline.

Method

The patient is asked to stand with the deformed foot on the microcellular rubber sheet. Pieces of sponge rubber are then placed under the toes to give as much toe spring as is possible but without creating too much pressure on the toes (Fig. II). With a shortened foot this is very nearly impossible and the foot has to be left flat. It is not the aim at this stage to create a great deal of moulding to the sole as the moulding will be formed at the finish with the cork and latex insole. The patient sits while the foot is vaselined.

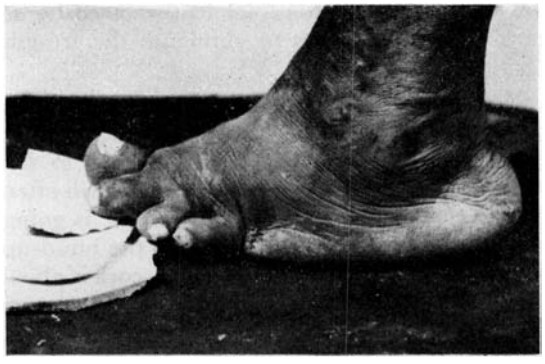

FIG. II

The foot on the microcellular rubber with the sponge rubber forming the toe spring in position.

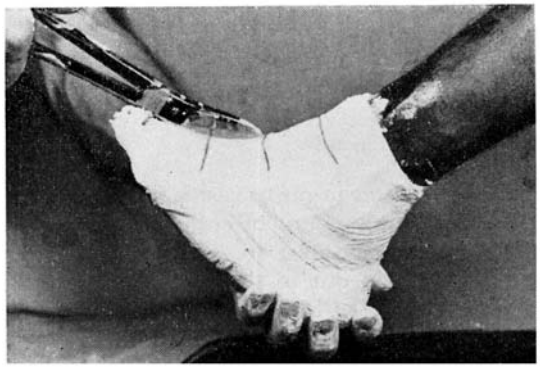

FIG. IV

With the rubber tube removed, cutting up the dorsum of the plaster.

The rubber tube is placed along the dorsum of the foot and is held in place by an assistant or by the patient himself. The foot is wrapped completely in plaster bandage to just above the maleoli, care being taken that there is adequate thickness around the heel. The foot is placed again on to the microcellular rubber sheet with the sponge rubber forming the toe spring in the correct position. The patient is asked to stand and to take equal weight on both feet. The plaster is moulded with the hands around the heel, ankle and the dorsum of the foot. With the indelible pencil lines are drawn across the bump formed by the tube (Fig. III). Just before the plaster starts to become warm the patient is asked to sit. The tube is pulled out and the plaster scissors are introduced into the gap left by the tube. The plaster is then cut right up the dorsum from the toes (Fig. IV) and removed from the foot by sliding it backwards and downwards; care being taken that the heel is not pushed out of shape, especially in the cases where the patient has a posterior protruding heel (Fig. V). The plaster cast is then closed up again aligning the pencil marks. Another plaster bandage is wrapped round it. The patient's name is written on the outside and it is left to dry.

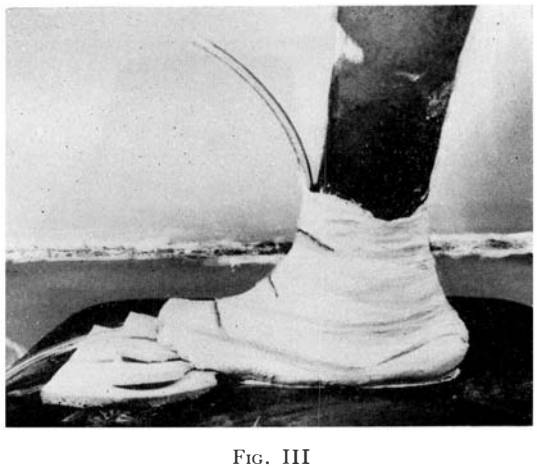

FIG. III

The plastered foot with the rubber tube in place. The patient is standing while the plaster dries.

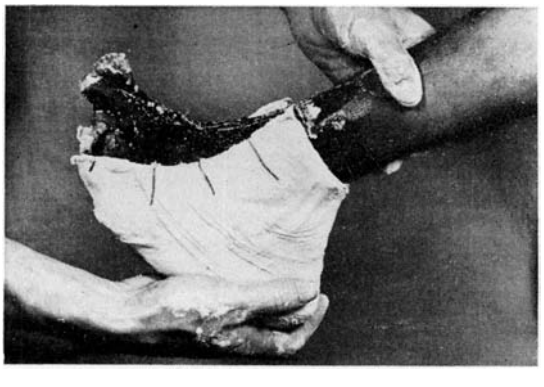

FIG. V

Sliding the plaster cast backwards off the patient's foot.

Making the Last

The last is made from polyester resin and cork dust, a method suggested by Mr John Gleave of Hong Kong.

Method

The dried plaster negative cast is filled with dry cork dust. The amount of cork dust needed is then weighed and put to one side. Polyester resin is weighed out to the quantity of 100% resin to 40% cork. The polyester resin is mixed in the usual way (Polyester Hand Book 1963) and then the cork is mixed well into it. The cork and polyester is put into the negative cast, a little at a time and is pushed well down using a spatula, care being taken that the toes are adequately filled. The cast is left to set for 24 hours. The plaster is then removed. If it is found that the surface of the last is at all crumbly then a further mixture of polyester can be painted onto it. A greater percentage of polyester can be used in the initial mixture, but this is apt to make the last too hard for the shoemaker when hammering in nails.

Last Adaptation

If a closed toe design of boot is going to be made the front of the last will have to be built out to a depth of 25 mm. with leather. The leather is tacked on while it is wet so that it can be formed to the correct shape; it should be in the shape of a toe cap and allow clearance inside the boot for the toes. If an open toe boot is to be used then only one layer of 6 mm thick leather needs to be tacked on to the front to eliminate the irregularities of the toes (Fig. VI).

As with other leprosy footwear where an insole is to be used inside the finished shoe the bottom of the last has to be built up to the required depth. The bottom of the last will often be uneven. This means that the insole is going to be of varying thicknesses and so the build-up must be the same. A sheet of 3 mm. cork is glued on to the bottom of the last. The high parts are then rasped down until the last shows through. Another layer of cork is glued on and also rasped down. This is done until the bottom of the last is flat medial to lateral but not eliminating the toe spring. Once the sole is flat, thicker pieces of cork can be used for completing the build-up. The thickness of the build-up depends mainly on the shape of the foot but at least 10 mm. should be left under the lowest part to allow room for a slight thickness of cork and latex moulding as well as the microcellular rubber inside the boot. The side of the cork build-up must be perpendicular when the last is in the upright position and not flare outwards or inwards; it should also blend into the sides of the last (Fig. VII).

If there is insufficient toe-spring in the last and the last has been adapted for a closed toe boot, the cork build-up under the leather packing can be rasped down to form a little toe-spring. This can be further increased by lightly glueing one or two layers of cork sheeting under the meta-

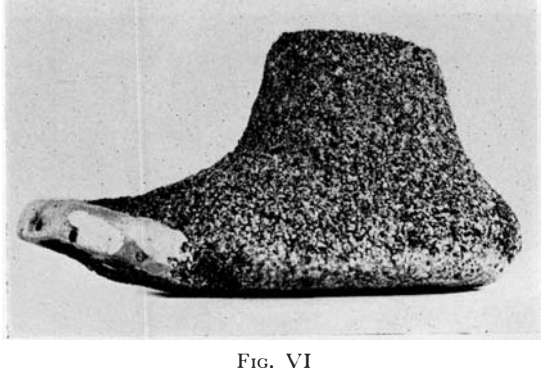

FIG. VI

The cork and polyester last, showing the toe spring or roll that has been formed by the sponge rubber padding and also the leather build-up over the toes in readiness for an open toe boot. The protruding heel can be seen.

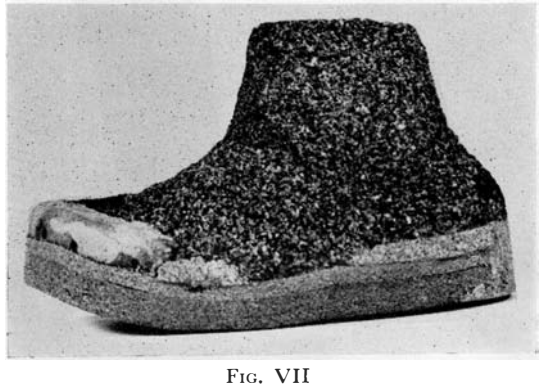

FIG. VII

The last with the cork build-up. This last is now ready for the shoe-maker.

tarsal shaft area of the last, these are rasped down to blend into the cork build-up and to form a roll. When the boot is finished this piece of cork is removed and glued in place inside the boot.

The last is now ready for the shoe maker. If he is not experienced in the making of orthopaedic footwear he may find difficulty to cut the correct shaped paper pattern for the upper. This can be solved by sticking strips of adhesive tape onto the last; these are then removed *en masse* and used as the model for cutting the paper pattern (Fig. VIII). A heel is usually not needed, but a low one should be used if the foot is going to be at all dorsiflexed when the patient stands.

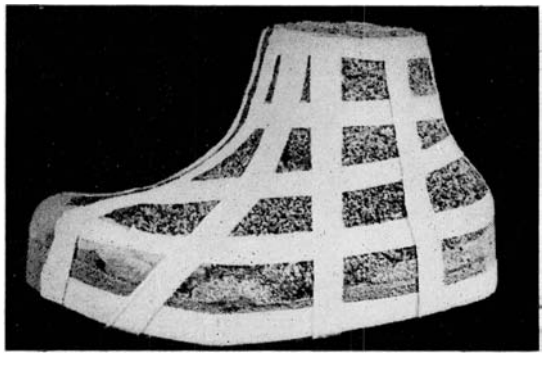

FIG. VIII

The last with strips of adhesive tape which will be removed en masse and used to cut the paper pattern.

At the finish the moulded cork and the latex insole is made in exactly the same way as already described. When an open toe boot is used, a strip of X-ray film is tucked into the front to prevent the cork from squeezing out (Fig. IX). At the finish it is removed and a strip of leather put in its place.

This work was supported by grants from the office of Vocational Rehabilitation, Department of Health, Education and Welfare of the United States Government.

FIG. IX

The moulded cork insole being formed. The strip of X-ray film can be seen preventing the cork from squeezing out of the front.

We wish to acknowledge thanks to the Swiss Emmaus Association for the training stipend that was provided for Mr J. P. Girling while he was in Europe. We also thank the firm of Scott Bader & Co. Ltd. for the technical advice and free materials that was given during the initial stages. Mr Sigamoney is thanked for the photographs.

REFERENCES

1. BAUMANN J. H., GIRLING J. P., BRAND P. W. (1963), Plantar Pressures and Trophic Ulceration, Journal of Bone and Joint Surgery, 45–B, 652.
2. BRAND P. W., Personal Communication.
3. British Standard 3350 (1961), Method of Measurement for Surgical Footwear.
4. GLEAVE J., Personal Communication.
5. HARRIS J., BRAND P. W., Patterns of Disintegration of the Tarsus in the Anaesthetic Foot, Journal of Bone and Joint Surgery, Accepted for Publication.
6. PRICE E. W. (1960), Studies of Planter Ulceration in Leprosy, VI: The Management of Plantar ulcers, *Leprosy Review*, 31:159.
7. Polyester Handbook 1963, Scott Bader & Co. Ltd., Printers Percy Lund, Humphries and Co. Ltd. London.
8. WARD D. J. (1962), Footwear in Leprosy, *Leprosy Review* 32: 94.

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Nerve Abscesses in Leprosy in Northern India

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Recently the subject of nerve abscesses in leprosy has been reviewed by Sehgal and Gupta, emphasizing the rarity of the condition. Similar observations have however been made time and again by various workers like Lowe (1934), Mukherjee (1956), Gupta (1962), Wheate (1964) and Brown (1957, 1965), from different parts of the world. No definite figures are available from India, but it has been stated that in India; especially north (Int. J. Leprosy 1955), nerve abscesses are of relatively frequent occurrence. In the present paper two cases of nerve abscesses are reported from the area, where not many cases have been recorded.

CASE HISTORIES

CASE 1

J. N., 30 years, male, attended the section of Dermato-venereology on 27th October 1965 of S. S. Hospital, BHU, Varanasi, with a well defined erythematous patch situated on the ulnar side of the dorsum of the right hand, having impaired sensations. This, according to the patient, started about seven months prior to attending the hospital. ON EXAMINATION: A well defined shiny erythematous patch was found on the dorsum of the right hand confined to the ulnar side. SENSATIONS: Temperature and touch were very much impaired. NERVES: Ulnar, median and radial nerves were thickened and tender. Clinically a diagnosis of *tuberculoid* leprosy was made. The biopsy from the skin lesion was taken and studied histologically. The section revealed mild hyperkeratosis, with atrophy and thinning of the epidermis. The dermis showed focal collection of chronic inflammatory cells, more so around the adnexa of the skin (Fig. 1). No acid-fast bacilli could be demonstrated.

The patient was put on *diamonodephenyl sulphone*. While he was on treatment (after two months) he noticed a small swelling, a little above the elbow joint. This went on increasing progressively. During the period he also experienced weakness and wasting of the muscles of the right hand. ON EXAMINATION: A swelling of the size of 1½″ x 1″ was found, showing fluctuation and tenderness along the ulnar nerve, just above the elbow joint (Fig. 2). TREATMENT: A vertical incision of the size of two inches was made along the ulnar nerve in the elbow region. On complete exposure, a swelling of the size of 1½″ x 1″ was seen along the thickened nerve (Fig. 3). An incision was made in the nerve sheath (Fig. 4). the pus drained out and the wound sutured. After the exposure and drainage the patient felt much better and constitutional symptoms lessened. Smears from purulent discharge showed degenerated polymorphonuclear leucocytes. No acid-fast bacilli could be demonstrated.

CASE 2

R. A., 12 years, male was admitted to medical ward of S. S. Hospital with an anaesthetic patch on dorsum of the right foot of one year duration. The patient did not pay attention to it, till he noticed a progressively increasing painful swelling just above the patch. In the course of six months, he developed two more similar swellings along the sural nerve, and two along posterior tibial nerve. They were very painful. ON EXAMINATION: A well demarcated reddish, atrophic patch of the size of one inch diameter was seen. SENSATIONS: Temperature and touch were impaired over the patch. NERVES: Lateral popliteal, sural and posterior tibial were thickened. SWELLINGS: Five swellings were seen, which varied from half to one and a half inches in size. They were fluctuating and located three along the sural and two along the posterior tibial nerve. The clinical diagnosis of *tuberculoid* leprosy with multiple leprotic nerve abscesses was made. Histopathology of the skin lesion showed tuberculoid granulomata. Two of these abscesses were drained surgically to relieve the acute symptoms. Smears from the pus showed degenerated leucocytes. However no acid-fast bacilli could be seen.

COMMENTS

Two cases of leprotic nerve abscesses are reported, in which clinical features and biopsy studies showed the evidence of *tuberculoid* leprosy. It is believed that leprosy nerve abscesses occur more frequently in tuberculoid type. Development of nerve abscesses during its course shows high degree of immunity as mentioned by Dharmendra (1960). Better prognosis may therefore be expected in such patients. However, it is worth undertaking operative procedure, as attempted in the present cases, to relieve the acuteness of the symptoms and signs, because the severity of the

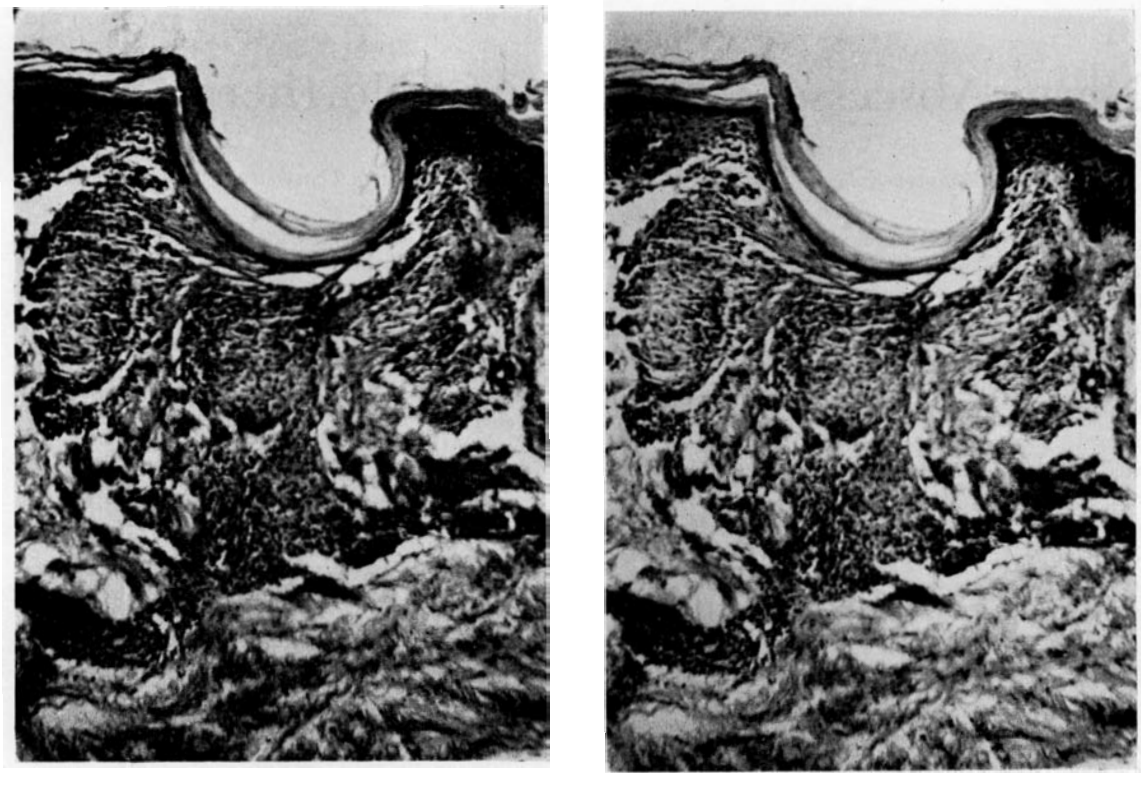

FIG. 1. Skin Histology of nerve abscesses.

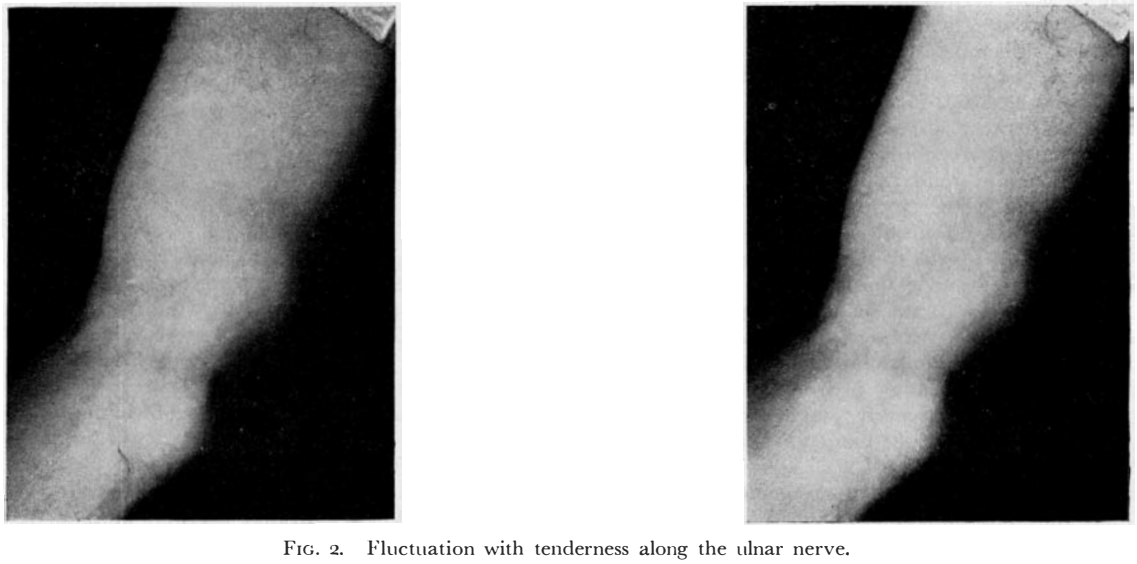

FIG. 2. Fluctuation with tenderness along the ulnar nerve.

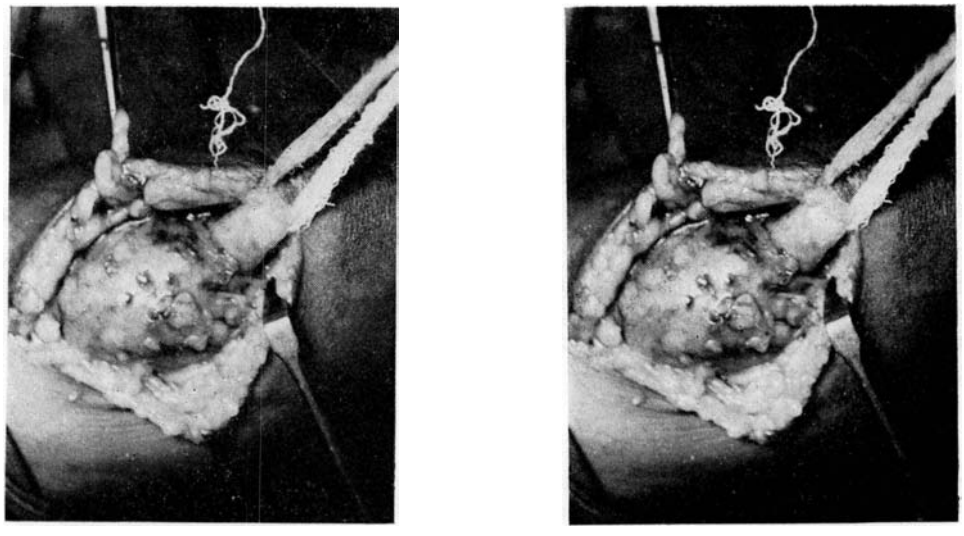

FIG. 3. Thickening of the nerve.

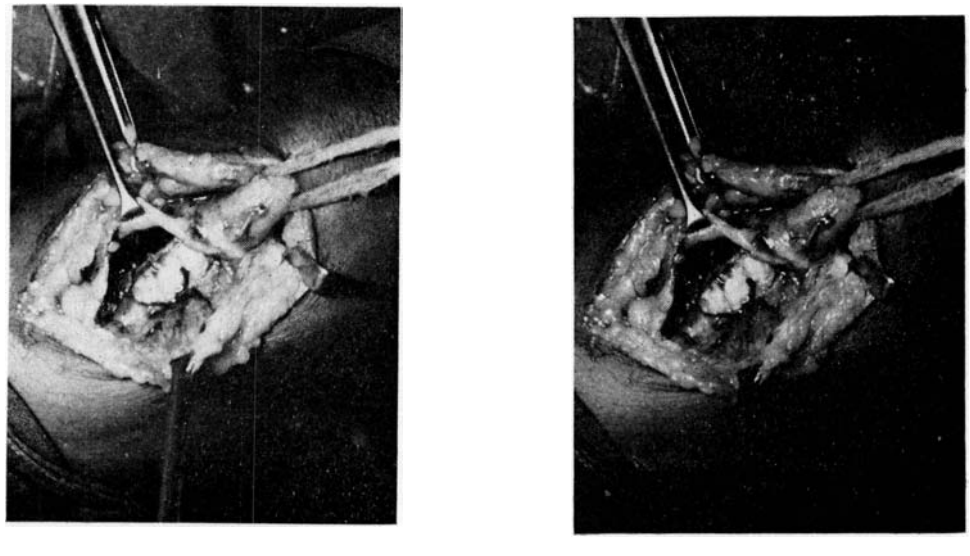

FIG. 4. Incision of nerve thickening.

neural signs is in direct proportion to the pressure exerted by the pus on the nerve fibres.

SUMMARY

Clinical features and corroborative laboratory findings in cases of leprotic nerve abscesses are described and their relatively infrequent occurrence is emphasized.

ACKNOWLEDGEMENTS

I gratefully appreciate Dr B. P. Verma, Lecturer, Orthopaedic Surgery, for carrying out the operative procedure, and Mr Grish Saxena for photography.

REFERENCES

BROWNE, S. G. (1957?): *Leprosy Rev.*, **28**, 20.
BROWNE, S. G. (1965): *Leprosy Rev.* **36**, 55.
DHARMENDRA (1960): Notes on Leprosy, The Ministry of Health, Govt. of India, p 37.
EDITORIAL (1955): Int. J. Leprosy, **23**, 69.
GUPTA, R. L. (1962): *Leprosy in India*, **34**, 205.
LOWE, J. (1934): Int. J. Leprosy, **2**, 304.
MUKHERJEE, N., and GHOSH, S. A. (1956): *J. Ind. Med. Assoc.*, **27**, 291.
SEHGAL, V. N. and GUPTA, I. M.: *Multiple Leprotic Nerve Abscesses in a Child* (Under publication).
WHEATE, H. W. (1964): *Leprosy Rev.*, **35**, 86.

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The Distribution of First Lesions in Leprosy

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During 1963 there was some discussion in the columns of Leprosy Review concerning the mode of entry of *Mycobacterium leprae* into the body (Spickett, 1963; Weddell & Palmer, 1963; Weddell, Palmer & Recs, 1963). It was suggested that some further work on the sites of first lesions might be of interest. While doing other epidemiological work on leprosy in South India, members of a Cambridge medical expedition were able to collect data about the sites of first lesion in 252 cases of leprosy.

The cases included in this study are those in which a clear single first lesion, hypopigmented patch or anaesthetic patch was described in the clinical notes. The results are divided into three groups according to the age at onset of leprosy, information obtained from clinical notes and occasionally by direct questioning of the patient. Bearing in mind that primary infection would have occurred about 5 years before recognisable symptoms appeared, an age of onset of 9 years or less may be regarded as probable infection during infancy, and 10–18 years, infection during childhood.

If the distribution of lesions is random, one would expect the distribution of lesions in different parts of the body to be proportional to their surface areas. These have been calculated using the Berkow scale (Berkow, 1924) making allowances for differences in shape at different ages. (Table 1.)

TABLE 1
Distribution of first lesions according to age

Site of first lesion	*Adults* (> 18 *years at onset*)		*Children* (10–18 *years at onset*)		*Infants* (< 10 *years at onset*	
	Expected	*Observed*	*Expected*	*Observed*	*Expected*	*Observed*
Head	12	14	8	6	6.5	7
Arms & Hands	25	42	16	24	7	7
Trunk, Buttocks & Thighs	66	39	43	38	17	18
Legs & Feet	27	35	18	17	6.5	5
Totals	130		85		37	

It can be seen that for adults the distrubution differs significantly from the random ($X^2_3 = 22$. $P = < 0.0001$). In infants the distribution is as predicted and in children it is intermediate to that in adults and in infants.

If, as has been suggested, blood dissemination occurs from the site of entry early in the disease, it would be expected that the distribution of first lesions would be random. If such were the case one would expect all age groups to show this random distribution, but in the above data this is true of infants and possibly of children. These results are more compatable with siting of the first lesion by some non-random process which affects the whole body to the same extent in infants, but on reaching maturity is more limited in its extent. The figures show a striking deficiency in lesions on the trunk, buttocks and

thighs in the adult group, and it has been observed that these are the parts of the body that are covered by clothing in this part of India. The excess of lesions found in the limbs is also found in the head and neck if the area covered by hair is excluded. Infants are completely unclothed and during the early years are handled to a great extent by their mothers, and this fits the observed random distribution of lesions in the infant group.

Our series showed no difference in the distribution of lesions in the two hands. (Table 2.).

TABLE II

First lesion on hand or forearm

Right	*Left*
24	28

We would therefore suggest that these figures support the hypothesis of entry through the skin, mediated by person to person contact or by minor trauma.

We should like to thank the medical staff of Karigiri, the Christian Medical College, Vellore and Vadathorasalor who allowed us to make use of their notes, the members of the Medico-Sociological Expedition and the many others in England and India who made this work possible.

This work was supported by grants from the Medical Research Council, the British Leprosy Relief Association and The Leprosy Mission.

REFERENCES

BERKOW, S. G. (1924). A method of estimating the extensiveness of lesions, burns and scalds, based on surface area proportions. *Arch. Surg. Lond.*, **18**, 138.

SPICKETT, S. G. (1963). Letter to the Editor. *Leprosy Review*, **34**, 154–156.

WEDDELL, G. & PALMER, ELIZABETH (1963). The pathogenesis of leprosy: an experimental approach. *Leprosy Review*, **34**, 57–61.

WEDDELL G., PALMER, ELIZABETH and REES, R. J. W. (1963). Letter to the Editor. *Leprosy Review*, **34**, 156–158.

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Role of an Indigenous Drug (*Achyranthes Aspera*) in the Management of Reactions in Leprosy Preliminary Observations

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INTRODUCTION

The treatment of reactions in leprosy has been largely empirical. Antimonials like potassium antimony tartrate (PAT), Fantorin and Stibatin have been mainly used for the past several decades. The antimalarials have come into use in recent years. In addition to these, there is yet another most potent group of drugs namely the corticosteroids (2).

Although, the drug of choice for these episodes are antimony preparations, especially 'PAT' because of its cheapness and therapeutic efficacy, but its use is restricted in rural conditions due to difficulty in its mode of administration, as it has to be administered intravenously and intromuscularly. Under these conditions antimalarials that could be administered orally come next in preference. But they are also not very safe considering their toxic manifestations in general. Corticosteroids are to be used in cases of severe or persistent reaction, agonising neuritis, or potentially serious eye complications. A serious drawback of corticosteroids is the rebound phenomenon when this therapy is discontinued. Therefore, further research is not unwanted.

Tripathi *et al* (1963), (3), have observed that an indigenous drug 'Achyranthes aspera' of Natural order Amarantaceae, seems to be effective, in reaction and quiescent stage of the disease. In order to obtain additional data on this indigenous drug, the present study was undertaken.

MATERIAL AND METHODS

A group of 12 cases of reaction selected from the leprosy clinic of the out patient department of S. S. Hospital, B.H.U., formed the material for study of the therapeutic aspects of the reactive state and were studied in detail from the clinical, bacteriological and immunological points of view before starting the actual drug therapy. Criteria of diagnosis, of types, stages and grades of reactions in leprosy are given in Figs. 1, 2 and 3 respectively.

All these cases were taking regular treatment from our out-door Leprosy-clinic which they were visiting twice a week. During the trial the dose of anti-leprosy drug DDS was either reduced or completely withdrawn, depending upon the severity of reaction. For the treatment of reaction a decoction of whole plant of Achyranthes aspera (Fig. 4), prepared by the methods described in classics of Ayurveda, was given orally. The dose of the decoction was 1 oz. twice daily. The patients in the stage of acute reaction were advised to take rest in bed. Intercurrent pathological conditions were attended duly. They were advised to take nutritious diet within their means and to observe the hygenic conditions as far as possible.

The ordinary treatment of leprosy patients was on a dosage of 10 mgm. of DDS daily which is increased by 10 mgm. every week to a maximum of 100 mgm. daily for 6 days a week. During the reactions the DDS dosage was either reduced or completely withdrawn as needed. For the treatment of reaction decoction of Achyranthes aspera was given orally at a dosage of 1 oz. decoction twice daily. The bacterial index was calculated in each case by taking smears from 8 sites.

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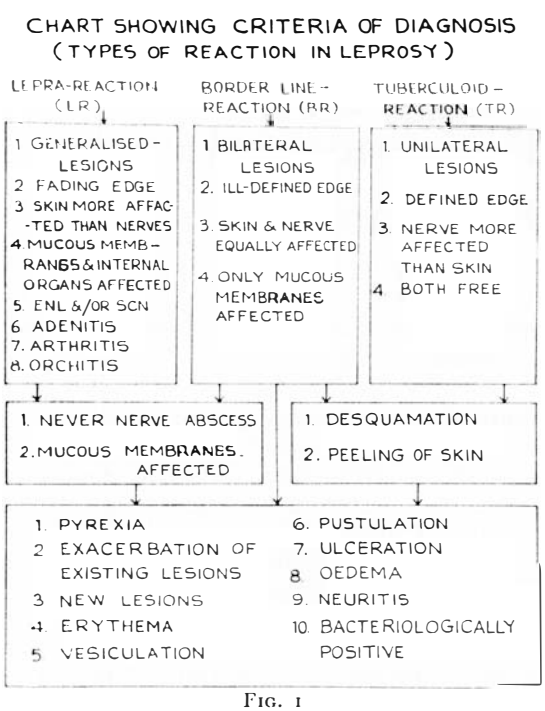

FIG. 1

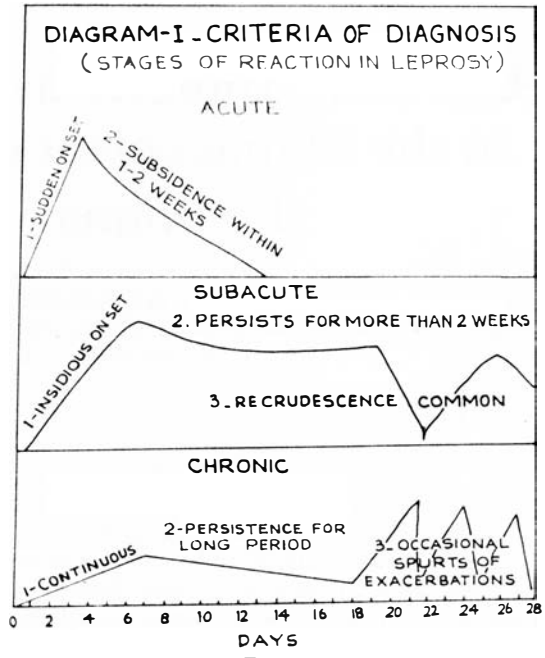

FIG. 2

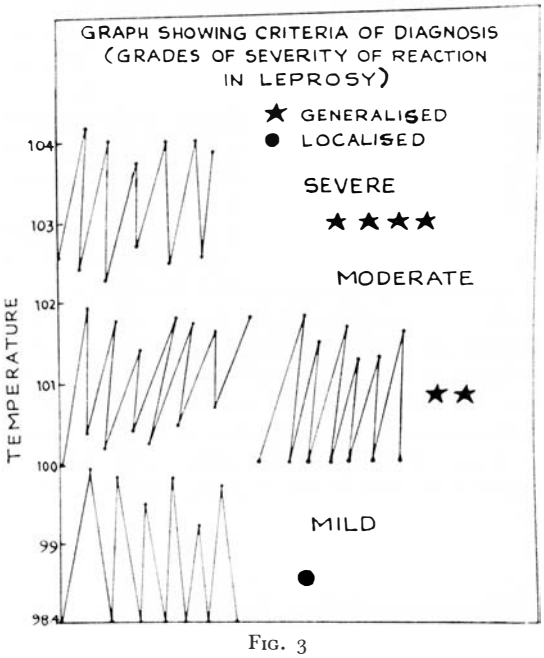

FIG. 3

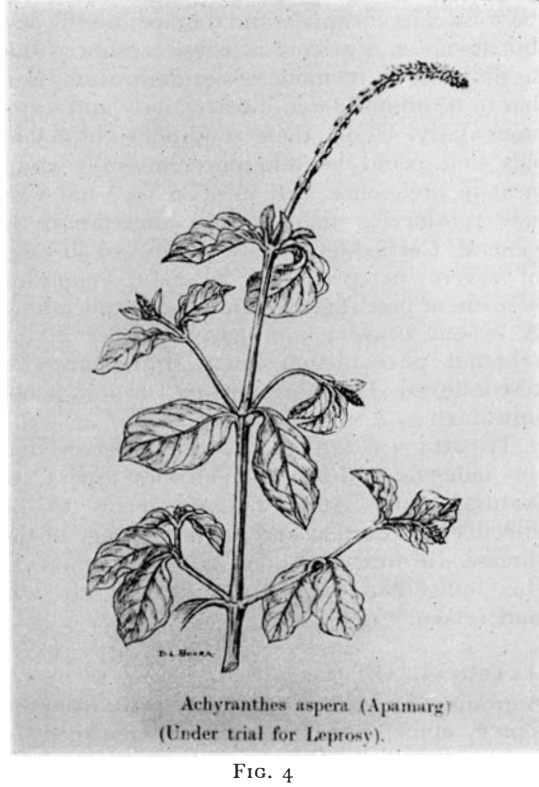

FIG. 4

Records of their initial condition and their successive progress were maintained. Smears were taken and studied at least twice before the beginning of the treatment to minimize errors. History and clinical conditions of the cases were recorded by filling up history sheets prepared especially for the purpose; Clinical pictures of the cases were charted with the help of notations given in 'Notes on Leprosy' by Dr Dharmendra (1). Weight records were maintained. Bacteriological Index (B.I.) was calculated in each case by taking smears from 6 sites and adding the degrees of positivity of all the smears and dividing the total by the number of smears examined for future assessment (1). Complete Haematological examinations were done. Urinalysis and stool examinations were also carried out.

The results were assessed clinically and bacteriologically after 30 days of the treatment.

OBSERVATIONS AND RESULTS

Out of 12 cases under investigation, 6 were of acute and subacute type of lepra-reaction, 3 of acute and subacute type of borderline reaction and 3 of subacute type of tuberculoid reaction. All these cases were in varying grades of severity, mild, moderate and severe (Fig. 5). Age and

TABLE SHOWING CLINICAL OBSERVATION
(STAGES AND GRADES OF SEVERITY OF REACTION CASES)

	TYPES OF REACTION			
STAGES	LR	BR	TR	GRADES
ACUTE	•	•	•	MILD
	•	•	•	MODERATE
	人	人	•	SEVERE
SUB-ACUTE	人人人	人人	人人人	MILD
	•	•	•	MODERATE
	•	•	•	SEVERE
CHRONIC	•	•	•	MILD
	•	•	•	MODERATE
	•	•	•	SEVERE

Fig. 5

DIAGRAM SHOWING AETIOLOGICAL OBSERVATIONS
(AGE & SEX INCIDENCE IN REACTION CASES)

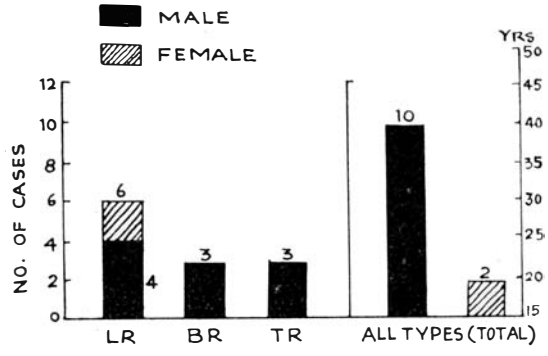

Fig. 6

sex incidence in reaction cases is given in Fig. 6.

Most of the cases showed a clear fall in the bacteriological index as well as clinical improvement. The most striking clinical improvement noted with this treatment was complete subsidence of Oedema which was invariably found in all the cases under investigation. It seems that the present drug under investigation has some anti-inflammatory as well as diuretic property which is yet to be confirmed. The cases also gained in weight and increase in Haemoglobin percentage (Figs. 7 and 8). This drug seems to have more useful effect in the therapy of reactions in leprosy of subacute and mild type. It can be administered safely to all cases irrespective of age and sex and is free of contraindications. In subacute and mild type of cases

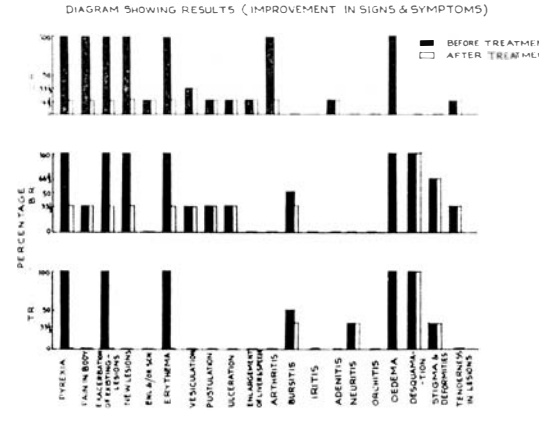

Fig. 7

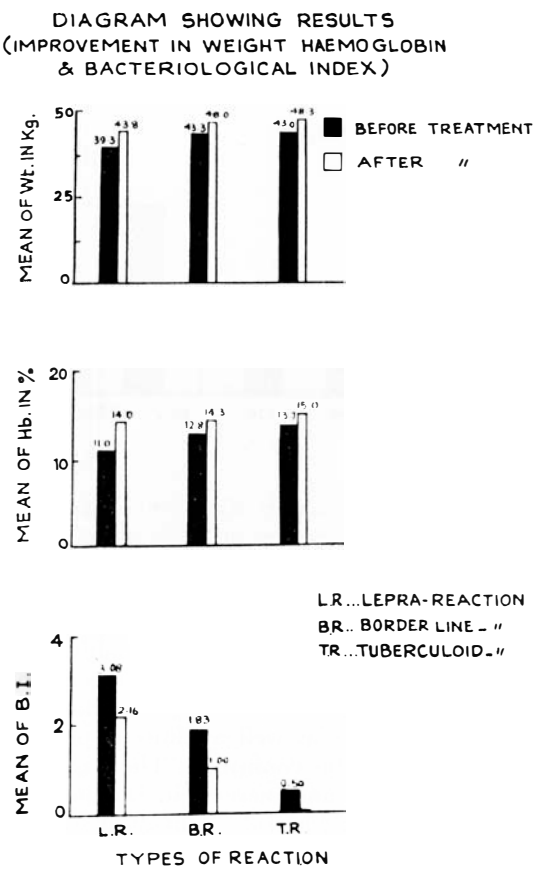

FIG. 8

withdrawal of anti-leprosy treatment is not necessary, but only reduction in dose, will suffice. For acute and severe reactions, of any type, it is necessary to administer other anti-reaction treatment along with this drug, because of slow response to treatment. Clinical pictures of the cases before and after treatment have been shown in Figs. 9 to 14.

In the acute and severe type of reaction, the lesions took twice as much time to subside as fever. In the subacute type also a similar feature was noticed. However the administration of anti-reaction drugs in the treatment of reactions in leprosy is not only sufficient but it is also essential to recognise and note its readily changing form or appearance. Because if they are not recognised and cared for it might lead to a catastrophic end. To elude reaction in leprosy cases, carefully designed and judicious therapy with sulphones is highly essential. It should be induced in a slowly and gradually increasing dose and the same be maintained for a longer period. For management of conditions like acute severe neuritis and palsies, splinting of affected nerve and part with appropriate physiotherapeutic exercises is most essential to ensure rest and prevent stretching of the nerve and paralysed muscles.

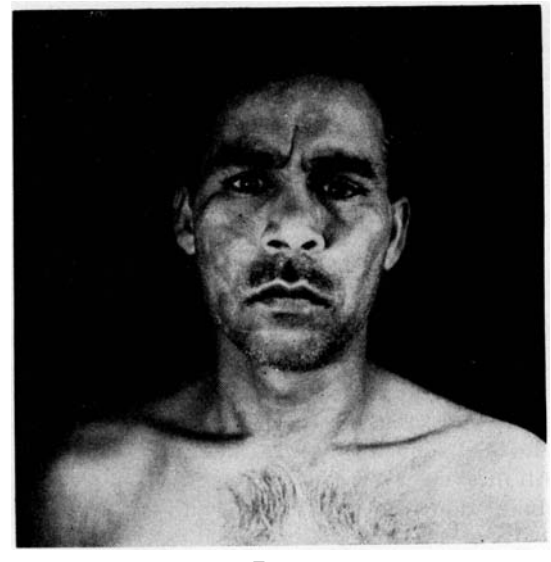

FIG. 9
Before treatment

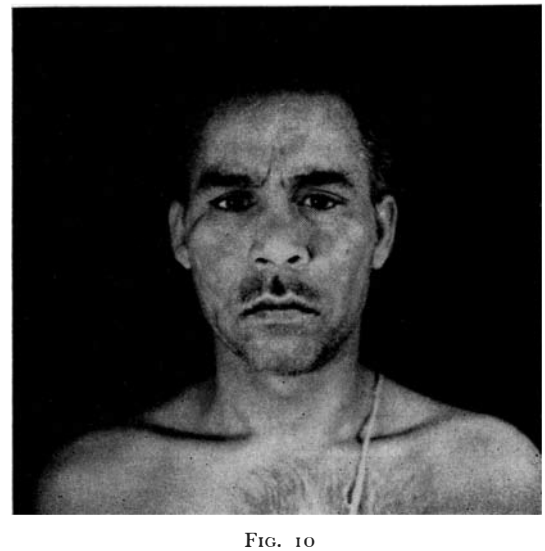

FIG. 10
30 days after treatment

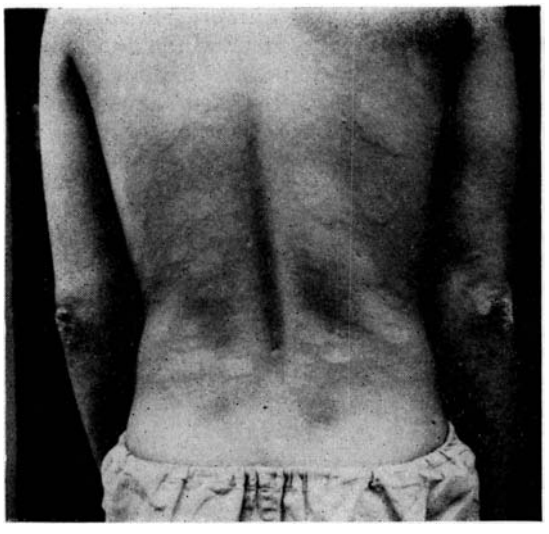

FIG. 11
A case of Lepro-reaction before treatment

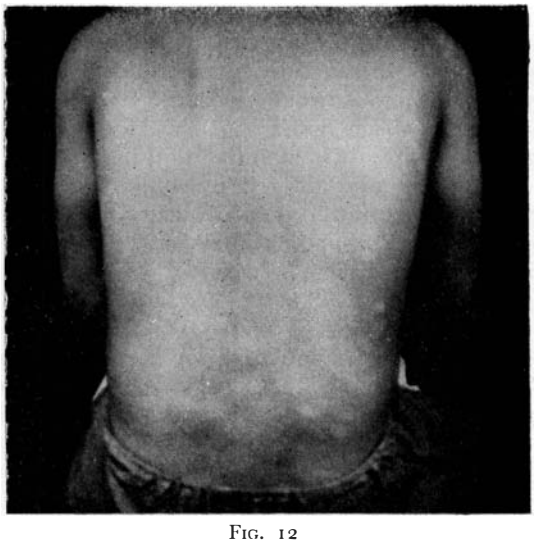

FIG. 12
30 days after treatment

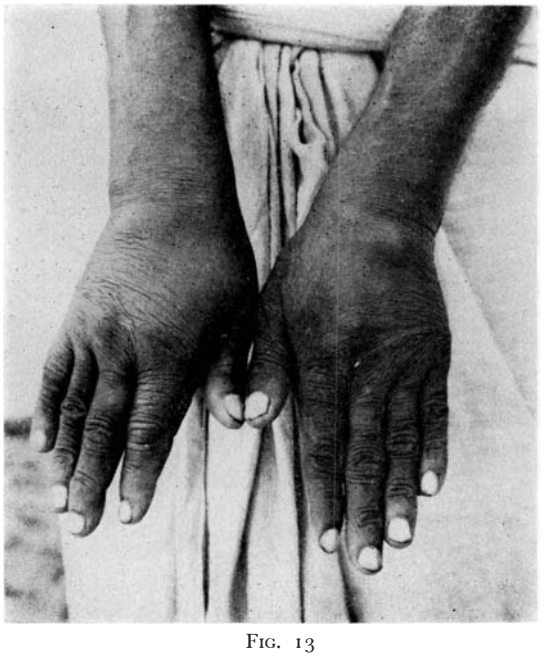

FIG. 13
A case of Border-line reaction before treatment

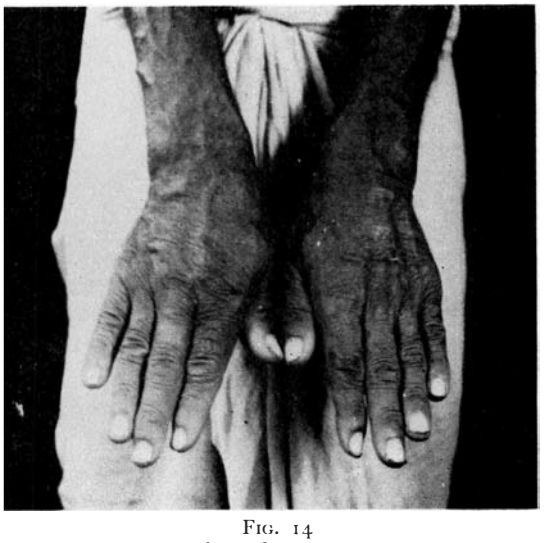

FIG. 14
30 days after treatment

SUMMARY

(1) It is observed that 'Achyaranthes aspera' is effective in the therapy of reactions in leprosy particularly in subacute and mild type.
(2) It has been noted that this drug has not produced any toxic manifestation in any case.
(3) If it is administered in conjunction with sulphone (specific anti-leprosy drug) the chances of reaction become limited and the rate of improvement becomes faster.

ACKNOWLEDGEMENTS

We gratefully acknowledge Dr K. N. Udupa, Superintendent, S. S. Hospital, B.H.U., for permission to utilise the hospital records as well as for his day to day encouragements and to Dr Y. N. Upadhyaya, Head of the department, for providing the necessary facilities and suggestions. We are also thankful to the patients for their willing co-operation throughout the trial.

REFERENCES

1. DHARMENDRA, 'Notes on Leprosy', Published by the Ministry of Health, Government of India; New Delhi, 1960.
2. RAMANUJAM, K., *et al*; 'Treatment of Lepra Reaction and some of its Special Manifestations', *Leprosy in India*, Vol. XXXVI, No. 1 Jan. 1964.
3. TRIPATHI, S. N., *et al*; 'Effect of Achyranthes aspera in the treatment of Leprosy' *Journal of Medical Science*, B.H.U., Vol. IV. No. 2, July 1963.

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The Clinical Dynamics of Pigment Loss

(A Study of 71 lesions in Eastern Nigerians)

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Little attention seems to have been given to the clinical investigation of the development of hypopigmented skin patches in leprosy. This is understandable, since we are naturally concerned to try and diagnose these lesions as early as possible, and once they are diagnosed our interest is focussed on observing their resolution always, with the hope that this will be accelerated by therapy.

How does an area of normally pigmented skin change into a macule? According to Rogers and Muir,[23] macules may appear by lateral spread, or they may already be of considerable size when first noticed. On the other hand Fite,[11] referring particularly to tuberculoid skin plaques, considered that these lesions blossom forth as such with rare exceptions, and do not enlarge laterally to more than a slight degree. Rogers and Muir[24] state that pigmentary loss in leprosy is due to interference with the function of the melanoblasts (the 'melanocytes' of contemporary histologists). It is much more commonly noticed in skins which contain a large amount of pigment. It is also seen typically in those lesions in which the melanin-containing cells of the stratum germinativum are most affected by the pathological process, that is to say, in those of tuberculoid and near tuberculoid conformation. In pure lepromatous lesions where a clear zone is interposed between the infiltrate and the basal layers of the epidermis, it tends to be absent or minimal.

In general, there is a close positive correlation between the location and timing of pigment loss and recovery, and the evolution and resolution of the epithelioid and giant cell granuloma. It was therefore considered that a study of the mode of onset of individual hypopigmented lesions might contribute to a better understanding of their microdynamics.

The functional relationship of pigment loss to the area of a skin patch theoretically involves three possibilities:–

1. Uniform or almost uniform loss of pigment over an increasing area,

2. Increasing loss of pigment over a static or almost static area and

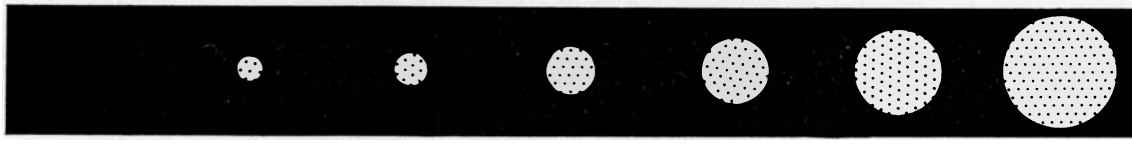
3. Increasing loss of pigment over an increasing area.

An attempt was made, by questioning patients attending clinics and segregation villages in Rivers Province, Eastern Nigeria, to see if macules did in fact develop in these three ways, and if so whether such development could be related to sensation, the type of leprosy, the sex and maturity of the patient and the skin bacteriology.

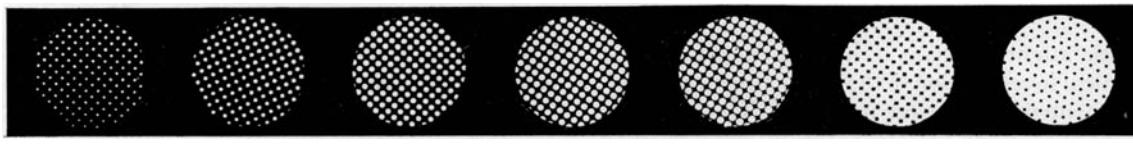
Method

Water-colour diagrams were painted in burnt umber on strips of thick buff-tinted cartridge paper which were then sprayed with a shellac varnish. These were taken on routine tours of outstations.

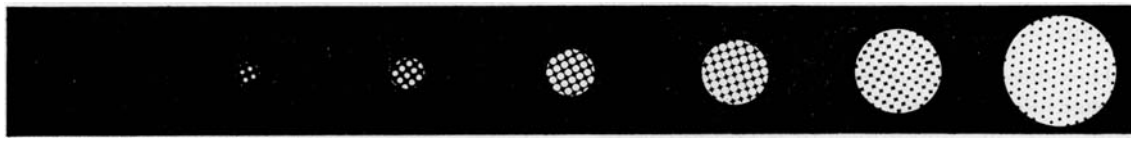
Each strip consisted of seven squares on which were depicted different stages of a developing 'macule'. The expanding macules were shown as six roughly circular areas, the smallest being of 1 cm. diameter, the other ones of diameters increasing by 1 cm. to the largest of 6 cm. diameter. For the macules of almost static area, the first circle was of 5 cm. diameter, the others increasing by 0.2 cm. to the largest, again of 6 cm. diameter. The lesion under study was pointed out to the patient, who was then asked about its development (in the case of very young children, the parent was questioned). He was shown the diagrams in random order, and in his own language it was explained to him that a patch could get bigger while remaining the same colour, as in 1.



or it could remain almost the same size while becoming paler as in 2.



or it could get bigger and become paler at the same time, as in 3.



The patient was asked to try and remember in which category the patch under examination belonged.

In order to avoid confusion, the patients were not questioned about the *history* of sensory changes in the lesions, nor was any attempt made to ascertain their rate of development. For the same reason, the following kinds of lesion were excluded:– (*a*) Markedly raised ones, for example major tuberculoid and 'infiltrated dimorphous'; (*b*) Those showing only slight pigmentary loss, for example some of those included under (*a*) above, and certain 'uncharacteristic' and near lepromatous macules; (*c*) Those that appeared to be regaining their pigment when first seen, either by spontaneous centrifugal spread, or by uniform fading of the lighter colour as a result of sulphone treatment or by both methods; (*d*) Those of less than 5 cm. diameter; (*e*) Those on parts of the body not directly visible to the patient, unless he or she lived with a close relative who had observed the progress of the lesion and was available for questioning.

Macules showing some erythema and lack of definition of edge, were not excluded, provided that they were of sufficient size – see (*d*) above – and pallor to be easily seen. These macules, having some of the appearances of tuberculoid and some of lepromatous lesions, are referred to in the Table under 'Classification' as dimorphous (= D).

In practice, this left seventy-one patients with flat or almost flat hypopigmented macules for assessment. With the method of assisted history taking, no patient questioned had to be excluded from the study because of inability to remember how the macule under examination had developed.

Results

These are shown in the Table. It will be seen that of the 11 lesions classified as dimorphous, 10 occurred in males and only 1 in a female, whereas of the tuberculoid lesions, 24 occurred in males and 36 in females. In this series of patients selected for the factor of pigment loss, the preponderance of males among those whose tissues show 'lepromatoid' behaviour, and of females among the pure tuberculoid types, is in accord with the findings of Kinnear Brown.[4] He found that in Uganda, although the incidence of the disease was very similar in the two sexes, significantly more males suffered from lepromatous leprosy, and more females had tuberculoid or non-lepromatous leprosy. In the present series,

TABLE

Serial No.	*Clinic*	*Classification*	*Maturity*	*Sex*	*Onset type*	*Smear*	*Anaesthesia*
1	Angiama	Dimorphous	Adult	Male	2	—	0
2	,,	D	A	M	2	—	0
3	,,	D	A	M	1	—	2
4	,,	D	A	M	1	E+Sk+	0
5	Degema	D	A	M	2	—	0
6	Edeoha	D	A	M	2	E+— Sk++	0
7	Gokana	D	A	M	3	+—	0
8	,,	D	A	M	3	+	2
9	,,	D	A	M	2	+	0
10	Angiama	D	Child	M	1	—	2
11	Umuagbai	D	A	Female	3	E++ Sk+—	0
12	Angiama	Tuberculoid	A	M	2	—	0
13	,,	T	A	M	3	—	2
14	,,	T	A	M	3		2
15	,,	T	A	M	1	—	0
16	Anyama	T	A	M	1	—	0
17	,,	T	A	M	3	—	2
18	Degema	T	A	M	2		2
19	,,	T	A	M	3	—	1
20	Edeoha	T	A	M	1	—	2
21	,,	T	A	M	1	—	2
22	,,	T	A	M	3	—	0
23	Ekowe	T	A	M	3	—	0
24	Gokana	T	A	M	3	—	2
25	Isoba	T	A	M	1	—	2
26	,,	T	A	M	2	—	2
27	Okodogu	T	A	M	2	—	0
28	Okwuzi	T	A	M	1	—	1
29	Sabagreia	T	A	M	2	—	0
30	Ubetta	T	A	M	1		2
31	Umuagbai	T	A	M	3	—	2
32	Anyama	T	C	M	2	—	2
33	Degema	T	C	M	2	—	2
34	Ekowe	T	C	M	1	—	0
35	Tabangh	T	C	M	3	—	2
36	Amassoma	T	A	F	3	—	0
37	,,	T	A	F	3	—	1
38	,,	T	A	F	3	—	0
39	,,	T	A	F	3	—	2
40	Angiama	T	A	F	2	—	0
41	,,	T	A	F	3	—	1
42	,,	T	A	F	1	—	0
43	,,	T	A	F	3	—	0
44	,,	T	A	F	1	+—	0
45	,,	T	A	F	3	—	1
46	,,	T	A	F	2	—	
47	,,	T	A	F	1	—	2
48	,,	T	A	F	1	—	2
49	,,	T	A	F	2	—	0
50	,,	T	A	F	1	—	2
51	,,	T	A	F	2	—	2
52	Anyama	T	A	F	2	—	2
53	Degema	T	A	F	3	—	0
54	,,	T	A	F	1	—	0

Serial No.	*Clinic*	*Classification*	*Maturity*	*Sex*	*Onset type*	*Smear*	*Anaesthesia*
55	Edeoha	T	A	F	1	—	0
56	,,	T	A	F	1	—	2
57	Eleme	T	A	F	3	—	1
58	Okwuzi	T	A	F	2	—	1
59	Omoku	T	A	F	3	—	2
60	,,	T	A	F	1	—	2
61	Sabagreia	T	A	F	3	—	1
62	Ulakwo	T	A	F	2	—	0
63	Umuagbai	T	A	F	2	—	2
64	,,	T	A	F	3	—	0
65	Wari-bogoama	T	A	F	1	—	0
66	Amassoma	T	C	F	3	—	0
67	Angiama	T	C	F	2	—	2
68	Anyama	T	C	F	3	—	2
69	,,	T	C	F	3	—	1
70	Okwuzi	T	C	F	1	—	1
71	Wari-bogoama	T	C	F	3		2

E=ears. Sk= other involved skin.

22 macules were of onset type 1., 21 of type 2. and 28 of type 3.

No statistically significant correlations were found between the onset type and any of the other features studied; but it is clear that macules having similar clinical appearance showed differences in development, recognised by the patients, which would be missed by the clinician unless specifically sought.

DISCUSSION

The underlying cause of pigment loss in leprosy has remained obscure. If it is associated with lepra bacilli at all, it must be only indirectly so, since there is very nearly an inverse relationship between the numbers of bacilli found in the dermis and the degree of hypopigmentation.

Certainly it is associated with an epithelioid and giant cell – or with a rather less specific – type of granuloma, involving the deeper layers of the epidermis. This granuloma, in its turn is assumed to be associated at some time in its evolution, with the presence of lepra bacilli. Neither pigment loss nor nerve damage seems, however, to be a feature of the cytologically tuberculoid, positive response to lepromin.

In lepromin testing, heated bacillary antigen is used, and here it seems reasonable to infer a true cause and effect relationship, the bacilli being destroyed by the granuloma elicited by them, but remaining largely intact in its absence.[10] With naturally occurring hypopigmented lesions, no such inference is possible. In the present study, the way that macules of type 2., appear, suggests an underlying reservoir of the depigmenting factor becoming effective over a wide area at one time. That there is *some* association of this factor with lepra bacilli is indicated by the fact that when found, these tend to be located near the periphery of hypopigmented lesions[15] and may be seen in cells not essentially different from those found in lepromatous tissue.[11] It is possible therefore, that the association is with a deeper, preceding, rarified, regressing patch of leproma, whose expansion has stopped or almost stopped. It is difficult however, to see how this concept *alone* could account for the expansion of macules of type 1., and particularly of type 3., in which pigment loss evidently increases directly with the increase in the skin area affected by the granuloma, almost certainly, in most cases, in the absence of bacilli.

Could it be that hypopigmentation is caused by the destruction of bacilli by the human host, or by their spontaneous disintegration? Its absence from positive lepromin responses and from resolving lepromata make either of these possibilities unlikely.

One way to account for the present findings would be to suppose that in type 2., macules a submicroscopic self-replicating agent has invaded

the germinative layer as a large sheet of particles, whereas in macules of types 1., and 3., the agent is spreading centrifugally from cell to cell. Melanocytes, we are told[12] are very numerous in the epidermis and are supplied with a complex system of dendrites that intertwine with, and end on epidermal cells, and supply them with pigment. They are believed to be of neuro-ectodermal origin, being derived from cells of the embryonic neural crest. They would make an admirable habitat for any sufficiently small living agent proliferating by cell to cell passage. In fact, it is known that many viruses are able to live in cells of epithelia, especially those of the skin, and a viral aetiology has long been suspected in leprosy.[16]

The observation of phage-like particles and 'ghost bodies' by Richards and Wade,[22] and in vitro comparison of the effects of sunlight on heated and unheated lepra bacilli,[6] suggested that each bacillus was being affected by a much smaller organism, which, it was postulated, was variably pathogenic to certain tissues of the human host. (It is of interest that intrabacillary ovoid particles of about 30μ diameter, apparently arising from the cell wall of a lepra bacillus, have been seen under the electron microscope.[14] In appearance these particles might be taken for virus bodies.). Examination of mixed lesions[7] pointed to certain aspects of the behaviour of the proposed organism, but it was not at that time thought that it could have an extrabacillary reproductive existence, nor that it could affect skin pigmentation. Recent work, however, appears to support this idea.

In view of the apparently minor direct pathogenicity of lepra bacilli, it seems likely that an immune mechanism could underlie many leprosy lesions, and indeed several workers [2 3 20] have shown serological similarities between leprosy and the so-called auto-immune diseases. There are some however, who consider that these diseases themselves may have an underlying infective aetiology. Thus Pease[21] considers that auto-immune diseases may be caused by sub-cellular particles of microbial origin which by a process of degeneration become incapable of independent existence, but which are probably capable of acting as episomes and invading the genetic processes of the mammalian host's cells so that these themselves produce foreign antigens.

The fact that bacteriophages, which in any case are widely believed to have a bacterial origin, may also play a part in disease, has lately received some attention.[13 17 18 19] Mankiewicz and van Walbeek[17] were able to produce lysis of a parent strain of tubercle bacillus H37Rv, by a mutant strain which had been repeatedly exposed to mycobacteriophage DW. The appearances they noted seem to be remarkably similar to those described by Richard and Wade,[22] and stained preparations of their lysogenic strain, looking like chains of streptococci bear close resemblance to many of the 'granular forms' of lepra bacilli familiar to every leprologist. The lysogenic bacteria did not elicit tuberculin reactions in guinea pigs. Mankiewicz and Béland[18] have been able to induce 'sarcoid like' lesions in guinea pigs infected simultaneously with mycobacteriophage DS6A and tubercle bacilli, and their findings in such animals also treated with cortisone could serve as a model for the study of lepra reactions in man.

Recently, Mankiewicz[19] has been able to demonstrate in vitro pathogenicity of bacteriophages that lyse mycobacteria and corynebacteria, for mammalian cells.

A further point of interest concerns the thiosemicarbazones. These, according to Dugeon[9] provide a link in the development of bacterial and viral chemotherapeutic agents. It is probably no coincidence that different members of this group of compounds have been found effective in the treatment of leprosy[8] and in the prophylaxis of smallpox.[1] The well known association between vaccinia and lepra reaction[5 25] is at least suggestive of a common defence mechanism against biologically related causes.

We cannot conclude without wondering whether *Mycobacterium leprae* as a species really exists. Are we perhaps seeing an infected mycobacterium and the relationships of the infecting organism with the cells of the human host, as well as with those of the microbial host?

If so, might it be possible to construct 'lepra bacilli' de novo, by infecting cultivable mycobacteria with phages or mammalian viruses? Whatever the answers to these questions – and to others – the accumulation of knowledge of the ecology of *very* tiny creatures in their natural micro-environments, must lend added impetus to

the alteration of much of the terminology of 'leprosy', already begun on humanitarian grounds.

SUMMARY

The development of 71 hypopigmented flat skin lesions in Nigerian patients, was studied.

The patients were able to recognise three developmental types of lesion, which, on clinical examination, showed no statistically significant affinities in relation to the other features found. It is believed that in skins sensitive to it, pigmentary loss is brought about by extrabacillary proliferation of a subcellular, self-replicating agent initially carried by mycobacteria, rather than by mycobacteria themselves.

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I am also most grateful to my colleague Mr. J. Bryant, a student of mathematics in the University of Durham, for his analysis of the figures; and to the Leprosy Inspectors and patients of Rivers Province, Eastern Nigeria, for their co-operation.

REFERENCES

1. BAUER, D. J., ST. VINCENT, L., KEMPE, C. H. and DOWNIE, A.W. (1963. Prophylactic treatment of smallpox contacts with N-methylisatin B—thiosemicarbazone. *Lancet*, **2**, p. 494.

2. BONOMO, L., DAMMACCO, F. PINTO, L. and BARBIERI, G. (1963). Thyroglobulin antibodies in leprosy. *Lancet*, **2**, pp. 807—809.

3. BONOMO, L., TURSI, A., TRIMIGLIOZZI, G. and DAMMACCO, F. (1965).L.E. cells and antinuclear factors in leprosy. *Brit. Med. J.*, **2**, pp. 689–690.

4. BROWN, J. A. KINNEAR, (1959). Factors influencing the transmission of leprosy. *Trans. R. Soc. trop. Med. Hyg.* **53**, p. 179.

5. BROWNE, S. G. and DAVIS, E. M. (1962). Reaction in leprosy precipitated by smallpox vaccination. *Leprosy Rev.* **33**, 4, pp. 252–254.

6. CORCOS, M. G. (1953). Human lepra bacilli exposed to sunlight will retain their acid fastness if they are first heated. *Leprosy Rev.* **24**, 3, pp. 67–70.

7. CORCOS, M. G. (1964) Mycobacterium leprae: a house divided? *Leprosy Rev.* **35**, 3, pp. 61–77.

8. DAVEY, T. F. (1964). *Leprosy in Theory and Practice*, Ed. Cochrane, R. G. and Davey, T. F. John Wright & Sons. Ltd., Bristol. pp. 395–396.

9. DUDGEON, J. A. (1965). Advances in the treatment of virus diseases. *Practitioner*, **195**, 1168, p. 503.

10 FERNANDEZ, J. M. M. (1954). Bacteriologia de la lepra. Investigaciones para dilucidar si el Mycobacterium leprae esta muerto o vivo. Valor del metodo de Ziehl Neelsen. *Med. Panamericana*, **3**. abstr. in *Int. J. Leprosy* (1962), **30**, 4, pp. 504–505.

11. FITE, G. L. (1951). The pathology and pathogenesis of leprosy. *Ann. N.Y. Acad. Sci.* **54**, 1, p. 28.

12. HAM, A. W. and LEESON, T. S. (1965). *Histology*. Pitman Medical Publishing Co. Ltd., p. 633.

13. HAYES, W. (1964). *The Genetics of Bacteria and Their Viruses*. Blackwell Scientific Publications, Oxford, p. 550.

14. IMAEDA, T. (1962) cited by WADE, H. W. in Undiscussed problems of the bacteriology of tuberculoid and borderline leprosy. *Int. J. Leprosy*, **30**, 4, p. 493.

15. KHANOLKAR, V. R. (1964). Pathology of leprosy. *Leprosy in Theory and Practice*. Ed. Cochrane, R. G. and Davey, T. F., John Wright & Sons Ltd., Bristol, p. 139.

16. MANALANG, C. (1931). Significance of findings in biopsy materials from lepers. *Philippine Health Service No. Bull.* **11**, pp. 633–638, cited by WADE, H. W. in "L" bodies or protoplasts of the leprosy bacillus? (1962). *Int. J. Leprosy*, **30**, 4, pp. 501–503.

17. MANKIEWICZ, E. and van WALBEEK, M. (1962). Mycobacteriophages. Their role in tuberculosis and sarcoidosis. *Arch. Env. Hlth.* (Amer. Med. Assn.), **5**, pp. 122–128.

18. MANKIEWICZ, E. and BÉLAND, J. (1964). The role of mycobacteriophages and of cortisone in experimental tuberculosis and sarcoidosis. *Amer. Rev. Resp. Dis.*, **89**, 5, pp. 707–720.

19. MANKIEWICZ, E. (1965). Bacteriophages that lyse mycobacteria and corynebacteria, and show cytopathogenic effect on tissue cultures of renal cells of "Cercopithecus aethiops". Canad. Med. Assn. J., **92**, pp. 31–33.

20. MATTHEWS, L. J. and TRAUTMAN, J. R. (1965). Clinical and serological profiles in leprosy. *Lancet*, **2**, pp. 915–918.

21. PEASE, P. E. (1965). *L-Forms, Episomes and Auto-immune Disease*. E. & S. Livingstone Ltd., Edinburgh and London, p. 36.

22. R.CHARDS, O. W. and WADE, H. W. (1949). Application of phase microscopy to the examination of the leprosy bacillus. *Memoria del 5 Congr. Int. de la Lepra*, 1948, La Habana, Cuba, p. 519.

23. ROGERS, L. and MUIR, E. (1946). *Leprosy*, Bristol: John Wright & Sons Ltd., p. 172.

24. ibid. p. 179.

25. ibid. p. 87.

Lep. Rev. (1966) **37,** 2, 127/128

Abstracts

1. **BGG Vaccination of Children against Leprosy: First Results of a Trial in Uganda,** by J. A. KINNEAR BROWN, and M. M. STONE, with an appendix by DR IAN SUTHERLAND. *Brit. Med. Journ.* 1966, **1**, 7–13.

A controlled trial of BCG vaccine in the prevention of leprosy started in the Teso district of Eastern Uganda in September 1960. By September 1962, 17,397 children, more than 80% of whom were aged under 10 years, had been included. All were relatives or contacts of known leprosy patients.

Those with negative reactions to an initial Heaf tuberculin test, or with Grade I or Grade II positive reactions, were allocated alternately to an unvaccinated group (8152 children) and a BCG-vaccinated group (8149 children). Those with Grade III or Grade IV reactions (1096 children) were all left unvaccinated, as were children who already had skin lesions due to leprosy.

In the course of the first follow-up of all the participants, between May 1963 and May 1964, more than 94% of the children were seen and examined for leprosy. A total of 116 cases of leprosy had developed during this period of about two years since intake. The incidence in the unvaccinated children was 11·0 per 1000, and in the vaccinated children 2·2 per 1000, which is one-fifth of the incidence in the absence of vaccination. Among those positive in Grade III or IV and left unvaccinated the incidence was 8.3 per 1000. The reduction in leprosy incidence in the vaccinated group did not appear to depend on the grade of initial tuberculin sensitivity (negative, Grade I or Grade II) nor on the age of the child at intake.

The results so far establish that BCG vaccination has conferred substantial protection against early forms of leprosy in Uganda for a period of about two years. Since these early forms may resolve spontaneously, it is of particular inportance to follow the trial population for some years to see how these lesions evolve, as well as to study the further incidence of the disease. About 8% of leprosy patients in Eastern Uganda have lepromatous leprosy, and it would be unwise to conclude that the present results will necessarily apply in other parts of the world, where the proportion of patients with lepromatous leprosy may be as high as 70%.

The Appendix by Dr IAN SUTHERLAND reports as follows:

By the end of 1961 information on the prevalence of leprosy at intake had been obtained from a total of 4542 child relatives and contacts of leprosy cases. (Children examined in the eitelas in which the intake appeared to have been loaded with unrelated patients were not included in this total). The information was used in the following manner to estimate the expected incidence of leprosy in the course of the trial.

Table A gives the findings in these 4542 children according to age, and shows the general tendency for the prevalence to rise until the age of 15. A straight line was 'fitted' to the prevalence rates to describe their upward trend, the standard statistical technique of weighted regression being used; this straight line provided an adequate fit to the data and there was no indication of curvature. The figures were therefore consistent with a steady increase in the prevalence rate of leprosy from zero at birth to a figure of over 60 per 1000 at the age of 15 years, the increase amounting to 4·62 per 1000 for each year of age.

TABLE A

Prevalence of Leprosy among 4542 Children at Intake Examination, according to Age

		Cases of Leprosy	
Age (Years)	*Total examined*	*No.*	*Prevalence per 1000*
0	633	0	0
2	810	2	2
4	934	13	14
6	749	16	21
8	546	30	55
10	345	17	49
12	309	13	42
14	165	10	61
16 or more	51	2	39
All ages	4542	103	23

If it is assumed that the population of child contacts is 'stationary'—that is, not changing in total size or age distribution—and that the risk that they will contract leprosy is also not changing, then the increase in the *prevalence* of leprosy from one year of age to the next may be taken as a minimum estimate of the annual *incidence* of the disease. (The true annual incidence of leprosy is likely to exceed this estimate, since a proportion of the cases, having developed, will from past experience resolve spontaneously and so not contribute to the prevalence at the next year of age). Subject to those assumptions, therefore, the estimated annual incidence of leprosy is this population of child contacts, in the absence of vaccination, was 4·62 per 1000 children per year.

At the time when this estimate was made a total of 4263 children (all in Grade 0, 1, or 2 initially) had been admitted to the unvaccinated group. According to the estimate 98 cases of leprosy would be expected to develop among them during a period of follow-up of five years. It was not known whether the population of child contacts was stationary, but it was suspected that, as a result of the treatment of known cases, the risk of contracting leprosy might be falling. In addition it was uncertain how effectively the participants could be traced for follow-up examinations, and whether it would be possible to continue these for as long as five years. In the circumstances it was safer to assume that perhaps only about half of the above total of 98 cases would develop and be detected in the course of the trial. If, say, 50 cases were found in the unvaccinated group, then it would not be permissible to

claim a clear benefit from BCG vaccination unless 25 or fewer cases were found in a randomly allocated vaccinated group of equal size (any lesser difference could be attributed to chance). Since it was important to be able to detect a reduction in leprosy incidence due to BCG vaccination, if it occurred, which was less than 50%, it was decided to increase the number of participants in the trial, and the intake was continued.

The final total of children in the unvaccinated group (Grade 0–II) was 8152. According to the same estimate of incidence a total of 75 cases of leprosy would have been expected to develop among these children during an average period of follow-up of two years. This may be compared with the total of 77 cases actually found in this group during the period of the first follow-up (the other 12 of the 89 cases shown in Tables IV and V had suspicious lesions which were not confirmed as leprosy until the second follow-up). Because of the various uncertainties and assumptions associated with the incidence estimate, it is clear that the agreement with reality is very much closer than could reasonably have been expected. It suggests, however, that the approach described in this appendix was not unrealistic, and represented a useful method of assessing when the intake to a trial of a prophylactic measure for a chronic disease was sufficiently large, from information obtained in the course of the intake. The approach has been placed on record in the hope that it may be useful to other workers in similar circumstances.

2. **Leprosy and Heredity** by AMADO SAUL and MANUEL DIAZ, *Dermatologia*, June 1965, Vol. 9, No. 2, pp. 157–169.

The authors presented a study in heredity in leprosy at the 2nd Mexican Congress of Dermatology held at Guadalajara, Jalisco, Mexico, April 1963. They said that leprosy is infectious and transmissible but is not considered a hereditary disease. Its attack index is very low and it is essentially familial with intra-domiciliary acquisition. A hereditary factor exists which determines whether a person, living under conditions favourable to infection, will or will not acquire the disease, and if he does, which type will develop. It is thought that an irregularly dominant factor P exists which neutralizes natural resistance and leads to the acquisition of lepromatous leprosy whereas it may be said that a person exposed to the infection, in whose family there have been no previous cases of the disease, will either show a positive Mitsuda or acquire the tuberculoid form.

The authors studied 1000 subjects, family relations in 10, and the problem of conjugal leprosy. The results seem to confirm the hereditary factor but further observations are essential.

3. **The epidermic melanocyte, tactile neurone** by A. R. AMORETTI. *Dermatologia*, June 1963, **9**, 2, pp. 197–209.

The author presents the hypothesis that the epidermal melanocyte is the prime tactile neurone. Denuded skin lacks tactile sensation and the tactile sensibility of depigmented areas is proportionate to the abundance of melanocytes. The melanocyte is the tactile cell of Merck composed of dendrites ready for stimuli. Its cellular body elaborates the stimuli and the efferent axon transmits the sensation to the second neurone which then crosses the spinal ganglia to penetrate into the posterior roots of the medulla. The temperature and pain sensations are subepidermal. In the first phases of neuritis of leprosy alterations of pain and temperature sensibilities are observed but not tactile changes. It seems that during these early phases the efferent fibres are less vulnerable than the afferent (dendrites of temperature and pain).

4. **Maintenance of cytopathic activity of Mycobacterium leprae in Eagle's medium supplemented by Mycobacterial extracts**, by A. L. OLITZKI and ZIPPORA GERSHON of the Department of Clinical Microbiology, Hadassah University Hospital and Department of Bacteriology, Hebrew University Hadassah Medical School, Jerusalem, a preliminary communication in *Israel J. of Med. Sci.* **1**, (5), 1965.

The authors point out HANK's explanation of the failure to obtain growth *in vitro* of *Mycobacterium leprae* as being due to its inability to obtain energy from the carbon sources ordinarily used. Failure has followed attempts at culture *in vitro* in human and simian cultures, and in cell-free media under symbiotic conditions with other micro organisms and an anaerobic yeast-glycerol medium. The authors tried to maintain the viability of *M. leprae* on a cell-free medium enriched by the products of saphrophytic mycobacteria (as was done in cultures of Johne's bacillus) and achieved some success with an extract of one mycobacterial strain, and succeeded in preserving the biological activity of *M. leprae* for a period of at least 5 months.

The separation of *M. leprae* from the host tissues was achieved as follows:

A nodule of 500 mg. was taken from an untreated patient, triturated with glass powder in a mortar and suspended in 10 ml. of phosphate-saline solution of pH 7.2. The cell debris was removed by centrifugation at 1000 r.p.m. for 5 min. and the bacteria were collected from the supernatant fluid by refrigerated centrifugation at 10,000 r.p.m. for 20 min. This procedure was repeated and the resultant final suspension contained acid-fast bacilli which failed to grow on Loewenstein medium.

The extracts of a typical mycobacteria were prepared as follows: three strains of atypical mycobacteria were transferred to fresh Loewenstein medium and harvested after a suitable period of incubation in 5 ml. of saline/slope. After a treatment for 3 min. in MSE-ultrasonic power unit at 150 v. the bacterial residues were removed by 2 subsequent filtrations through 3 Sl Seitz filters. The filtrates were aoutclaved and tested for sterility on Loewenstein medium.

The authors give 2 Tables and 2 illustrative figures and 2 summaries, 1 in French and 1 in Spanish' It would be useful to translate these.

'The biological activities of *Mycobacterium leprae* have been observed, and used as indicators of their viability *in vitro* (1) The production of a cytopathic substance which acts on the cultures of murine monocytes. (2) The production of an increasing turbidity in the Eagle medium enriched by a sonic extract of a saphrophytic mycobacterium.

After 4 consecutive passages in this medium, which extended over a period of 5 months after the separation of the bacteria from the tissues of the host, the biological activities still persist.

Note. The reference to murine monocyte cultures is referred to in Fig. 2).

Letters to the Editor

28th December, 1965

Dear Sir,

A recent paper by Tran-Van-Bang and Nguyen-Huan-Truong from the Institut Pasteur de Viet-Nam entitled 'Le Lepreuc Est Un Malade Mental' published in the Bulletin De La Societe De Pathologie Exotique, Volume 57 (Nov. – Dec.) 1964, page 1200 states that in their experience 'mental depression is frequent and can lead to suicide.' The article gives no figures but hints that suicide is not infrequent.

As this statement is not in tune with our recent experience we have studied the number of suicides occurring in the Sungei Buloh Leprosarium over the past 23 years and have found that although 17 people committed suicide during that time nobody has done so since 1954. It seems to me that this finding is not without interest and does perhaps reflect the generally higher optimism that has prevailed about the treatment of patients with leprosy. I wonder whether a similar change for the better has been noted in other leprosaria.

J. H. S. Pettit, M.D., M.R.C.P.

January 11th, 1966

To the Editor:

My respected friend and mentor, Dr Robert Cochrane, in a highly respectable effort to prevent the unwary clinician from taking a positive nasal mucosal smear for acid fast bacilli too seriously, has accomplished just the opposite by saying (LR 36:196) 'taking a nasal smear does not *necessarily* establish a diagnosis of leprosy.' The italics are mine. The inference could well be that it only establishes a diagnosis of leprosy if it is positive! The fact is that it *never* establishes a diagnosis of leprosy – and, further, that a nasal smear should never even be taken with this purpose in mind, since it cannot ever confirm or exclude the diagnosis. It is dangerous to so use it even in the most expert hands; it is inexcusable for the inexpert to use it. I know Dr Cochrane believes this. It's only that he didn't say it!

Harry L. Arnold, Jr., M.D.

15th January, 1966

Dear Dr Ross Innes,

In *Leprosy Review* No. 4, 1965, you published a letter from Dr F. Contreras, in which he discusses the advisability and the legitimacy of experiments on healthy persons by attempting to infect them with leprosy. The writer, subscribing to the opinion which I put forward in *Leprosy Review* No. 1, 1964, categorically rejects the idea of such experiments. He thinks, however, that I misunderstood the substance of the article by him and Gay Prieto in Mem. Congreso Int. Leprol. (Madrid, 1953), inaccurately translating the text.

In this connection, I quote an extract from the editorial 'Twenty Years of Medical Activity in the Fight Against Leprosy (1948–1959)', published in the '*WHO Chronicle*', vol. 14, no. 1, 1960, p. 8 and written, as may be seen from the footnote, with the assistance and concurrence of Dr Gay Prieto (the first author of the article which provoked the discussion):'. . . It is illustrated by the exceptional clinical observations made by Gay Prieto and Contreras on a young man of 26 years, who for 14 months was inoculated with the blood of patients with strong lepra reactions, with the mucus from lepromatous patients laden with bacilli, and with biopsy fragments. After 8 years of observation he had not developed the slightest suspicion of leprosy.'

V. Loginov

Lep. Rev. (1966) **37**, 2, 131/132

Report on Leprosy Work for the Year 1964 at Ndanda Leprosarium, Tanzania.

In-patients Only

GENERAL INFORMATION

Remaining patients on December 31st 1963:		572
	Discharges:	130
		442
	Deaths:	4
		438
	Absconded:	49
		389
	Not returned from leave	27
		362
	New admittances:	231
		593
	Re-admittances:	35
Remaining patients on December 31st 1964:		628

On December 31st, 1964 there were 56 more patients resident than on the same date the previous year – the highest number of in-patients ever.

CLINICAL CLASSIFICATION

	Men	*Women*	*Children*	*Total*	
Lepromatous	205	63	31	299	
Tuberculoid	130	91	108	329	628

CLASSIFICATION ACCORDING TO TREATMENT

	Men	*Women*	*Children*	*Total*	
Sulphones	313	133	138	584	
Other drugs	22	21	1	44	628

Of the 628 in-patients 584 are being treated with the SULPHONES.

SEX	Men	335	
	Women	154	
	Children	139	
	Total		628
DEATHS	due to disease		0
	due to other causes		4
BIRTHS	male children	7	
	female children	5	
	Total		12

CLASSIFICATION ACCORDING TO TREATMENT

		Men	*Women*	*Children*	*Total*
Sulphetrone inj.	*L*	171	43	25	239
	T	11	5	12	28
DDS	*L*	14	9	4	27
	T	112	73	95	280
Hydnosulphon	*L*	4	1	2	7
	T	0	2	0	2
UCB	*L*	0	0	0	0
	T	1	0	0	1
Isoniasid	*L*	3	2	0	5
	T	0	4	0	4
Conteben	*L*	1	0	0	1
	T	1	0	1	2
Hydnocarpus oil	*L*	12	8	0	20
	T	5	7	0	12
		335	154	139	628

CLASSIFICATION OF DISCHARGES ACCORDING TO TREATMENT

		Men	*Women*	*Children*	*Total*
Sulphetrone inj.	*L*	32	6	0	38
	T	3	1	2	6
DDS	*L*	4	1	0	5
	T	33	22	18	73
Other drugs	*L*	1	1	0	2
	T	5	1	0	6
		78	32	20	130

DISCHARGES ACCORDING TO RESULT

		Symptom free	*Arrested*	*Improved*	*Total*
Men	*L*	6	14	12	32
	T	29	10	7	36
Women	*L*	1	4	3	8
	T	12	8	4	24
Children	*L*	1	1	0	2
	T	16	2	0	18
		65	39	26	130

Of the 26 patients discharged as IMPROVED 12 actually were transferred to Clinics for further treatment.

DEATHS: There were four deaths only – none due to leprosy.

27–1–64	1 woman	*L*	about 45 years	general condition, heart failure
1–2–64	1 man	*L*	about 25 years	post operative ileus
20–2–64	1 man	*L*	about 60 years	nephritis
Sept. '64	1 boy	*T*	about 10 years	snakebite – during holidays at home

REACTIONS AND DRUG INTOLERANCE: About 4% of the patients are sensitive to the sulphones or cannot tolerate the standard drugs at all. A total of 68 patients had to be hospitalized – some for long periods – due to severe reactions.

34	were	lepromatous	treated	with	sulphetrone injections.
6	,,	,,	,,	,,	DDS.
2	,,	,,	,,	,,	other drugs.
3	,,				new patients.
5	,,	tuberculoid	,,	,,	Sulphetrone injections.
14	,,	,,	,,	,,	DDS.
3	,,	,,	,,	,,	other drugs.
1		,,			new patient.

10 patients had two relapses.

8 patients had three relapses.

There was no severe case of drug dermatitis.

OPERATIONS: Dr Welfare, the Medical Officer of the Leprosarium performed the following operations at Ndanda General Hospital:

Hernia inguinales	3
Hydrocele	4
Neurolysis	5
Amputation of leg	2
Amputation of breasts	1
Ileus	2
Extirpation of metatarsals	11
Extirpation of glands	2
Skin transplantations	2

Besides the above operations very numerous minor operations were done at the small Theatre in the Leprosarium.

GENERAL HEALTH: The general health of the patients has been good. A total of 157 patients had to be hospitalized for longer or shorter periods following either major or minor operations, incisions of ulcers, sequestrotomies, etc. Also because of malaria, dysentery, diarrhoea, bronchitis, etc. etc. 11 patients were so debilitated that they had to be hospitalized at once. We had two slight outbreaks of chickenpox – 1 case in March and 2 cases in October. 8 patients developed measles and one patient who had been inoculated against smallpox last year developed smallpox. We have good facilities for isolating these cases and due to this probably the diseases could be prevented from spreading.

RE-ADMITTANCES: The number of re-admittances was high, 35 in all of whom 30 were men and only 5 women – no children were re-admitted. 24 men and 6 women were lepromatous and 2 men and 3 women tuberculoid cases. ALL of them had had treatment with the SULPHONES previously.

8 patients had been discharged as symptom free.
3 ,, ,, ,, ,, ,, arrested.
7 ,, ,, ,, ,, ,, improved.

The remaining 17 patients had either absconded or had not returned from leave. All of them came back in poor condition, leprosy being very acute.

Book Review

WHO Expert Committee on Leprosy, Third Report.
WORLD HEALTH ORGANIZATION: *Technicsl Report Series*, 1966, No. 319; 31 pages. Price: 3/6. $0.60. Sw.f. 2.--. Also Published in French and Spanish. Available through H.M. Publishing Office, Oxford St, London.

This valuable report contains 29 pages and should be in every leprologists' hands.

This report is divided into two sections: the first on leprosy control, the second on research. The first part is a review of present knowledge on the epidemiology and chemotherapy of leprosy and sets out the diagnosis and the classification to be used in field projects. In order to overcome present difficulties, it recommends that a system of priorities should be adopted. In countries with limited budgets, all means available should be concentrated on dealing with infectious cases and their contacts. In wealthier countries, with better medical facilities, as many non-infectious cases as possible should also be dealt with. Funds for leprosy control should not be diverted to reconstructive surgery since, however dramatic the achievements of reconstructive surgery, the aim of control is to prevent disabilities by early diagnosis and treatment rather than leave them till they need correction. As in all other campaigns against disease, detailed planning and organization are necessary.

The second part of the report is devoted to research. The Expert Committee remarks that every aspect of leprosy control discussed in the report emphasizes the need for intensified research, which it considers should be conducted in general centres of research throughout the world. Fundamental advances have been made in leprosy research since the publication of the Second Report in 1959. The infection has been transmitted to animals; proliferation of the bacillus, though to a limited extent, has been reported to occur in mouse monocytes and rat and human fibroblasts; and certain morphological characteristics of the bacilli have been found to offer a clue to infectiousness, as well as a measure of the response to chemotherapy. These advances open the way to further investigations, and the report examines what is being done and makes recommendations on the directions research in leprosy should take in the future.

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