

LEPROSY REVIEW

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THE BRITISH EMPIRE LEPROSY RELIEF ASSOCIATION.

VOL. XXIII. No. 2.

APRIL, 1952.

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The Orthopaedic Care of Leprosy
Patients

The
Pan-American Leprosy Conference
A Leprosy Survey in Zanzibar
and Pemba

Suspension of
Diaminodiphenylsulphone in Leprosy

Thickening of the Ulnar Nerve
in Leprosy and its Treatment

Reviews

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Edited by DR. R. G. COCHRANE, Medical Secretary of the British Empire Leprosy Relief Association, 167 Victoria Street, London, S.W.1, to whom all communications should be sent. The Association does not accept responsibility for views expressed by the writers.

NOTES ON CONTRIBUTORS

- MR. PAUL BRAND, F.R.C.S. is Orthopaedic Surgeon and Associate Professor of Surgery at the Christian Medical College, Vellore, S. India.
- DR. ROBERT COCHRANE is Medical Secretary of the British Empire Leprosy Relief Association and Consultant in Leprosy to the Ministry of Health.
- DR. J. ROSS INNES is Inter-territorial Leprologist to the East Africa Commission.
- DR. A. T. ROY has worked for over 20 years in leprosy, and is Senior Medical Officer of the Leprosy Home & Hospital at Purulia.
- DR. S. N. CHATTERJI is Assistant Research Worker in the Leprosy Department of the School of Tropical Medicine, Calcutta.



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KING GEORGE VI

Since our last issue the peoples of the Nation and Commonwealth have sustained a grievous loss in the death of His Majesty, King George VI. I saw the sorrow of the Indian Nation when their beloved Ghandiji was taken so suddenly away. I was privileged to be in India at the time of His Majesty's passing, and I can sincerely state that a similar sense of loss passed through the whole Republic of India. The grief expressed on the day of the passing of the King, the attendances at the Memorial Services throughout the land all indicated the deep affection His Majesty had won in the hearts of the Indian people. King George VI, by his understanding, made a significant contribution towards India's emergence as a free and sovereign Republic in the Commonwealth of Nations, and his name throughout that land will ever be beloved.

Both we and the thousands of patients whom we seek to help, share deeply with all his late Majesty's loyal subjects a sense of deep loss on his sudden death. His Majesty, as Patron of B.E.L.R.A., had shown real personal interest in the work of leprosy relief and healing, and had himself sponsored two infected children, both of whom had been discharged free of symptoms.

We, along with all the devoted subjects of Her Majesty who suffer from leprosy, whose cure and relief was one of His Majesty's deepest concerns, would extend to Her Majesty, the Queen, Queen Elizabeth the Queen Mother and all the members of the Royal family, our sympathy and expression of profound grief and loss.

R. G. COCHRANE.

EDITORIAL

We publish in this number a short report of the Medical Secretary's visit to the Pan-American Leprosy Conference. This visit was of great value, particularly as it afforded an opportunity to discuss in some detail the various terms now used by workers in relation to the classification of leprosy. A great deal of attention has been given to this subject since the Havana Congress, and it is to be hoped that much of the confusion which has arisen over the use of such terms as "Incharacteristic," "Reactional Tuberculoid," "Dimorphous" etc. will be resolved. There appears to be a general acceptance of a group of lesions which show atypical features, and cannot be included in the true tuberculoid picture. The very fact that workers throughout the world are recognising that this group exists at least indicates acceptance of a type of leprosy which has caused confusion for a very long time; whether these lesions are classified as reactional tuberculoid or as exclusively localised lepromatous macular lesions is of little importance. It is hoped that as a result of more detailed attention being given to the lepromin reaction and histopathological changes that the classification of leprosy will become better understood and more generally accepted, not only by leprosy workers but by others, and that any modification of present classifications will not offend generally accepted dermatological and immunological concepts. As a suggestion for further thought on this matter, it is proposed that a start be made by dividing all lesions, whether macular, infiltrated or polyneuritic, into three groups:—those which show a strong and definite lepromin reaction; those which are negative, and finally those in which the lepromin reaction is weak and variable. It is believed that a true appraisal of the clinical lesions of leprosy can only be based on the lepromin test, and if this were done the classification of leprosy would be better appreciated, and the two polar groups—as described by the S. American workers—would become more clearly defined. The editor is of opinion that transition from the so-called tuberculoid to leproma only occurs in those cases in which the lepromin is weak or variable, and all these lesions should be placed in the Dimorphous, Border-line or Intermediate category.

We are glad to publish an article from Purulia by Dr. A. T. Roy. A recent visit to India and Malaya indicates that there are workers in both countries who prefer to inject the parent sulphone rather than give it orally, and Dr. Roy appears to be one of these. In this article attention should again be drawn to the disparity between clinical improvement and the slowness of the bacteriological

results under sulphone therapy. While sulphone therapy is rightly hailed as the greatest therapeutic advance made in the long battle against the *M. leprae*, we cannot ignore certain disquieting features, for excessive optimism tends to give rise to wishful thinking. We would in this connection emphasise two points: (1) The relatively long period which elapses (two to five or more years) before the patient becomes negative; (2) The continued presence of acid-fast granules in the nerves after the bacilli have disappeared from the skin may indicate a resistant form of the *M. leprae*. Admittedly sulphone resistance has not yet been demonstrated, but we should not readily assume that it cannot take place. It is to be hoped that now the controversy over the form of sulphone which should be used has largely been resolved, all workers will maintain a critical attitude, so that the excessive optimism of 30 years ago, when the hydnocarpus remedies were re-discovered, will not be repeated, and continued search will be made for newer remedies. This search should be based on an increasing understanding of the methods by which the tissues of the body meet the attack of this obstinate mycobacterial invader. Leprosy lends itself to the dramatic touch, but it is only by patient, persistent, and constant endeavour that the ultimate prize—the conquest of leprosy—will be won.

We make no apology for reprinting an article by Dr. Paul Brand, Orthopaedic Surgeon to the Christian Medical College, Vellore. Although this contribution first appeared in the *Journal of the Christian Medical Association of India* in January, 1950, and much work has been done since, it will serve to open our eyes to the growing possibility of orthopaedic surgery and physiotherapy in leprosy, and result, we sincerely trust, in the recognition of the fact that modern treatment of leprosy does not only offer an excellent chance of the patient becoming free of his disease and non-infective, but ensures that this desirable result can be accomplished without deformity. It is to be hoped that ere long it will be accepted that there is now no excuse for deformity of the hands to take place, and with patient study we trust this will be the case also in regard to the feet. Complacency in this respect, and undue emphasis on therapeutics, may retard the attention which should be given to leprosy by orthopaedic surgeons and physiotherapists, and will prolong the long night of agony through which the crippled case, though “cured” by sulphones, has still to pass.

We would also draw attention to an article by that experienced worker, Dr. Chatterji. This has been reprinted from *Leprosy in India* and will indicate that practical attempts are being made to relieve deformity, and much may be done in the absence of expert orthopaedic advice.

In this number is a contribution by Dr. Ross Innes, the Inter-territorial Leprologist for the East African Territories. Zanzibar and Pemba have a leprosy problem which seems possible of control within a comparatively few decades, and the appointment of a BELRA worker to Pemba to start a control unit, it is hoped, will hasten this desirable end, so that it may be said that in one part of the world, by modern methods, leprosy has been controlled and is now no longer a public health problem. This is a challenge that BELRA has accepted, and we look forward to progress being made in this direction.

INCREASE IN RATE OF SUBSCRIPTION

Owing to the greatly enhanced cost of production, and to the fact *Leprosy Review* is at present being published at a loss, we regret that it is necessary to raise the price of the annual subscription to 15/-, including postage, commencing with the July number. Individual numbers will cost 3/6d. plus postage.

THE ORTHOPEDIC CARE OF LEPROSY PATIENTS

PAUL W. BRAND

(Reprinted from The Journal of the Christian Medical Association of India, Burma and Ceylon, January, 1950.)

My purpose today is to introduce the idea that there is a place for the work of an orthopedic surgeon in the care of leprosy patients.

Those who have looked after these people in the past have usually had in their minds two main aspects of treatment. First has been the search for a cure of the disease. Much patient research has been done over many years to this end and at last it seems that some measure of success is on the horizon. The new sulphone group of remedies gives us ground for cautious optimism that certain types of leprosy can be cured. The second line of approach has been the care of those patients who are frankly incurable. This is by far the greater part of the work of many leprosy institutions. Housing has been provided for them; nursing, food and religious comfort, and many other things to make their lot more tolerable and to enable those who cannot look after themselves to be looked after by somebody else.

My acquaintance with leprosy has been short, but from the very beginning it has seemed to me that a change of emphasis is needed in this part of its treatment. While we do not wish to decrease the amount of loving care which is expended upon the helpless patients, our primary object should be to prevent them becoming helpless, or, if they already regard themselves as helpless, to change their attitude by showing them that they can in fact develop an ability to look after themselves and to become useful members of society. The person who is being nursed is better off than the person who is helpless and destitute, but it is still better for that person to be made able to be self-reliant and to have some confidence and self-respect.

This is not the same as saying that the disease must be cured. The vast majority of these deformed patients are either incurable, as far as their disease is concerned, or else are, for all practical purposes, already cured. That is to say, the activity of the leprosy has ceased and they are left simply with the residual paralysis and deformity. These latter can be tackled along orthopedic lines by means of splints, exercises, massage, and by operation in such a way that the patients usefulness may be maintained or improved. And then one must see that they are given useful and gainful occupations to suit their limited capabilities.

The orthopedic problems in leprosy are mainly those of anaesthesia and of paralysis and we are concerned with these things in the hands and the feet.

Hands—Anaesthesia. The problem of anaesthesia in the hands must be taken seriously if one is to maintain the usefulness and activity of these organs. Every time a patient injures or burns his fingers a scar is produced and that scar by its contraction adds something to the tendency for the fingers to flex permanently into the claw-hand position which is our chief problem in rehabilitation of the hands.

In designing occupations for these patients, therefore, one must foresee anything which is likely to burn the hands, and allow a minimum of contact with sharp objects that may cut them. Damage is also often caused by local friction such as the continuous running of cotton over the index finger of a woman who is spinning. It is important therefore that no patient with anaesthetic hands should be allowed to do cooking or plumbing and if they are employed in occupations such as carpentry and cobbling where there are sharp instruments to be handled they should have careful instruction about the way to handle their tools so that their hands are always well clear of cutting edges.

Where there is continuous friction, as in spinning and weaving industries, the part of the hand that is subject to the friction should have a leather finger saddle or a pad which can take the friction from the skin. All patients, especially when starting a new occupation, should have regular hand-inspection by the doctor or the welfare officer, and any redness or sign of blistering immediately investigated, the hand put at rest, and his work watched when he starts again to see the exact cause of the pressure of friction so that it may be removed. This may involve altering the shape of the handles of his tools or instructing him in a new way of holding them, or the wearing of a leather pad as before described.

It will be found that many women are reluctant to give up cooking, and members of their family may urge them to continue it. The important part of cooking to these people's minds is the preparation of the food. If they are allowed to do this part they are quite willing for somebody else to do the actual cooking, involving the handling of pots and pans and fire. Thus, several families may co-operate—each wife preparing her own food, and one person who has normal hands doing the actual cooking for several families or groups.

Paralysis. The paralysis of the hand in leprosy is something which appears never to have been fully investigated, but during the past year I have been following a large series of cases with some care and it seems that increasing weakness of the hand follows a certain definite and well defined pattern. The first muscles to go are the interossei between the ring and little fingers. The weakness then spreads to the lumbricals and interossei across the palm from the ulna to the radial side, and when almost all power has been lost in these muscles then the muscles of the thenar eminence are involved—the opponens pollicis paralysis preventing opposition of the thumb. The long flexors and long extensors of the fingers and thumb, however, retain their power, as also normally do the flexors and extensors of the wrist.

In the normal action of grasping, the hand begins with the fingers extended and the thumb opposed to them. The first part of the grasp is chiefly the lumbrical movement of flexion of the metacarpophalangeal joint (Figure A I). As the hand closes upon the object it is grasping, the long flexors take up the task and the interphalangeal joints are flexed making the fingers curve around the object and grip (Figure A II). The leprosy patient, having lost his lumbricals, initiates his attempt to grasp by flexing his interphalangeal joints while the metacarpophalangeal joints remain extended (Figures BI and BII). Only when the interphalangeal joints are fully flexed do the metacarpophalangeal joints begin to flex. Thus the fingers are "rolled up" into the palm and present

their dorsal surface, of finger nails and distal knuckles, to the object to be grasped rather than the palmer surface of the fingers (Figure BIII). Any object therefore except a very narrow one is pushed out of the hand by the clenched fingers rather than grasped. It will be noticed that most patients with weak hands do their grasping by adducting the thumb against the second metacarpal and do not use their fingers at all.

We have therefore to try to provide a substitute for the lumbricals and for the *opponens pollicis*.

The other problem is to prevent contracture of the fingers in the clawhand position. The reason this develops is that for full extension of the fingers is required the co-operation and simultaneous action of the lumbricals, *interossei* and the long extensors. The long extensors normally extend the metacarpophalangeal joints and the lumbricals extend the interphalangeal joints. If, however, the metacarpal joint is flexed, this flexion enables the long extensors to extend the interphalangeal joints. A test therefore of early loss of the intrinsic muscles of the hand is to ask the patient to flex the metacarpophalangeal joints and to extend the interphalangeal joints at the same time (as in Figure A I). This is quite impossible with paralysis of the lumbricals.

When patients find that they cannot extend their interphalangeal joints, they try to do it by contracting their long extensors more powerfully. This only produces hyperextension at the metacarpophalangeal joint but does not extend the interphalangeal joint at all. Therefore the interphalangeal joints are never extended, and when they have remained flexed for a long time they become stiff in that position. Capsular contractions develop and before long result in the familiar permanent claw-hand.

The first duty of the medical officer is to see that this contracted claw-hand never develops. The secret of this is to realise that passive flexion of the metacarpophalangeal joint allows active extension of the interphalangeal joints. In other words, if we imitate the action of the lumbricals in flexing the metacarpophalangeal joint then the long extensors are able to extend the interphalangeal joints. The simplest way to do this is to press the proximal phalanx forward into flexion with the other hand and then instruct the patient to straighten his fingers. If no permanent contracture has developed he will be able at once to extend these joints. If he is now instructed to perform this movement regularly many times in a day and accompany it by massage to his fingers using a vegetable oil, such as gingelly oil, he will maintain the mobility of his fingers and no contracture will ever develop. I have devised a splint patterned somewhat after the knuckle-duster splint described originally by Haighet. (See illustration.) This splint is simply a mechanism

for keeping the metacarpophalangeal joint flexed whilst allowing mobility to the rest of the hand. It is my habit in cases where there is weakness or commencing contracture of the fingers to make the patient wear this splint for two or three hours at a time twice a day and whilst wearing it to exercise his fingers through their full range, and between whiles to rub oil into his fingers and keep them mobile. In this way many a commencing contracture can be completely straightened out and a mobile hand produced.

This does not of course bring back power to the paralysed muscles. We know of no way of doing that, but it does improve the capacity to grip.

The next problem is to try and devise something which will do the work of the lumbricals and form a new and wholly active hand. This work is still in the experimental stage and I have nothing to report about the methods or results at present.*

Finally we are concerned with the hands which are fully contracted in a permanent way so that even passive extension is quite impossible and the grip is wholly absent. In these cases it seems impossible to regain the movements of the interphalangeal joints, but if an operation is performed on these joints to open them out to about 90° for the proximal interphalangeal joints and 160° for the distal and fix them in that position, then the movements of the metacarpophalangeal joints, which are never lost, will enable quite a reasonable grip to be achieved.

In assessing the occupation for the individual, the chief task is to decide upon the kind of tool which their hand is capable of holding. As a rule the more advanced the disease the narrower must be the handle of the tool. Even an advanced claw-hand can hold a handle $\frac{1}{2}$ in. in diameter; in milder cases 1 in. or $1\frac{1}{2}$ in. in diameter. The milder cases can grip and leave go their tools quickly while the more advanced cases need a trade where, having once grasped their tool, they may keep it in their hands for longer periods without having to put it down. Some trade in which there is only one instrument to use or one handle to turn is the one to choose for these patients.

Feet—Anaesthesia. The feet are subject to more pressure and more trauma than hands, so the skin is more liable to give way. Trophic ulceration is therefore more a problem in the feet than in the hands.

The two factors which produce ulceration are sustained pressure and active injury.

In general people who wear shoes or sandals suffer from sus-

* See note added to article.

tained pressure and people who walk bare-foot from direct injury. The reason for the former is that normal rough ground gives a more even pressure over the surface of the feet than shoes do. The ideal ground surface for leprosy sanatoria would be sand, because as the foot sinks into the sand a little at each step the weight of the body is transmitted through every part of the foot—the sand having adjusted its shape to the shape of the foot. No part of the sole therefore takes more pressure than any other part, and pressure sores are unlikely. Grass turf is the next best surface as here again the springy nature of the ground allows it to be moulded to the contours of the foot. The worst type of surface is probably a hard gravel which is both unyielding and sometimes sharp.

Shoes when they are new present a flat surface to the sole of the foot and therefore the weight of the body is transmitted through a small area of the heel and the metatarsal heads—whereas all the area of the instep takes no weight at all. As shoes become older and a slight moulding of the leather takes place a correspondingly bigger area of the feet shares the pressure; but even so it is never distributed as well as it is in the barefoot patient unless the soles are especially designed and built to conform to the contours of the feet and to allow the whole of the skin of the feet to share equally in the weight-bearing process. It should be a rule that all shoes for leprosy patients should be sewn throughout. Nails should never be used—they are a frequent cause of ulcers when they begin to come through the sole as the shoe wears.

Probably, except with tight shoes, the factor “sustained pressure” does not operate while the patient is walking because the action of walking depends upon alternately resting on the foot and then raising it immediately from the ground. This allows intermittent circulation to all parts of the sole. The real damage is done whilst patients are standing or squatting, with no feeling of discomfort to make them change their posture and shift their weight from one leg to the other. These patients are content to stand still in one posture or to squat for very long periods at a time and the part of their skin which takes their weight is continuously compressed and has no blood supply for long enough to cause complete local gangrene. This is the essence of the etiology of the trophic ulcer.

It is of fundamental importance that no patient with anaesthetic feet should ever be allowed to stand still or to squat for more than 5 minutes at a time.

It should be the duty of the medical officer to see that everywhere in sanatoria there are placed large numbers of simple stools, benches, or even flat stones upon which the patients may sit when they wish to rest.

The matter of properly built shoes and sandals is also important. All patients who show any tendency towards ulceration near the metatarsal heads should have a metatarsal bar fixed to their shoes. This is a simple bar of leather usually $\frac{5}{8}$ th of an inch thick, nailed or sewn to the underside of the sole behind the point at which the weight of the body is taken through the metatarsal heads. If you look at the underside of the sole of a shoe or a sandal, you will see clearly where the chief weight of the body is taken, because that part of the sole will be worn whereas the nonweight-bearing part will not be worn so much. A metatarsal bar must be placed behind the point of maximum wear and tear. Almost all doctors place these bars too far forward on the sole. The object of the bar is to transmit weight to the instep and to spare the metatarsal heads.

It is also helpful but rather more expensive to try and get the sole of the shoe or sandal correctly moulded to the shape of the foot. I have been trying to do this by using sponge rubber in the sole and cutting the sponge rubber to the shape of a clay-mould which I make for the patients by requiring them to stand squarely with their whole weight resting on a lump of clay. The cobbler is then instructed to build a sole with the same lumps and hollows as the clay mould, and then finish off with a soft upper.

The aim of the medical officer should be to prevent ulcers of the foot rather than to cure them, and in the work of prevention it is necessary to inspect all anaesthetic feet and their shoes at regular intervals. The sanatorium cobbler should be in attendance at these inspections and should be a skilful workman with enough intelligence to understand the principles that are involved. Danger signs should be looked out for such as cracks in the sole of the foot and callosities and if shoes are found to be responsible for uneven pressure they should be adjusted so that they do not cause harm. Shoes should always be resoled before they have become badly worn out because constant contact with the foot will have moulded the inner part of the shoe to the foot and this inner moulded leather should be preserved for as long as possible, even for many years, by constantly adding a flat piece of leather as an under-sole before the inner-sole becomes worn. This should be done without removing the remnants of the worn under-sole, as in this way the original shape of the shoe is better preserved. The factor of direct trauma in the causation of ulcers operates in proportion to the amount of walking that the patient does, and the kind of ground he walks upon. Any long walk on rough ground will give opportunity for sharp stones to cut or wear away the skin of the sole of a bare-foot patient or for friction to produce a blister on the foot of one who wears ill-fitting shoes.

Intelligent patients who are helped fully to understand these principles should be able so to regulate their activity that an ulcer becomes a rare occurrence. When it does occur further activity must be restricted until it is soundly healed.

Paralysis. When the muscles of the foot are weakened the balance of the foot becomes unstable, and this leads to one side or end of the foot taking more pressure than the other; and consequently ulcerating.

The commonest weakness in leprosy is that of the dorsiflexors of the foot and of the peronei. This leads to a drop of the fore-foot and of the lateral edge of the foot respectively. The leprosy patient with drop foot develops a high stepping gait, but even so he is not able to prevent his toes from dragging a little on the ground. And these patients get ulcers on the ends of their toes and on the metatarsal heads—not so much from pressure as from friction. They do better with shoes, and better still with stiff-soled shoes which have a strap running from the top of the shoe to a strap around the upper end of the calf keeping the foot dorsiflexed. It is more satisfactory if a metal upright is fixed from the heel of the shoes up the calf to support a strap around the top of the calf and the strap lifting the toe of the shoes. The same device may be used for cases with paralysed peroneal muscles but in this case the point of attachment of the lifting strap must be to the lateral side of the foot so as to replace the pull of the peronei.

The question then arises, can we relieve the “ drop foot ” by any orthopedic operation? Here I am unable to give a final answer. I can say that it seems that there will not be much scope for tendon transplantation operations—because as leprosy paralysis is so frequently progressive the muscles that one uses for transplantation this year may themselves become paralysed next year. There seems to be more hope for the type of operation of tendon fixation or suspension where the dorsiflexor tendons are rigidly fixed into the anterior surface of the tibia, but we have not yet performed enough of these operations to be able to report on the results. Some form of foot stabilization may prove to be the best answer in the long run, although we have yet to find whether the bones and tendons of a leprosy patient will stand the kind of operative interference which this will require.

Ulcers. In dealing with established ulcers many of you will have had more experience than I have. I will just mention one or two points that I believe to be important. The first is that where possible every established ulcer should be treated by complete rest to the foot—that is no weight-bearing. Ideally the patient should

use crutches until the ulcer is healed or else stay in bed, failing which a thick ring pad should be bandaged to the foot surrounding the ulcer so that when the foot bears weight the actual ulcer may not come in contact with the shoe or the ground. A probe should be used frequently to find out whether the ulcer goes down to bare bone. Those that do not are probably best treated by injections of hydnocarpus oil deep to the ulcer floor. When the ulcer involves the bone an X-ray should be taken to see whether the bone is diseased.

Next to continued weight-bearing, chronic osteomyelitis is probably the commonest cause of persistence of trophic ulcers. Therefore when a probe is found to reach bare bone in an ulcer, an X-ray should be taken to discover the extent of the osteomyelitis, and unless this is very localized indeed it is usually best to proceed at once to the removal of the diseased bone. This is best done through a dorsal or lateral incision so as not to leave a weight bearing scar. I have found that if one removes a bone such as a metatarsal through a dorsal incision one is usually able at the same operation to excise the edges of the ulcer on the ventral surface and to sew it up. A dorsal incision should never be sutured up but should be left open packed with vaseline gauze to granulate, as described by Cochrane in his Textbook of Leprosy. The depth of the ulcer will therefore be drained dorsally until the wound heals up and the big scar will be on the dorsum of the foot instead of on the weight-lifting surface.

The last resort in the care of the foot is the operation of amputation, but it should not be delayed if it will allow the patient to walk on a sound stump rather than to remain incapacitated by a widely ulcerated foot with wide chronic osteomyelitis.

I have become convinced rather against my orthopedic instincts that it is justifiable and even wise to amputate as near as possible to the ulcerated area—that is to say for metatarsal head ulceration a Lesfranc's amputation or a Chopart or Piragoff is quite justifiable even though a skin flap may be within an inch of the open ulcerated area. There are three reasons for this:

Firstly, because these patients are so poor and their number so large we cannot afford to provide them with proper artificial limbs for high amputations.

Secondly, patients with leprosy seem to have a better capacity to heal than the average patient who requires an amputation. This is probably partly because there is no diminution in the blood supply of these feet as there is in so many other cases of local gangrene.

Thirdly, the battle against trophic ulceration is frequently a

retreating battle and it may be that the amputation stump itself will become ulcerated after a few years. If one has done a low amputation, there is an opportunity to give the patient a second or even third amputation at successively higher levels — as for instance Lesfranc's followed by Symes followed by Site of Election; each time leaving the patient with a sound stump which may last many years.

Because these operations are always performed near gross sepsis it is important that the incision should not be primarily sutured completely but the skin flaps should be packed open to granulate, perhaps for secondary suture later on.

One should take care that the amputation scars are off the weight bearing area of the stump, and one should give to an amputation stump the same, if not greater, care than we have recommended for the anaesthetic feet. A careful mould of the stump should be taken and a sponge rubber socket made for the stump and fitted with a sole-plate.

I hope that I have left you with the impression that I know very little about my subject, because I wish to urge upon you all the fact that very little is known, and that it is our duty to press forward in this branch of surgery. We must keep before us a realisation that although many of our patients are hopelessly diseased as far as cure is concerned, a little careful application of the science and art of orthopedic surgery may be able to change their outlook from one of helplessness and despondency to one in which they can look forward to a life of usefulness and activity.

I wish to acknowledge the help and encouragement I have received from the staff at the Lady Willingdon Leprosy Sanatorium, and in particular from the Medical Superintendent, Dr. H. Paul, the Welfare Officer, Mr. Rama Krishna Ayyer, and from the Director of Research, Dr. R. G. Cochrane.

A note supplied by Dr. Paul Brand, dated March, 1952, reads as follows:—

Since this article was presented (May, 1949), the author has had a considerably wider experience of hand reconstruction in leprosy, and reports that experience has confirmed his original impression that the long muscles of the arm are very rarely paralysed, except that the flexor carpi ulnaris is paralysed nearly as frequently as the small muscles. This means that the long flexors, and particularly the flexor digitorum sublimis, are available for tendon transplantation, and that they are unlikely subsequently to

become paralysed. Some dozens of hand reconstructions have been attempted, and in many cases a full grasp has been restored, and in every case the hand has been left significantly more useful than it was before. The operations which are recommended are a transplantation of the flexor digitorum sublimis for the lumbrical in the index and little finger, and substitution of the flexor sublimis of the long finger, split and used, half for the lumbrical of the long, and half for the lumbrical of the ring finger; and the use of the flexor digitorum sublimis of the ring finger for the opposition and abduction of the thumb. These operations have been only slightly modified from the classical operations described by Bunnell in his book, "Surgery of the Hand," and by other authors on the same subject. The operations will be more fully described and annotated in further articles which will be appearing in surgical journals. It seems that general principles of hand surgery may be used in leprosy, except that a little longer time should be allowed for healing, both of skin and of tendons, and special care must be taken with plasters and tight bandages to avoid superficial pressure necrosis of the skin. The results, when followed by proper education and rehabilitation, are most gratifying.

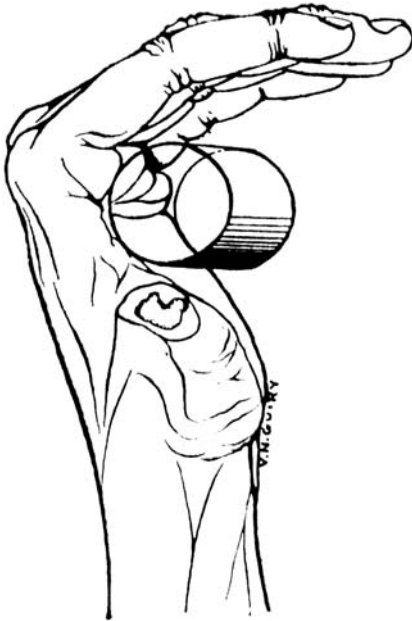


Figure A I.—Normal hand beginning to grasp. Thumb not shown. Metacarpophalangeal joints more flexed than interphalangeals.



Figure A II.—Normal grasp complete.

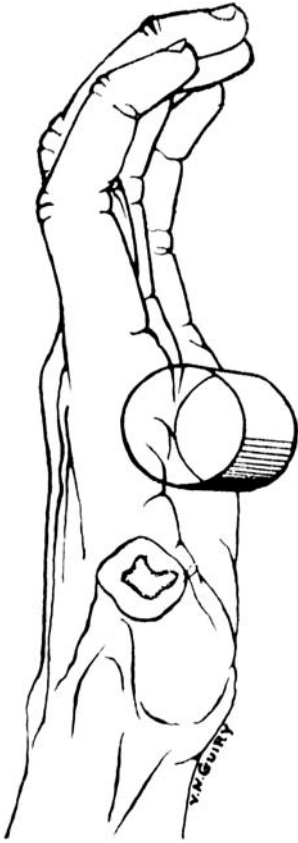


Figure B I.—Mild claw hand open before grasping.

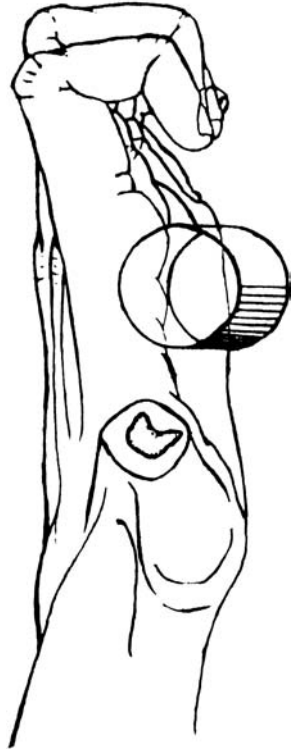


Figure B II.—Same hand beginning attempt to grasp. Note interphalangeal joints flex before metacarpophalangeal joints.

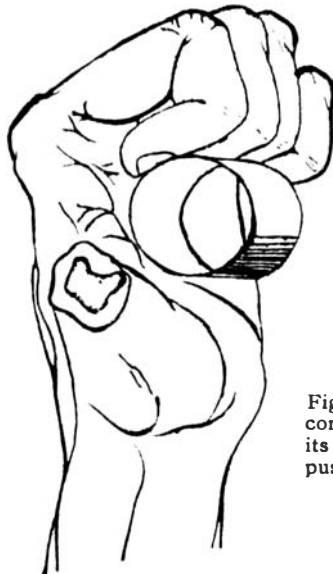


Figure B III.—Grasp complete but failing to hold its object which is being pushed out of palm.

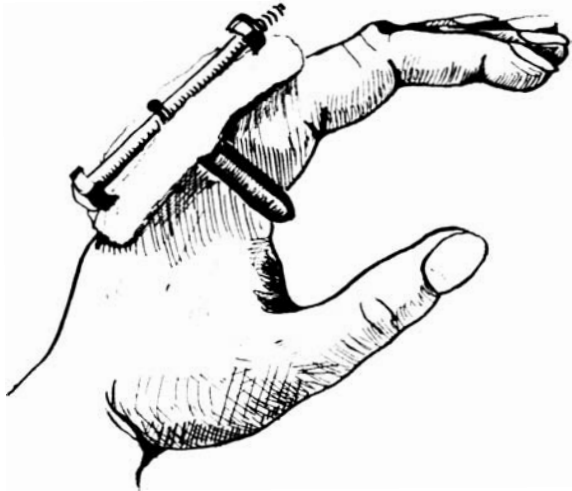
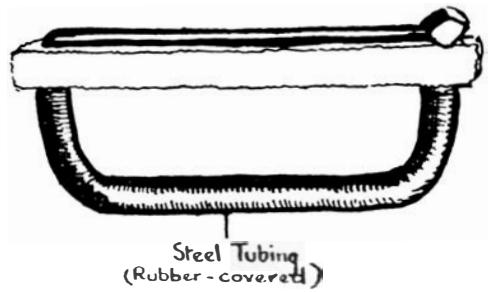


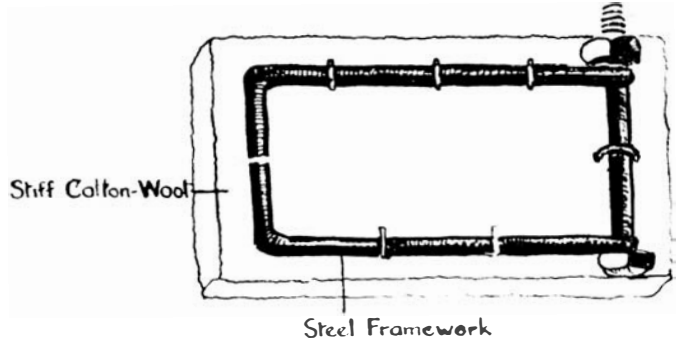
Figure IV.—Hand-splint for commencing contractures in position.



(a) Side view. (b) front view.



Steel Tubing
(Rubber-covered)



Stiff Cotton-Wool

Steel Framework

Figure V.—Drawing of simplified knuckle-duster splint for commencing claw-hand.

REPORT OF A VISIT TO THE PAN AMERICAN
LEPROSY CONFERENCE AT BUENOS AIRES,
ARGENTINA, AND TO THE UNITED STATES

R. G. COCHRANE

I left London Airport on December 4th, 1951, and reached Rio de Janeiro on December 5th. On arrival there I was met by Dr. H. C. de Souza-Araujo, of the Instituto Oswaldo Cruz, and Mr. Godkin, of PROFAR, who also had connections with the Wellcome Foundation in London. I spent two hours at the Instituto Oswaldo Cruz discussing with Dr. de Souza-Araujo his experiments on the growth of *M. leprae*, and subsequent inoculation into animals. While this work is of extreme interest, and will be followed with the hope that more permanent results will be seen, I am personally of the opinion that claims that the organism is *M. leprae* have not been altogether substantiated. The immunological work on animals does not tally with what one would expect from lepromin made from a specific organism, and until more evidence is forthcoming one has to note this work, and the enthusiasm of Dr. de Souza-Araujo, and await further confirmation of his studies.

I also had the pleasure the next day of meeting Dr. Azulay, who is a histopathologist working in co-operation with the clinician. I also visited the leprosy institution near Rio de Janeiro. As I was spending so short a time in South America, it was impossible for me to devote attention to the many aspects of leprosy which are present in that country, and I felt that the most profitable way I could use my time was to concentrate on histological work. In discussions with Dr. Azulay it seemed apparent—and this was later confirmed in my visits to Sao Paulo and Buenos Aires—that the South American workers were describing under the division of tuberculoid leprosy two conditions, namely reactional tuberculoid and tuberculoid in reaction. The former types of lesions were apparently those cases which showed either a negative or weakly positive lepromin test, and in which the histology was indicative of what has been previously described as intermediate, border line, or dimorphous lesions, and in discussing the sections with histopathologists it was evident that the tuberculoid cases in South America were divided into those which showed strongly positive lepromin, and little or no tendency to reversal of the lepromin reaction and those in which the lepromin reaction was negative, or weakly positive, and were liable to show a reversal of the lepromin

reaction and change to leproma. My conversations with Dr. Azulay were both stimulating and encouraging.

I proceeded on December 6th to Sao Paulo and spent the whole of December 7th there. I was privileged to meet Dr. Nelson de Souza Campos, who took me to see his work in the preventorium, and showed me some of the experiments which he was carrying out in relation to B.C.G. and the lepromin test. There seems to be little doubt that B.C.G. vaccination will change a lepromin negative to a lepromin positive (Mitsuda), and this positivity of the lepromin has been maintained in many instances for two years, although the tuberculin allergy after B.C.G. changed over to the negative side after one year. Whether the lepromin reaction would remain positive if the child were exposed to further contact with open cases is a finding which could only be confirmed over a very long period. From the studies of the Philippine workers and those in Bombay, it seems that all persons who become inoculated with the *M. leprae* pass through a positive allergic phase. In other words, the lepromin is initially positive, and the various factors, such as further infection or multiplication within the skin of *M. leprae*, and clinical manifestations of the disease in later life may result in altering the reaction from positive to negative. It is therefore too soon as yet to conclude that B.C.G. gives a permanent positive tissue immunity to the *M. leprae*. Nevertheless, the work which Dr. Nelson de Souza Campos has been doing is extremely detailed and interesting, and one will follow these studies with the very greatest interest, and the fact that B.C.G. vaccination is being done, in connection with the lepromin test, in other territories, will add great interest to the subject. As I have said, however, it is premature yet to draw any final or definite conclusions.

I had the pleasure of meeting Dr. Lauro de Souza Lima for a short while, but was unable to visit his institution as he was due to leave for Buenos Aires the next morning after my arrival.

I returned to Rio de Janeiro and prepared to go on to Buenos Aires on the Saturday, to attend the Pan American Leprosy Conference. Unfortunately, owing to a strike of ground staff, all planes were grounded, and my departure was delayed. I hardly need to describe how, at the very last moment, I was able to secure a seat in a Scandinavian plane, and was the only foreign delegate who was fortunate enough to attend the Conference! In this connection I should like to express my very great sense of gratitude to Mr. Godkin and his co-Director in PROFAR, Mr. Estill, for without their active co-operation and assistance it would have been quite impossible for me to reach Buenos Aires in time for the Conference.

I arrived in Buenos Aires at 3 a.m. on the morning of Sunday,

December 9th, and was present at the opening of the technical sessions. I unfortunately missed the welcome to the Conference and delegates. All the proceedings were conducted in Spanish, and it was very difficult for a delegate who was not familiar with the language to follow the papers. I do not propose further to describe in detail the papers that were given at the Conference, and shall refer only to the two main topics which were of widespread interest to those studying leprosy, namely, (1) classification and the possibility of change of type, and (2) sulphone therapy.

I myself presented a paper on the classification of leprosy, with particular reference to histopathology, and this paper was received very kindly by the Conference, and the general outline of classification which I presented was accepted as a basis for the discussion on classification. While the Conference was not able to make any definite changes in the Havana classification, because this is a matter only for an international congress, there was general agreement that the indeterminate group of cases divided themselves into (a) those with a histology showing simple inflammatory changes, and (b) those with a histology showing granulomatous changes. The former were those lesions which showed incharacteristic histology and covered in general what one would consider the pre-lepromatous lesions, and possibly the simple macular lesions of the Cairo classification, as well as resolved lesions. Those described under indeterminate lesions with granulomatous changes appeared to include all those cases which had been variously described as border line, intermediate, dimorphous, and reactional tuberculoid. It is interesting to note that the Calcutta workers are now describing this group of lesions as lepromatous lesions with exclusively localised macules,* and these lesions appear to belong to the type described as atypical leproma.

The papers on classification and the change or reversal of type from "tuberculoid" to leproma, were of extreme interest, and I have no doubt that the contributions and the discussions with regard to classification at this Conference will be of very great assistance in formulating views on classification at the next international leprosy congress in 1953.

With regard to sulphone therapy, it was extremely unfortunate that the French delegation were delayed in Brazil on account of the strike of ground staff, and were unable to arrive in time for the Conference. Hence, the presentations of the French workers on DDS was not given, and therefore their views in reference to the advantages of using DDS over other sulphone preparations could

* *Leprosy in India*, Vol. XXIII (1951) No. 4, 200.

not be given adequate consideration. A paper by Dr. E. H. Payne was of interest with reference to two special points (1) that it may not be correct to conclude that because a drug is active against the mycobacterium of tuberculosis it will also be active against the mycobacterium of leprosy, and (2) that more attention should be paid to the monosubstituted derivatives of DDS, as he felt that these were probably more active, not only than the disubstituted ones, but also than the parent substance. The work on monosubstituted sulphones will be followed with the greatest of interest.

There were the usual functions attached to the Conference, and one of the interesting visits was to the Eva Peron Home for Healthy Children. This was situated some miles outside Buenos Aires, and was a very beautiful home. The children appeared to be very happy and contented. The Argentine Government deserves congratulations on this work. In addition a visit was paid to the Sanatoria Sommer, under the directorship of Dr. Fiol. During this visit cases were demonstrated, and I had the privilege of discussing general surgical and orthopaedic questions with one of the visiting surgeons, and the Sub-Director of the Sanatoria. The delegations were received by His Excellency, General Peron. After his address the official delegates from the various countries had the privilege of a personal interview with the President, who cordially welcomed the delegations that were able to attend the Conference, and outlined in an interesting way the general situation with regard to leprosy in Argentina. I also had the privilege of an interview with the British Ambassador at the Embassy, and met the staff.

Apart from the attendance at the official meetings of the Conference, I had the pleasure of meeting such S. American workers as Dr. Basombrio, Dr. Schujman and other Argentine leprologists. On the day before I left I had the advantage of a long discussion with those interested in histopathology in Dr. Basombrio's laboratory. I was able to demonstrate some of my own histopathological material, and I feel sure that this discussion enabled us to come to a better understanding with reference to each other's point of view, and was personally of extreme value to me. I would like to express my personal thanks for the friendship and cordiality of all the S. American leprologists, and would record my gratitude to Mr. Frederick Thompson, of Burroughs Wellcome & Co., Mr. Pat Moxey, of Toc H, and others who made my stay of such extreme interest.

I left Buenos Aires on Saturday, December 15th, and arrived in New York on the 16th, to be greeted with a temperature of 20 degrees below zero. I had the privilege of meeting the Executive Committee of the American Leprosy Missions, who very kindly

gave me a dinner in New York, at which I spoke, and was most gratified and encouraged at the enthusiasm of all those I met and talked with on leprosy.

I left for Washington on December 18th and had a conference with the Committee of the Leonard Wood Memorial at the Institute of Pathology. It was a very great pleasure to be able to demonstrate my histopathological material and discuss with those interested in the pathological aspect details concerning this, and I should like to express my grateful thanks to Mr. Perry Burgess and Prof. Karsner for arranging this most instructive and helpful meeting. Unfortunately Dr. Doull was delayed owing to inclement weather, and I was unable to meet him in Washington. I left Washington on December 23rd and arrived back in London at 4.15 p.m. on Christmas Eve.

In closing this report on my visit to S. America, I should like personally to acknowledge my indebtedness to the Wellcome Research Foundation for making a very generous donation, which covered the cost of my travel. I would also like to express my gratitude to the American Leprosy Missions and the Leonard Wood Memorial for their kind entertainment in New York and Washington.

A LEPROSY SURVEY IN ZANZIBAR AND PEMBA

JAMES ROSS INNES.

INTRODUCTION.

Between the dates 16th July and 16th August 1951, the author was loaned by the East African High Commission to the Zanzibar Government for a leprosy survey of Zanzibar and Pemba. With the generous and assiduous assistance of all members of the administrative and medical departments of His Highness's Government, it was found possible to complete an adequate survey in both islands, by examining samples of the populations in a great number of places. There was not a corner of the country into which the survey team did not penetrate, and the team included Mr. L. G. W. Vear, a leprosy worker from the British Empire Leprosy Relief Association.

In deference to local sentiment, it was found necessary in Zanzibar island to omit the examination of females, but in Pemba Island adequate numbers of females also volunteered for examination.

The island of Zanzibar has a land area of 640 square miles and a population of 150,000, whilst Pemba has a land area of 380 square miles, and a population of 114,000. Both lie close to the coast of East Africa, and latitude 5 S. passes through Pemba, and latitude 6 S. through Zanzibar. Both have a long history, and in modern times comprise a Sultanate and Protectorate with a British Resident. The islands are noted for their clove top crops, coconuts, and similar products. The climate is warm and humid.

In effecting the survey, it was found necessary to travel by ship, motor car, launch, dhow, canoe, and on foot. Zanzibar was covered in 12 working days, Pemba in 13. Certain of the Tumbatu people in Zanzibar refused co-operation with the survey, but most people co-operated splendidly, particularly in Pemba, where a total of 21,903 people were obtained for examination. In Zanzibar, the total was 10,786. The leprosy rate emerged as very moderate in both islands. Already in existence are two small leprosaria, of about 50 inpatients, one at Walezo in Zanzibar, the other at Makondeni in Pemba.

SECTION A. RESULTS OF LEPROSY SURVEY IN ZANZIBAR.

Date	Places	Male pop. at risk	No. of males examined	No. of cases of leprosy	Crude rate per 1,000	Corrected rate per 1,000
17 July	Zanzibar City ...	20,000	2,000	4	2.0	1.5
18 "	Mtoni, Bububu, Bweleo, Fumba ...	4,000	637	2	2.9	2.17
19 "	Fuoni, Uzini, Kidimni, Ndagaa, Mchangani ...	5,000	1,006	7	6.9	5.17
20 "	Mkamburimsafa, Mwera, Ndiyani, Chwaka, Uroa ...	3,000	924	3	3.2	2.4
21 & 22 July	Bungi, Kikungwi, Unguja, Ukuu, Pete, Kitogani, Jambiani, Paje, Bwejuu, Prison Farms ...	6,000	1,118	2	1.7	1.27
23 July	Muyuni, Kizimkazi, Kibutani, Mtende, Makunduchi ...	7,000	851	4	4.7	3.51
24 "	Mfenesini, Mekundu, Fujoni, Mangapwani, Misufini, Makoba ...	4,000	686	5	7.2	5.4
25 July	Kazole, Mahonda, Mbiji, Donge, Mko-kotoni, Mkwajuni ...	5,000	1,536	9	5.8	4.3
26 "	Tumbatu I. ...	1,500	71	—	—	—
27 "	Kinduni, Mgambo, Kinyasini, Upenja, Chaani, Chutampa, Moga ...	5,000	1,781	9	5.0	3.8
28 "	Potowa, Kidoto, Kigunda, Nungwi ..	4,000	138	2	—	—
	Grand totals ...		10,748	47	4.3	3.9

NOTES.

The correction for lack of females is based on the assumption that two males to every female contract leprosy.

For Zanzibar the average leprosy rate per thousand population may thus be taken as 3.9 per 1000, and the estimated total number of leprosy cases existing as 600.

SECTION B. RESULTS OF LEPROSY SURVEY IN PEMBA.

Date	Places	Whole pop. at risk	No. of persons examined	No. of cases of leprosy	Leprosy rate per 1,000 pop.
1 Aug.	Konde, Chimba ...	4,000	946	7	7.4
2 "	Shumba, Micheweni, Wingwi ...	7,000	1,350	7	5.1
3 "	Wete, Gando ...	6,000	2,052	10	4.8
4 "	Daya, Nyale ...	3,000	596	2	3.3
6 "	Kinazini, Mianzini, Pandani ...	6,000	1,417	8	5.6
7 "	Kojani I., Mchamgandogo, Kiwani ...	5,000	1,865	9	4.8
8 "	Ziwani, Wawi Aerodrome, Mkanjuni ...	7,000	2,289	12	5.2
9 "	Kiuyu, Ole, Kangangani ...	3,500	1,376	7	5.0
10 "	Chake Chake ...	4,000	1,388	6	4.3
11 "	Chonga, Chambani .	5,000	1,766	13	7.8
13 "	Fufuni ...	4,000	1,783	16	8.9
14 "	Jundamiti, Kengeja	5,000	2,829	15	5.1
15 "	Mkoni, Makongwe, Chokocho, Panzi I.	4,000	2,246	10	4.4
Total ...			21,903	122	5.5

NOTES.

As an adequate representation of females as well as males was obtained in Pemba, the leprosy rate per 1000 may be taken as 5.5. The estimated number of cases of leprosy existing in Pemba is 600.

SECTION C. ANALYSIS OF THE CASES OF LEPROSY FOUND. 169 CASES.

(1) *Sex.* The 47 cases in Zanzibar were all males, as only males offered for the survey. Of the 122 Pemba cases, 45 were females.

(2) *Age incidence.* (a) *Children.* The number of cases of age 14 years and under were 37, or 21.8 per cent. of the whole.

ages	14	13	12	11	10	8	7	6
number	2	2	6	7	4	5	6	5

(b) adults, or those over 14 years, were 132 cases

ages	60	55	50	45	40	35	30	25	20	18	16	15
number	4	3	11	13	16	17	29	21	12	2	1	3

(3) *Racial or tribal origins.* The race or tribe, as given by the patient or his relatives, was in the 169 cases as follows:—

Shirazi	64 cases	Mgoni	1 case
Arab	28 "	Somali	1 "
Tumbatu	19 "	Mwera	1 "
Swahili	15 "	Zigua	1 "
Wahadimu	6 "	Muganda	1 "
Wanyamwezi	8 "	Zarai	1 "
Kojani	5 "		
Yao	4 "		169 cases
Kikuyu	3 "		
Ndingo	3 "		
Makonde	2 "		
Kadum	2 "		
Mzeramu	2 "		
Nyasa	2 "		

It was interesting to find that there were 14 cases of leprosy who derived from Central or East African mainland tribes, and had been born in Zanzibar and Pemba.

Also strangers from the mainland, some 16 cases of leprosy, had the following varying degrees of residence:—

37 years	1 case	2 years	1 case
30 years	4 cases	6 months	1 "
20 years	2 "	5 months	1 "
10 years	2 "	2 months	1 "
8 years	1 case	1 month	1 "
4 years	1 "		

We see in this reflection of the probable source of most of the leprosy of these islands in the past and present, namely the importation of slaves in the past, and paid labour today, from the African mainland. About 18 per cent. of all cases of leprosy found were of *mainland origin*.

(4) *Living Conditions.* Of these cases, 28 or 16 per cent. lived alone, but 82 lived in house contact with a total of 252 children. In all cases, housing was inadequate in light and air entry, and overcrowded. The custom of multiple wives was observed in the case of 7 men, who had three wives to two wives apiece. The percentage living alone is much higher than has been observed in surveys on the mainland, where it is usually about 5 per cent. who live alone, so some greater degree of the understanding of the need for isolation, is evident. The child cases in contact point, as ever, to the way leprosy is perpetuated in the community.

Groups of family leprosy, that is, one or more members of the same family afflicted with leprosy, were observed in 10 distinct instances. The original source of the infection in the family was usually a parent or elder relative.

(5) *Clinical type of leprosy.*

Lepromatous leprosy, the infectious type, was found in 32 cases, or 18. per cent.

There was a great predominance of elevated tuberculoid lesions of leprosy, these cases numbering 103, or 60 per cent.

Intermediate and polyneuritic cases were of lesser importance.

SECTION D. THE LEPROSY PROBLEM IN ZANZIBAR AND PEMBA.

The position is that we must expect to deal with some 600 cases of leprosy in each island. This is a moderate problem, contrasting with the East African countries of Tanganyika, Kenya, and Uganda, where, as my previous surveys have shown, we have to deal with 215,000 cases.

Already in existence are the two small leprosaria of Walezo and Makondeni, where about 50 cases each are in residence. Of these leprosaria, the use of the sulphone drugs was introduced in Walezo after my visit in February, 1951, and at Makondeni, their use is just being started. Both these leprosaria are controlled by visiting physicians, and resident staff comprises a Catholic sisterhood at Walezo, and a health department assistant at Makondeni.

In small islands like these, there is much more prospect of an attempt at complete control of leprosy being effective within a reasonable time, if the mechanism of control is enhanced and improved. To go on with the present mechanism, even with the use added of the sulphone drugs, success will not be assured. True success will follow when a control officer is appointed for each island, who will gain the confidence of the people, and ensure that each case is found and encouraged to come in. A considerable proportion of the cases are of the moderate type, and readily brought to the stage of arrest of the disease. I do not recommend that cases should be treated at home or by dispensary medication. In such small countries, it is practical and safer to insist on institutional treatment in all cases, whether infectious, or the little infectious tuberculoid and indeterminate cases. Admitted cases have the advantage of the more steady clinical observation and the use of laboratory aids such as the lepromin test and blood examinations, all of which aid success. It is important also that cases should be encouraged, if their clinical state permits, to engage in open-air work, which definitely helps in cure. In domiciliary cases, such work is hard to ensure.

The outline of a plan which I consider will meet the leprosy problem in Zanzibar and Pemba is as follows:—

(1) The sulphone drugs should be stocked in adequate amount and provided free by Government. Ancillary drugs should also be provided.

(2) The existing leprosaria should be enlarged to be able to

contain a total of 150 patients. New patients' houses should be built, and extra African staff attached. Enough agricultural land should be provided to give all patients ample cultivation work, and light industries of the carpentry type provided for. Medical control should be arranged for on the daily attendance basis.

(3) The help of the British Empire Leprosy Relief Association should be sought to provide a BELRA lay worker for each island, the duties of whom would be to help the leprosarium in every way possible, and above all to tour constantly and get to know the island and the people, to gain the confidence of the people, and to arrange the treatment of cases of leprosy, and conduct future survey work.

(4) *A house* to be provided by the Zanzibar Government in a convenient spot near to each leprosarium. This may mean building a new house near Makondeni in Pemba, but in Zanzibar an existing house may be found. One, two, or three miles distance from the leprosarium is suitable.

(5) The provision by the Zanzibar Government of a *motor vehicle* and running costs of same to each BELRA worker.

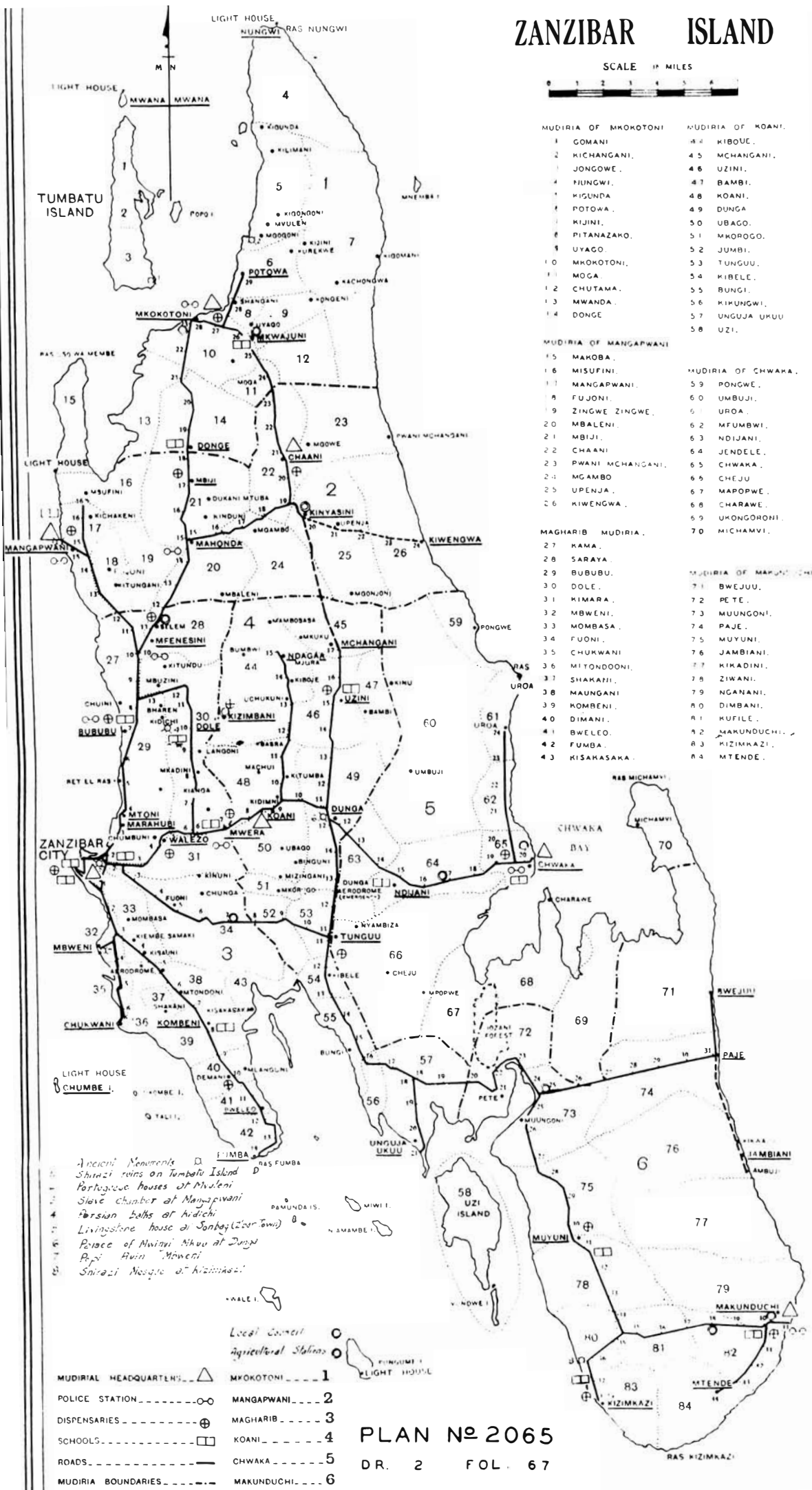
(6) *The spread of the information* also through District Commissioners and Mudirs amongst the people that modern treatment and care for leprosy sufferers are now to be provided, and that they will be welcomed if they come forward. If accommodation is not immediately available, advice on modified home segregation and the benefits of separation from child contacts can be disseminated, not only by the BELRA workers, but by all administrative officers and influential numbers of the community.

(7) The resistance of the Tumbatu people to the leprosy survey in certain places might suggest that a hard core of non-co-operation might spoil the ultimate success of the plan of leprosy control. I suggest that if *good work* is done for those who do come, the ultimate attitude of any possible objectors will be changed to a wish to share in the benefits available, for their people and themselves.

(8) *The infiltration of foreign leprosy cases* needs to be thought of. Some accepted method of inspection of African labour for leprosy must be set up. Every country has the right to inspect visitors and immigrants for leprosy. The chief difficulty is the control of all the coasts for landings from dhows. When public opinion grows, later it might be possible to introduce some plan of inspection. The position as regards these islands is that leprosy exists and owes its origin to imported leprosy in the past. The seasonal importation of labour remains still a potential source of new cases.

ZANZIBAR ISLAND

SCALE 10 MILES



- | MUDIRIA OF MKOKOTONI | | MUDIRIA OF KOANI | |
|----------------------|------------|------------------|-------------|
| 1 | GOMANI | 44 | KIBOUE |
| 2 | KICHANGANI | 45 | MCHANGANI |
| 3 | JONGOWE | 46 | UZINI |
| 4 | MUNGWI | 47 | BAMBI |
| 5 | KIGUNDA | 48 | KOANI |
| 6 | POTOWA | 49 | DUNGA |
| 7 | KIJINI | 50 | UBAGO |
| 8 | PITANAZAKO | 51 | MKOROGO |
| 9 | UYAGO | 52 | JUMBI |
| 10 | MKOKOTONI | 53 | TUNGUU |
| 11 | MOGA | 54 | KIBELE |
| 12 | CHUTAMA | 55 | BUNGI |
| 13 | MWANDA | 56 | KIKUNGWI |
| 14 | DONGE | 57 | UNGUJA UKUU |
| | | 58 | UZI |

- | MUDIRIA OF MANGAPWANI | | MUDIRIA OF CHWAKA | |
|-----------------------|-----------------|-------------------|------------|
| 15 | MAKOBA | 59 | PONGWE |
| 16 | MISUFINI | 60 | UMBUJI |
| 17 | MANGAPWANI | 61 | UROA |
| 18 | FUJONI | 62 | MFUMBWI |
| 19 | ZINGWE ZINGWE | 63 | NDJANI |
| 20 | MBALENI | 64 | JENDELE |
| 21 | MBIJI | 65 | CHWAKA |
| 22 | CHAANI | 66 | CHEJU |
| 23 | PWANI MCHANGANI | 67 | MAPOPWE |
| 24 | MGAMBO | 68 | CHARAWE |
| 25 | UPENJA | 69 | UKONGORONI |
| 26 | KIWENGWA | 70 | MICHAMVI |

- | MAGHARIB MUDIRIA | | MUDIRIA OF MAKUNDUCHI | |
|------------------|------------|-----------------------|------------|
| 27 | KAMA | 71 | BWEJUU |
| 28 | SARAYA | 72 | PETE |
| 29 | BUBUBU | 73 | MUUNGONI |
| 30 | DOLE | 74 | PAJE |
| 31 | KIMARA | 75 | MUYUNI |
| 32 | MBWENI | 76 | JAMBIANI |
| 33 | MOMBASA | 77 | KIKADINI |
| 34 | FUONI | 78 | ZIWANI |
| 35 | CHUKWANI | 79 | NGANANI |
| 36 | MITONDOONI | 80 | DIMBANI |
| 37 | SHAKANI | 81 | KUFULE |
| 38 | MAUNGANI | 82 | MAKUNDUCHI |
| 39 | KOMBENI | 83 | KIZIMKAZI |
| 40 | DIMANI | 84 | MTENDE |
| 41 | BWELEO | | |
| 42 | FUMBA | | |
| 43 | KISAKASAKA | | |

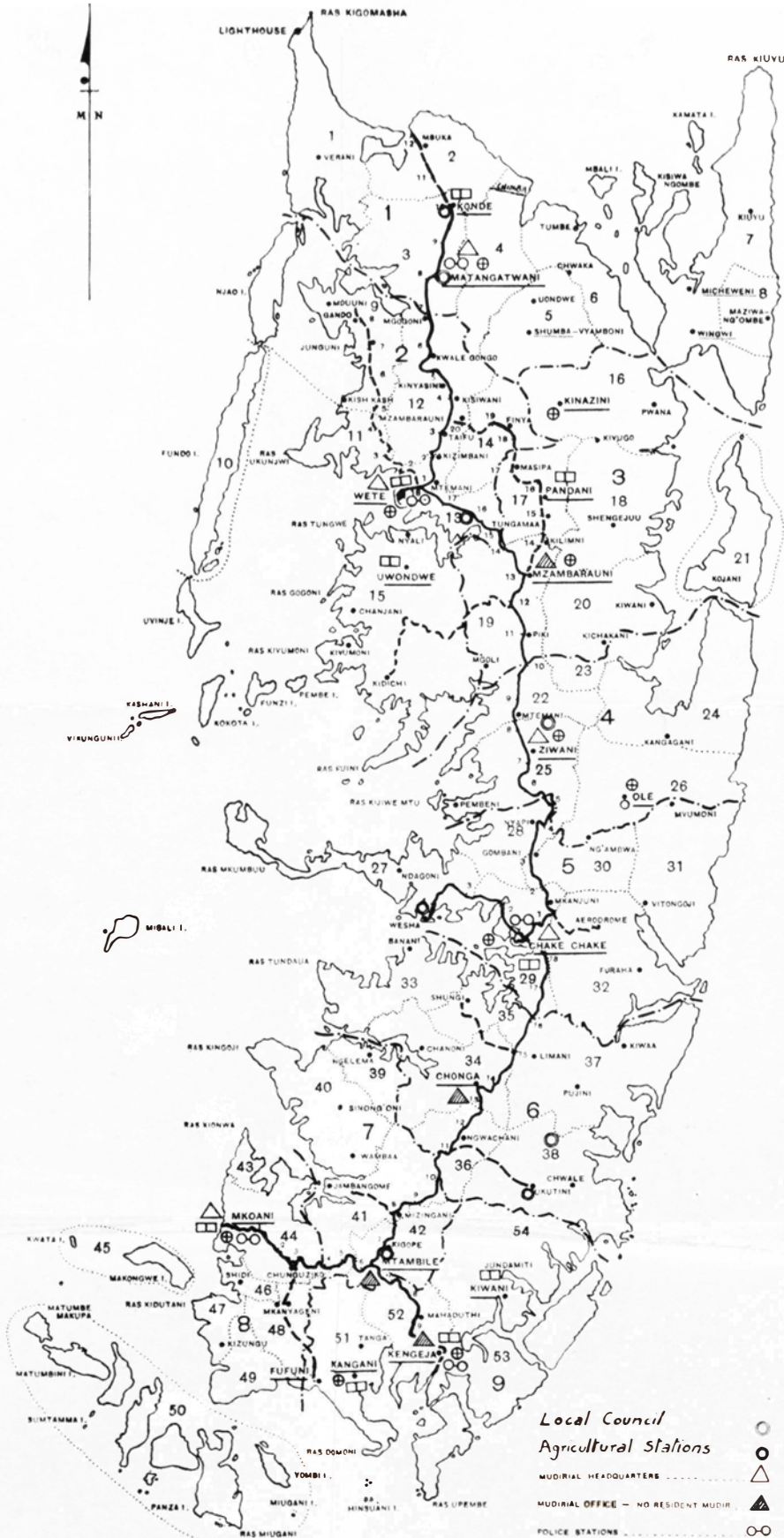
- Ancient Monuments*
- 1 Shirazi ruins on Tumbatu Island
 - 2 Portuguese houses of Mvuleni
 - 3 Slave chamber at Mangapwani
 - 4 Persian baths at Kidichi
 - 5 Livingstone house at Sonbagi (Zor Towi)
 - 6 Palace of Mwinyi Mkuu at Dunga
 - 7 Papi Avin Mkwani
 - 8 Shirazi Mosque at Kizimkazi

- Local Council Agricultural Stations*
- MUDIRIAL HEADQUARTERS: △
 - POLICE STATION: ○
 - DISPENSARIES: ⊕
 - SCHOOLS: □
 - ROADS: —
 - MUDIRIA BOUNDARIES: - - -

PEMBA ISLAND

SCALE

1/2 Inch to 1 English Mile



MUDIRIA OF MATANGATWANI

- 1 VERANI
- 2 MSUKA
- 3 MATANGATWANI
- 4 KINOWE
- 5 SHUMBA-VYAMBONI
- 6 TUMBE
- 7 MICHEWENI
- 8 MAZIWA-NG'OMBE

MUDIRIA OF WETE

- 9 JUNGUNI
- 10 FUNDO
- 11 UKUNJI
- 12 KWALE GONGO
- 13 WETE
- 14 FINYA
- 15 MTAMBWE

MUDIRIA PIKI

- 16 KINAZINI
- 17 MASIPA
- 18 SHENGEJUU
- 19 PIKI
- 20 MCHANGA MDOLO
- 21 KOJANI

MUDIRIA OF KISIWANI

- 22 KISIWANI
- 23 KICHOKOCHWE
- 24 KIUYU
- 25 MIVUNOISHIRINI
- 26 OLE

MUDIRIA OF CHAKE CHAKE

- 27 NDAGONI
- 28 KWALE
- 29 CHAKE CHAKE
- 30 NG'AMBWA
- 31 VITONGOJI
- 32 WAWI

MUDIRIA OF CHONGA

- 33 KILINDI
- 34 CHANONI
- 35 SHUNGI
- 36 NGWACHANI
- 37 PUJINI
- 38 CHAMBANI

MUDIRIA OF JAMBANGOME

- 39 NG'LEMA
- 40 WAMBAA
- 41 JAMBANGOME
- 42 MITATUNI

MUDIRIA OF MKOANI

- 43 MAKOMBENI
- 44 MKOANI
- 45 MAKONGWE
- 46 SHIDI
- 47 MICHENZANI
- 48 MKANYAGENI
- 49 CHOKOCHO
- 50 PANZA

MUDIRIA OF KENGEJA

- 51 FUFUNI
- 52 KENGEJA
- 53 MOWAMBE
- 54 KIWANI

Local Council
Agricultural Stations

- MUDIRIAL HEADQUARTERS
- MUDIRIAL OFFICE - NO RESIDENT MUDIR
- POLICE STATIONS
- DISPENSARIES
- SCHOOLS
- ROADS (EARTH) (METALLED)
- BOUNDARIES (SHEHAL) (MUDIRIAL)

PLAN No 2066

DR. 2 FOL. 68

SUSPENSION OF DIAMINODIPHENYLSULPHONE IN LEPROSY

A. T. Roy.

When the proprietary sulphones proved successful in the treatment of leprosy, attention naturally turned to the use of the parent sulphone, 4:4' diaminodiphenylsulphone (DDS or DADPS) as a possibly more economic method of therapy.

Cochrane (1) started injecting a suspension of DDS intradermally into the macules. Later on, on skin estimation, he found that DDS could be recovered from the skin tissues in which the sulphone was deposited. This finding made him abandon intradermal injections and adopt the subcutaneous injection. He found very favourable results from subcutaneous injections of 25% suspension of DDS in groundnut oil. This report helped all leprologists to get rid of the alarmingly heavy cost of the proprietary preparations. Molesworth and Narayanswami (2), advised by Cochrane, took up the experiment. They used a 20% suspension in place of 25% in purified and deodorized neutral cocoanut oil and reported favourable results in 100 lepromatous cases, after a year's trial.

In August, 1950, Muir selected 170 cases for DDS injection. Out of these, 140 cases, 75 males, 39 females and 26 children, have been included in this report. All of them were lepromatous cases. The plea for writing this is to show the progress of these cases after the completion of 1 year's treatment.

Preparation of the Suspension.

A 20% suspension was made first in hydnocarpus oil and sterilized in an autoclave at 15 lb. pressure for half an hour. A cork with two glass tubes, one straight and long reaching the bottom, and the other short and bent, were fitted to the container of the sterilized suspension. This bottle was often shaken and emptied in a small sterilized glass bowl, in small quantities at a time. From this bowl the syringes were filled by a laboratory assistant who, after drawing in and out the suspension several times, to make the injectable quantities of the same uniformity, handed them over to the injector. Next the hydnocarpus oil was changed to refined cocoanut oil with the same percentage of DDS.

DOSAGE AND METHOD OF INJECTION.

All injections were given bi-weekly and subcutaneously in the

extensor surfaces of arms and thighs in rotation. All the patients, male, female and children, got the same dose of 1 cc (.2 grm.) of the suspension. Observation of reaction, dosage, examination for depot formations and detailed bacteriological examinations were done only by the doctor in charge of the experiment, to ensure accuracy and uniformity of the result.

METHOD OF BACTERIOLOGICAL EXAMINATION AND ASSESSMENT OF PROGRESS.

This was done before the experiment was started, and repeated after 12 months. Five smears were examined from the most infected positive sites of each case on both occasions, and the average result of the five smears from +4 to +1 was taken as the bacteriological index, e.g. the most positive smear was counted as +4 and the least positive was counted as +1, +2 and +3 for less or more midstages. By adding the counts of the 5 smears, and dividing these 5, the bacteriological index was assessed.

“Partly negative” = Those cases who have improved much, but revealed only a few bacilli in all the 5 smears, e.g. (4 in 30 fields).

“Much improved” = This was reckoned when the present bacteriological index was less by 1 or more, e.g. if the initial B.I. was +4 and became less than +3.

“Moderately improved” was given to those just less by +1 from the initial bacteriological index, e.g. +4 to +3, or +3 to +2.

“Slightly improved” = Those whose bacteriological index had improved by less than 1.

“Stationary” and “Worse” are self explanatory.

RESULTS.

(1) Cases males 10, 30, 54, 72 and 103, females 22, 25, 30, 36 and 55 (total 7.1%), have become partly negative after an average of 18.64 and 18.2 gm. of DDS respectively. The maximum improvement is in case F.36. The Bacteriological Index came down from +4 to +4/5ths, and in case Male 103, B.I. from 1+ to 2 bacilli in all the 5 smears.

(2) 37 cases (26.7%) improved a great deal. The Bacteriological Index became less by more than 1. Cases Male 8, 18, 49, 74, 78, 97, and female 4, 9 have improved more than the other cases of this group. The Bacteriological Index became less than 1 only.

(3) 18 cases (12.8%) became moderately improved. The B.I. lessened after 1 year's treatment just by one.

(4) 47 cases (33.5%) improved slightly.

(5) 20 (14.3%) and 16, or 11.4%, remained stationary or became worse respectively.

TABLE 1.

Results and average total dose of DDS in males and females separately.

Advancement.	No.		Total %	Dosage Avg. in Grm.		Period
	Male	Female		Male	Female	
Partly negative ...	7	5	8.5	18.64	18.2	12 months
Much improved ...	17	10	19.3	18.6	19.2	" "
Mod. improved ...	10	8	12.8	19.1	19.7	" "
Slightly Improved	30	17	33.5	18.9	18.1	" "
Stationary ...	16	4	14.3	19.1	19.7	" "
Worse ...	9	7	11.4	19.8	19.7	" "
	89	51				

TABLE 2--see pages 76, 77 and 78.

Observation and Discussion.

SUSPENSION OF DDS POWDER.

Cochrane used groundnut oil, though he has not given any details regarding its purification, acidity etc. It may well be surmised that he used purified and injectable oil. Molesworth et al changed the groundnut oil to the acid free purified cocoanut oil, which was thinner than groundnut oil and could be injected easily, using a medium sized (Gauze 23) hypodermic needle. The author used filtered groundnut oil, hydnocarpus oil and acid free, purified and deodorised cocoanut oil, all sterilised. Groundnut oil was found to be the thickest, and difficulties were encountered specially for mass treatment. Hydnocarpus and cocoanut oil could be used easily.

DEPOTS.

The formation of depots was found in all. Groundnut oil took a longer time to be absorbed, but if massaged well, as has been done here, the number of depots become minimised. It is interesting to note that cases M. 44, 45, 85 and 114 always formed depots after DDS injection, be the suspension in cocoanut, hydnocarpus or groundnut oil.

Doses:—As has been mentioned, 1 cc of 20% (0.2 gm) was all along given twice a week (per week .4 gm), irrespective of age

TABLE 2.

Showing the results of the examinations in details, sex by sex.

PARTLY NEGATIVE Males							PARTLY NEGATIVE Females						
Case No.	Name	Length of treatment in weeks.	Total DDS in grms.	Bacteriological index before treatment.	B.I. at end of period.	No. of days reaction.	Case No.	Name	Length of treatment in weeks.	Total DDS in grms.	Bacteriological index before treatment.	B.I. at end of period.	No. of days reaction.
10	Ronu	39	15.6	+2	$\frac{4}{5}$	—	22	Mano	51	20.4	+2 $\frac{1}{5}$	$\frac{4}{5}$	—
30	Nobin	47 $\frac{1}{2}$	19.4	3 $\frac{3}{5}$	$\frac{4}{5}$	—	25	Sushila	43	17.2	+3 $\frac{1}{5}$	$\frac{4}{5}$	52
54	Opindra	45 $\frac{1}{2}$	18.2	2 $\frac{4}{5}$	$\frac{1}{5}$	15	30	Nishu	50	20.4	+2	3 $\frac{3}{5}$	—
72	Nobin	51	20.4	+1	+ $\frac{1}{5}$	—	36	Kehsori	32 $\frac{1}{2}$	13.4	+4	$\frac{4}{5}$	61
76	Amrit	47	18.8	+2 $\frac{3}{5}$	+ $\frac{4}{5}$	3	55	Jotsna	51	20.4	+2 $\frac{2}{5}$	$\frac{4}{5}$	—
79	Joseph	50 $\frac{1}{2}$	20.2	+1 $\frac{3}{5}$	+ $\frac{1}{5}$	—							
103	Jogu	50	20.4	+1 $\frac{1}{5}$	2 bacilli	9							
MUCH IMPROVED													
8	Khudu	32	12.8	+3 $\frac{1}{5}$	+1	24	4	Subni	46	18.4	+2 $\frac{4}{5}$	+1	31
18	Bokul	20	8.0	+3 $\frac{4}{5}$	+1	31	9	Singho	48	19.2	+2 $\frac{2}{5}$	+1	7
21	Mongol	51 $\frac{1}{2}$	20.6	+4	+2 $\frac{4}{5}$	—	10	Ambika	47	18.8	+3 $\frac{3}{5}$	+1 $\frac{3}{5}$	10
22	Bhim	50 $\frac{1}{2}$	20.2	+3	+1	—	17	Jhabri	49 $\frac{1}{2}$	19.8	+3 $\frac{1}{5}$	+1 $\frac{3}{5}$	—
43	Durjon	45 $\frac{1}{2}$	18.2	+3 $\frac{2}{5}$	+1 $\frac{1}{5}$	18	24	Thelia	45 $\frac{1}{2}$	18.1	+3 $\frac{3}{5}$	+2	21
46	Chuttu	38	19.2	+3 $\frac{4}{5}$	+2 $\frac{1}{5}$	15	31	Kemola	49	19.6	+4	+2 $\frac{4}{5}$	17
49	Sorbo	49	19.6	+4	+1 $\frac{1}{5}$	7	34	Ruou	50	20.4	+4	+2 $\frac{3}{5}$	10
53	Pundee	48	19.2	+3 $\frac{1}{5}$	+1 $\frac{4}{5}$	21	38	Hira	47	18.8	+4	+2 $\frac{4}{5}$	21
62	Horinath	50	20.4	+4	+2 $\frac{1}{5}$	—	42	Sauri	46	18.4	+3 $\frac{1}{5}$	1 $\frac{4}{5}$	49
64	Akhoy	47 $\frac{1}{2}$	19.4	+2 $\frac{2}{5}$	+1 $\frac{1}{5}$	21	50	Jamila	51 $\frac{1}{2}$	20.6	2 $\frac{3}{5}$	1 $\frac{1}{5}$	—
70	Monu	49	19.6	+2	+1	9							
74	Nokul	47 $\frac{1}{2}$	19.4	+3 $\frac{1}{5}$	+1	—							
78	Menon	48 $\frac{1}{2}$	19.4	+2 $\frac{1}{5}$	+1	—							
81	Nimai	49	19.6	+4	+2 $\frac{3}{5}$	—							
86	Gobardhan	50 $\frac{1}{2}$	20.2	+4	+1 $\frac{4}{5}$	—							
97	Bihai	51 $\frac{1}{2}$	20.6	+2 $\frac{1}{5}$	+1	—							
99	Motilal	50	20.4	+4	+1 $\frac{1}{5}$	—							
MODERATELY IMPROVED													
14	Gopal	48	19.2	+3 $\frac{1}{5}$	2 $\frac{1}{5}$	14	26	Indi	50	20.4	+1 $\frac{2}{5}$	+ $\frac{2}{5}$	7
33	Akhey	50 $\frac{1}{2}$	20.2	+3 $\frac{2}{5}$	2 $\frac{2}{5}$	3	28	Rupi	49 $\frac{1}{2}$	19.8	+3 $\frac{3}{5}$	+2 $\frac{2}{5}$	10
35	Konthiram	50 $\frac{1}{2}$	20.2	+3 $\frac{3}{5}$	2 $\frac{3}{5}$	3	35	Mongli	50	20.4	+3 $\frac{4}{5}$	+2 $\frac{4}{5}$	3

45	Thakurlal	...	47	18.7	+2	+1	24	39	Nilmoni	...	42	16.8	+24 $\frac{1}{5}$	+14 $\frac{4}{5}$	9
63	Noru	...	46 $\frac{1}{2}$	18.6	+2	+1	18	43	Babi	...	51	20.4	+13 $\frac{3}{5}$	+3 $\frac{1}{5}$	—
89	Chamru	...	39 $\frac{1}{2}$	15.5	+4	+3	51	49	Taramoni	...	50 $\frac{1}{2}$	20.2	+23 $\frac{3}{5}$	+12 $\frac{3}{5}$	7
95	Biswaneth	...	51 $\frac{1}{2}$	20.6	+13 $\frac{3}{5}$	+3 $\frac{1}{5}$	—	51	Habi	...	49 $\frac{3}{5}$	19.8	+12 $\frac{3}{5}$	+4 $\frac{1}{5}$	7
98	Purna	...	45 $\frac{1}{2}$	18.2	+32 $\frac{3}{5}$	+12 $\frac{3}{5}$	38	52	Khetu	...	51 $\frac{1}{2}$	20.6	+33 $\frac{3}{5}$	+23 $\frac{3}{5}$	—
100	Jogeswar	...	50 $\frac{1}{2}$	20.2	+12 $\frac{3}{5}$	+3 $\frac{1}{5}$	—								
101	Banku	...	49	19.6	+33 $\frac{3}{5}$	+23 $\frac{3}{5}$	—								

SLIGHTLY IMPROVED

1	Radha	...	49	19.6	+4	+34 $\frac{1}{5}$	14	1	Golapi	...	50 $\frac{1}{2}$	20.6	+2 $\frac{3}{5}$	2 granules	3
6	Charu	...	48	19.2	+24 $\frac{3}{5}$	+22 $\frac{3}{5}$	7	6	Alomoni	...	48 $\frac{1}{2}$	19.3	+14 $\frac{4}{5}$	+1	10
9	Rakhal	...	50	20.4	+33 $\frac{3}{5}$	+3	3	7	Sundora	...	46	18.4	+14 $\frac{4}{5}$	+12 $\frac{3}{5}$	17
16	Gulba	...	47 $\frac{1}{2}$	19.0	+12 $\frac{3}{5}$	+1	14	8	Opi	...	48	19.2	+14 $\frac{4}{5}$	+12 $\frac{3}{5}$	17
17	Baneswar	...	49	19.6	+4	+31 $\frac{3}{5}$	—	12	Phudi	...	42	16.8	+2 $\frac{3}{5}$	+1 $\frac{1}{5}$	21
20	Damri	...	47 $\frac{1}{2}$	19.4	+4	+31 $\frac{3}{5}$	14	14	Buchi	...	51 $\frac{1}{2}$	20.6	+1	+1 $\frac{1}{5}$	—
27	Bhuru	...	50	20.4	+33 $\frac{3}{5}$	+33 $\frac{3}{5}$	7	15	Rojoni	...	46	18.4	+4	+33 $\frac{3}{5}$	52
28	Bijoy	...	47 $\frac{1}{2}$	18.9	+31 $\frac{3}{5}$	+24 $\frac{4}{5}$	21	18	Rudun	...	47	18.8	+34 $\frac{4}{5}$	+33 $\frac{3}{5}$	14
32	Sarjoram	...	42	16.7	+4	+34 $\frac{4}{5}$	24	19	Rongi	...	51	20.4	+1	+1 $\frac{1}{5}$	—
37	Haradhan	...	48 $\frac{1}{2}$	19.3	+4	+32 $\frac{3}{5}$	14	23	Phulu	...	51	20.4	+34 $\frac{4}{5}$	+32 $\frac{3}{5}$	—
47	Bhaskar	...	36 $\frac{1}{2}$	14.6	+34 $\frac{4}{5}$	+32 $\frac{3}{5}$	45	27	Binoti	...	49	19.6	+4	31 $\frac{1}{5}$	14
48	Kashinath	...	33	13.2	+4	+31 $\frac{3}{5}$	31	29	Champa	...	47 $\frac{1}{2}$	19.4	+21 $\frac{1}{5}$	13 $\frac{3}{5}$	38
50	Nilamber	...	45	17.9	+11 $\frac{1}{5}$	+4 $\frac{1}{5}$	35	37	Khandhi	...	50	20.4	+11 $\frac{1}{5}$	4 $\frac{1}{5}$	—
51	Balak	...	48 $\frac{1}{2}$	19.4	+11 $\frac{1}{5}$	3 $\frac{1}{5}$	21	46	Dugi	...	46	18.4	+31 $\frac{1}{5}$	24 $\frac{4}{5}$	35
52	Lalmohan	...	35	14.0	+11 $\frac{1}{5}$	4 $\frac{1}{5}$	—	47	Gendu	...	51	20.4	+24 $\frac{4}{5}$	23 $\frac{3}{5}$	—
55	Ghasiram	...	47 $\frac{1}{2}$	19.4	+4	+4 $\frac{1}{5}$	21	48	Adu	...	49 $\frac{1}{2}$	19.8	+22 $\frac{3}{5}$	14 $\frac{4}{5}$	10
60	Menon	...	50 $\frac{1}{2}$	20.6	+3	+22 $\frac{3}{5}$	—	53	Balika	...	37 $\frac{1}{2}$	15.4	+4	—	—

SLIGHTLY IMPROVED

		Males					
67	Horish	...	51	20.4	+13 $\frac{3}{5}$	+1	—
68	Dhonu	...	50	20.4	+12 $\frac{3}{5}$	+1	—
77	Kitu	...	50 $\frac{1}{2}$	20.2	+4	+34 $\frac{4}{5}$	7
80	Purna	...	51	20.4	+14 $\frac{4}{5}$	+1	7
82	Rothu	...	49 $\frac{1}{2}$	19.8	+4	+32 $\frac{3}{5}$	10
86	Ebadot	...	48	19.2	+33 $\frac{3}{5}$	+31 $\frac{3}{5}$	14
90	Dibaker	...	46	18.4	+4	+31 $\frac{3}{5}$	56
93	Suru	...	48 $\frac{1}{2}$	19.4	+2	+14 $\frac{4}{5}$	14
96	Ronjit	...	51	20.4	+12 $\frac{3}{5}$	+4 $\frac{1}{5}$	3
102	Kartik	...	51 $\frac{1}{2}$	20.6	+24 $\frac{4}{5}$	+23 $\frac{3}{5}$	—
107	Sibsankar	...	48 $\frac{1}{2}$	19.3	+32 $\frac{3}{5}$	+24 $\frac{4}{5}$	7
108	Kopildee	...	49 $\frac{1}{2}$	19.8	+4	+34 $\frac{4}{5}$	16
114	Lachmi	...	51	20.4	+2	+14 $\frac{4}{5}$	—

Females

- STATIONARY

Case No.	Name	Length of treatment in weeks.	Total DDS in grms.	Bacteriological index before treatment.	B.I. at end of period.	No. of days reaction.	Case No.	Name	Length of treatment in weeks.	Total DDS in grms.	Bacteriological index before treatment.	B.I. at end of period.	No. of days reaction.
4	Budhu	50½	20.2	+3/5	+3/5	7	3	Rotni	48	19.2	+4	+4	4
15	Dhanonjoy	44½	17.8	+4	+4	42	5	Sashee	50	20.4	+4	+4	—
23	Umesh	49	19.6	+1 1/5	+1 1/5	10	41	Dashi	51½	20.6	+4	+4	—
25	Premasaran	51½	20.6	+1 1/5	+1 1/5	—	44	Chepi	47½	19.4	+4	+4	14
34	Sashee	46	18.4	+4	+4	28							
39	Domon	50	20.4	+1	+1	—							
41	Chuttu	41	16.4	+3	+3	63							
42	Satrugghna	48½	19.4	+2 2/5	+2 2/5	—							
56	Sripeti	40	16.0	+4	+4	66							
59	Doyasagar	50	20.4	+4	+4	—							
69	Budhan	50	20.4	+4	+4	—							
83	Robi	47	18.8	+3 3/5	+3 3/5	14							
85	Ganesh	50½	20.2	+2 4/5	+2 4/5	3							
91	Dinobondhu	49	19.6	+4	+4	—							
92	Domon	50	20.4	+4	+4	—							
104	Nodu	46	19.2	+4	+4	—							

WORSE

11	Rashu	48	19.2	+1 3/5	+3	21	11	Kodom	41	16.4	+2 1/5	+3	52
13	Dulal	50	20.4	+1	+1 1/5	—	15	Bhadu	51½	20.6	+3 3/5	+3 3/5	—
26	Usman	48½	19.4	+3 3/5	+3 3/5	14	16	Subodhani	49½	19.8	neg.	2 bac.	—
29	Phakir	48½	19.4	+2	+2 2/5	21	21	Dongi	48½	19.4	+2 4/5	+3 3/5	14
87	Ismael	47½	19.0	+3 3/5	+3 3/5	10	33	Rosoana	45	18.0	+1 4/5	+3 3/5	45
94	Sashee	51½	20.6	+2 1/5	+3 3/5	—	40	Ulia	49½	19.8	+3 3/5	+4	3
105	Motilal	51	20.4	+3 1/5	3 3/5	—	45	Beji	51	20.4	+4/5	+1	—
110	Joseph	51	20.4	+1 4/5	+2 2/5	—							
113	Sakir	51	20.4	+2 1/5	+3 4/5	—							

and sex. Cochrane used .5 gm. to 2.5 gm. per week in most of his cases. Molesworth used .2 gm. to 1 gm. per week.

LEPRA REACTION.

Eighty-nine cases, or 63.5%, suffered from reaction. Cases F. 53, 25, 11, 15, 33 suffered most from 77, 52, 52, 52, 45 days respectively; of these 3 improved slightly and 2 became worse. Cases M. 56, 41, 90, 47, 15 suffered from 66, 63, 56, 45, 42 days respectively. Three improved slightly, 3 remained stationary and none became worse.

In the reactionary stages while being treated with the parent sulphone, the nodules have been observed as diffuse swellings. Discrete nodules were fewer in acute reaction. In 4 cases the swellings softened and, on incision, thick pus came out. On staining, the specimen contained *M. leprae* and pus cells only. In one case, most of the eruptions ulcerated.

SUMMARY.

One hundred and forty cases have been treated with 20% DDS suspension for 1 year. Groundnut oil, hydnocarpus oil and refined deodorised cocoanut oil have been used as suspending agents. Bacteriological improvements are shown in Tables 1 and 2 in short and in detail, sex by sex: Lepra reaction has been discussed. Depot formation has been observed.

CONCLUSIONS.

One hundred and four, or 74.1% of cases, have improved with a dose of .4 gm. per week for one year. There was very little difficulty and treatment could be continued easily without any supplementary treatment, to almost all. Except in 4 cases out of 89, or 63%, reaction cases needed very little special care for the continuation of treatment. DDS can be suspended easily in any vehicle. Refined hydnocarpus oil costs only two-thirds of the purified cocoanut oil, and depots may be seen with any of the vehicles used for the suspension of DDS, but more particularly when arachis oil is used.

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THICKENING OF THE ULNAR NERVE IN LEPROSY AND ITS TREATMENT

S. N. CHATTERJI.

(Reprinted from "Leprosy in India," Vol. XXIII, No. 1, Jan. 1951)

INTRODUCTION.

Thickening of the ulnar nerve is a well known phenomenon in leprosy. But it is commonly believed that the ulnar nerve is thickened in every case of leprosy. This is not a fact. Patients are sometimes sent to us by medical men with notes of definite diagnosis of leprosy but with an expression of disappointment that they failed to detect any thickening of the ulnar nerve. In those cases there was really no thickening of the ulnar nerve and it was not likely to be thickened as the skin lesions were not in the distribution of this nerve. The reverse is also occasionally seen. That is the nerve is sometimes reported to be thickened when it is not, or it is missed when it is really thickened. This may lead to wrong diagnosis. Another important point is that the detection of thickening of the ulnar nerve has an important bearing in the treatment of leprosy. It is because, if this condition be not detected and properly treated, in due time a patient may be cured otherwise, but he may develop deformities which will remain as a stigma throughout his whole life and, being unable to earn his livelihood, he will remain a burden to the society. Besides that the patient will frequently suffer from blisters and ulcers forming in the affected hand and ultimately there may be loss of digits. This is my apology for introducing this subject.

SITES OF THICKENING OF THE ULNAR NERVE AND ITS RELATION WITH SKIN LESIONS.

This nerve is usually involved when there is anaesthesia or a patch in the ulnar side of the hand. The dorsal branch of the nerve in the hand or near the wrist or the main trunk in the arm may be involved. Sometimes the main trunk is found thickened when there is a patch on the elbow or on the forearm, although these parts are not supplied by the ulnar nerve. The infection is carried to the nerve by lymphatics and this mode of infection is known as the ascending type of infection. Later anaesthesia and deformity may appear in the hand supplied by the nerve. As the main trunk is a mixed nerve and as its involvement causes weakness and deformities of the hand, we shall only refer to the main trunk.

METHOD OF EXAMINATION.

The examiner should stand in front of the patient. The patient's elbow should be slightly bent and the examiner should palpate the nerve trunk first in the sulcus nervi ulnaris and then it should be traced upwards. To verify the presence of thickening, the nerve on the opposite side should be examined and a comparison should be made. It should be remembered that the ulnar nerve is palpable in normal persons. Therefore a nerve should not be considered thickened only because it is palpable. Very rarely the course of the nerve may be found abnormal and it may be found in front of the medical epicondyle. If the examination be not done properly slight thickening of the nerve may be missed or when the nerve is considerably thickened it may be mistaken for a tendon.

PATHOLOGY.

When a nerve is thickened it is swollen. The epineurium is thickened and adherent to the nerve bundles. If the epineurium is incised the affected nerve bundles are found larger in size and pinkish in colour. Nerve fibres are found friable. Yellowish areas indicating caseation may be found in the affected nerve bundles. Thickening of the nerve may be of different degrees. It may be slight, moderate or considerable. Sometimes the thickened nerve is adherent to the surrounding tissues. The nerve may be uniformly thickened or there may be irregular thickening caused by caseation. Sometimes abscesses are found inside the nerve and they may be of different sizes. Big abscesses may sometimes burst through the skin and evacuate spontaneously.

SIGNS AND SYMPTOMS.

The thickened nerve is usually tender on pressure and even on gentle percussion the patient feels tingling sensation passing down the nerve. However, it should be remembered that tingling sensation may be produced by hard pressure on normal nerves. Sometimes the nerve is acutely inflamed and painful which may keep the patient awake.

When the main trunk is involved certain signs and symptoms are noticed in its distribution sooner or later. The patient feels weakness in the hand. There may be difficulty in side to side movements of the fingers, difficulty in writing and putting on clothing, etc. Later the grip becomes weak and as a result of that he becomes more and more unable to carry on with his work. When the condition is acute there may be pain and burning sensation in the hand and the hand may become hypersensitive to touch.

Gradually wasting of the muscles becomes noticeable. The patient becomes unable to straighten the fingers completely, particularly the little and ring fingers and he is unable to spread the fingers

in a fan shaped way. In a further advanced condition there is considerable wasting of the muscles supplied by the ulnar nerve, and more bending of the fingers giving rise to typical claw-hand. Ultimately trophic ulcers may appear and may lead to loss of phalanges or loss of digits.

Usually no steps are taken to prevent the development of deformities when there is a chance of such developments and when deformities are already present they are considered permanent lesions and beyond the chance of any improvement with injections. But it has been found out by careful investigation that deformities can usually be prevented from developing, or when they are already there they can be corrected considerably and the suffering of the patient can be mitigated thereby.

TREATMENT.

The treatment should vary according to the condition of the nerve and of the muscles supplied by the nerve, presence of pain, acute inflammation or abscess formation. It should also be considered whether the nerve pain is due to general lepra reaction. When there is lepra reaction the patient should be treated for that condition with calcium and antimony preparations. With the subsidence of reaction, skin lesions subside and thickening of the nerve goes down, at least to some extent, and the patient is relieved of the pain.

When there is no lepra reaction and when the nerve is slightly thickened and tender, injections of hydnocarpus oil in small doses subcutaneously by the side of the nerve and also in the affected hand are found useful.* Usually this is not done and injections are given only in skin lesions and not by the side of the nerve and when active lesions subside and when there is only slight hypopigmentation, anaesthesia and deformity of the hand injections are either stopped or given only intramuscularly in the buttocks. The result is that gradually there is more anaesthesia, wasting and deformity of the part and frequent blisters and ulcers form, causing endless trouble to the patient. Therefore along with the injections in other parts, injections should also be given in the affected hand and also by the side of the nerve from time to time. The treatment should be continued even when active lesions subside. This should be done not with a view to have any specific action of the hydnocarpus oil but to stimulate the muscles of the hand and reduce the thickening of the nerve.

The patient should be advised to exercise the hand, if possible with a pair of spring dumb-bells and also massage the hand with

* (This possibly acts as a form of counter-irritation, and must be applied with great care.—EDITOR)

some oil daily for 1 hour. Kneading of the muscles, rubbing of the fore-arm and hand from above downwards and pulling of the fingers are necessary.* The hand should be protected from too much heat and cold. Therefore the patient should be advised not to touch anything hot and avoid going near a fire and also use woollen gloves in cold weather. Usually these measures are a valuable aid to the prevention of deformities and ulcerations. Even cases showing slight signs of deformities may benefit by this treatment.

When deformities are more advanced or when there is nerve abscess or acute nerve pain, not relieved by other methods, decapsulation of the nerve should be done without delay to prevent further deformity. Subsequently routine treatment, massage and exercise etc. as described before should be followed to correct the residual deformity and weakness etc. not being corrected by operation alone. Splinting of the hand is also another useful measure. In many cases encouraging results have been obtained by following this combined treatment. There was complete relief of pain in the majority of cases. In some cases deformities were entirely corrected and there was complete return of strength. In other cases there was partial improvement, and further deterioration, appearance of trophic ulcers and loss of digits were prevented. In most of the cases there was some return of sensation and therefore the incidence of accidental burn and blister formation became less and less. In a small number of lepromatous cases who suffered from repeated reaction this method of treatment gave only temporary relief. But subsequent reaction made their condition worse.

Decapsulation or removal of the sheath of the nerve can be done under local anaesthesia. The nerve is situated behind the intramuscular septum which should be incised. Then the epineurium should be incised and dissected off carefully all round the thickened portion (2 to 4 inches) of the nerve in the arm. The rationale of this operation is that the nerve sheath becomes very dense and unyielding and causes pressure on the nerve fibres with the resulting severe pain and later there is degeneration and atrophy of the nerve fibres and of the muscles supplied by them. Subsequently there may be loss of digits which makes the patient completely crippled. Removal of the sheath of thickened nerve relieves tension inside the nerve bundles and thus prevents the unhappy sequence of events. But we should not depend upon this operation alone. This is only a part of the treatment. It should be combined with routine treatment of leprosy, massage and exercise of the affected hand.

*The method of massage and splinting is detailed in Dr. Branil's article (p.51).

ILLUSTRATIVE CASES

(a) Weakness or deformities corrected by injections, massage and exercise.

- (i) P.K., Case No. 7521.
Early neural case.
Duration 6½ years.

Before treatment

24.9.43.

Lesion on the rt. little finger. Rt. ulnar + + + (caseous). Pain in rt. ulnar and rt. hand. Weakness in rt. hand. Difficulty in writing.

- (ii) R.D. Case No. 8658.
classification doubtful.
(NS₉a₉+ ? L).

Before treatment. Fig. 1 (a)

20.7.48.

Rt. ulnar + +

Rt. little, middle and ring fingers are bent. Difficulty in writing. Weakness in rt. hand. Difficulty to raise heavy things. Feels shy to go out on account of the deformity.

- (iii) G.R., Case No. 8271.
Early neural case.
Duration 4 years.
Machine assistant.

Before treatment.

11.6.46.

Rt. ulnar + +

Pain in rt. hand. Difficulty in writing. Difficulty in doing manual work. He was selected for decapsulation failing improvement with injections, massage, etc.

After treatment

12.1.45.

Lesion faded completely. Muscular strength considerably restored. Slight return of sensation. Thickening of the ulnar nerve less than before.

4.3.9.

Lesion faded. Sensation restored in the dorsum of the rt. hand but slight anaesthesia present in the palm. No pain and no weakness in rt. hand.

After treatment Fig. 1 (b)

22.2.49

Out of four fingers two fingers are now bent. (Rt. little and ring fingers bent.)

24.1.50.

Very slight bending of rt. little and ring fingers and therefore he can now go out without raising suspicion and without feeling shy. No difficulty in writing. Hand writing has improved. He can grip properly and raise heavy things. Lesion faded considerably and there is some return of sensation.

7.1.50.

Lesion faded, Rt. ulnar less thick. Pain subsided. Return of strength in rt. hand. Some return of sensation. Therefore decapsulation was not done.

After treatment.

- (iv) M.M., Case No. 8526.
Early neural case.
Duration 2½ years.

Before treatment.

18.10.47.

Lt. ulnar + +

Pain in left ulnar and in left hand. Weakness in left hand and therefore difficulty in manual work, difficulty in putting on clothing and in ablutions after defaecation.

- (v) R.P.S. Case No. 8691.
Neural case, moderately advanced.
Duration 2 years. Draftsman.

Before treatment.

21.9.48.

Left ulnar + + +

Pain in left ulnar, Pain and burning sensation in left hand. Weakness in left hand and therefore could not grip with the left hand or straighten the fingers properly. Therefore he felt it difficult to do the work of a draftsman.

- (vi) S.M. case No. 8990—Fig. 2 (a)
Neural case.
Duration 5 years 8 months.

Before treatment.

23.11.49.

Slightly red, thick and anaesthetic lesion covering the left hand, forearm and arm. Left little, ring, middle and index fingers are bent. Left ulnar and left superficial radial nerves thickened.

Weakness and deformities corrected by decapsulation combined with other treatment.

- (i) Sk. H., Case No. 7403.
School Teacher Early neural case.
Duration 6½ years.

Before treatment.

7.5.43.

Rt. ulnar + +

(caseous)

Pain in rt. ulnar and rt. little finger. Rt. little finger bent. Loss of strength in rt. hand. Difficulty in writing. Could not grip.

25.5.43.

Decapsulation done.

After treatment.
Present condition after treatment.

Left ulnar still thick. Pain relieved. Normal strength in left hand. No difficulty in doing any work.

After treatment.

10.1.50.

Lesions subsided. Pain in the nerve and left hand relieved. Increased strength in left hand. He can now grip with the left hand and straighten the fingers properly. Therefore there is not much difficulty in doing his work.

After treatment. Fig. 2 (b)

4.7.50.

Lesion subsided considerably. It is now hypopigmented and flat. Sensation restored in some places. Excepting the left index finger which is still slightly bent other fingers have become straight. Increased strength in left hand. Left ulnar and left superficial nerves less thick.

After treatment.

15.4.44.

Nerve less thick.
More strength in rt. hand. Rt. little finger less bent.

30.1.50.

No pain in rt. ulnar and rt. little finger. Deformity of the right little finger corrected considerably. Return of strength in rt. hand. No difficulty in writing and the grip is normal. Slight wasting of muscles present but further deformity prevented.

ILLUSTRATIVE CASES—*Contd.*

- (ii) S.D., Case No. 5088.
Motor Car Driver.
Early neural case.
Duration 14 years.

Before treatment.

19.8.35.
Left ulnar + +
Weakness in left hand. Difficulty in driving motor car.

22.8.35.
Injections started.
28.1.39.
Slight deformity of left hand noticed.

After treatment.

28.1.39.
Decapsulation done.

30.1.50.
Lesion faded. Considerable return of sensation. Almost normal strength in left hand. Slight wasting present. No difficulty in driving motor car.

- (iii) B., Case No. 7950.
Early neural case.
Duration 5 years.

Before treatment. Fig. 3 (a)

8.5.45.
Rt. ulnar + + +
Pain and tingling in rt. hand and rt. ulnar. Weakness in rt. hand. Rt. little, ring and middle fingers bent. Difficulty in putting on clothings. Difficulty in doing manual work. Could not take food with his rt. hand. Used to feed himself with his left hand.

17.5.45.
Injection started.
25.5.45.
Decapsulation done.

18.1.46.
Lesion faded.
Rt. middle and ring finger are now straight.
Slight bending of the rt. little finger.
No pain.

13.6.47.
More strength in rt. hand.

After treatment. Fig. 3 (b)

24.9.49.
Further improvement. All the three fingers have become straight. Therefore correction is complete. No difficulty in doing work.

(c) Improvement noticed soon after decapsulation.

- (i) G.R.M., Case No. 8998.
Moderately advanced neural case.
Duration 8 months.
A girl of 15 years.

Before treatment.

25.11.49.
Rt. ulnar + + +
Right little, ring, middle and index fingers bent. Could not grip or do any work or feed herself. Could not even raise a glass of water. Could not write. Pain in rt. hand and rt. ulnar.

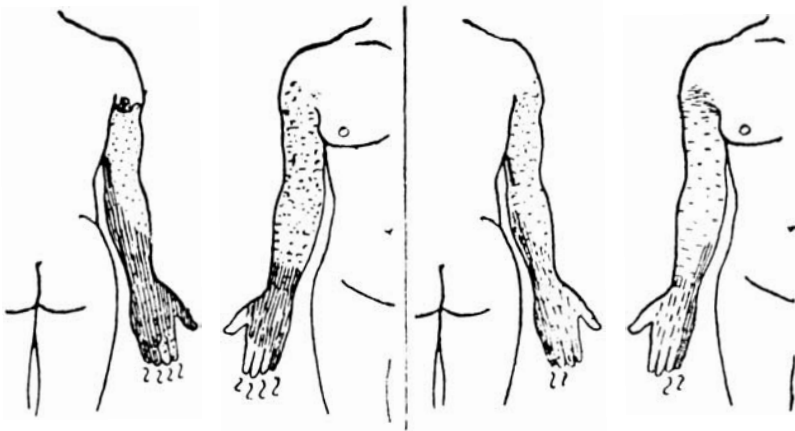
After treatment.

13.12.49.
Decapsulation done.

1.2.50.
Deformity in rt. hand less. Fingers not so bent as before. Some return of strength in rt. hand. She can now grip and do some work, although the grip is not so strong. No pain and tingling sensation in rt. hand and in rt. ulnar. She can now write, feed herself and raise buckets full of water with her right hand.

R. D. Case No. 8658. Figure 1 (a).

R. D. Case No. 8658. Figure 1 (b)



Before Treatment.

After Treatment.

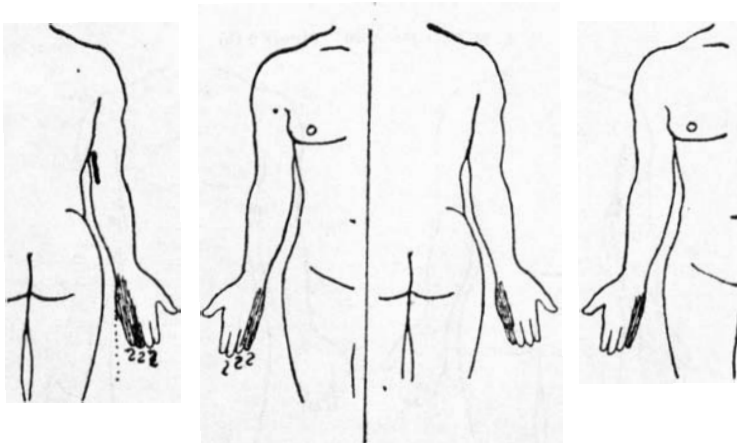
N. B.—Symbols used for different signs:—

- | | | | | | |
|-----------------------------|---|---------------------|----|----------------------|-------|
| (1) Anaesthesia | | (2) Bent fingers | ∪∪ | (3) Hypopigmentation | ===== |
| (4) Erythema and thickening | ■ | (5) Thickened nerve | — | | |

B, Case No. 7950.

Figure 3 (a).

Figure No. 3 (b)



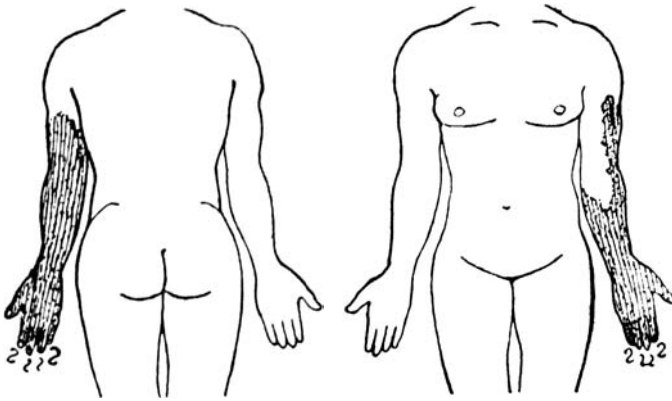
Before Treatment.

After Treatment.

N. B.—Symbols used for different signs:—

- | | | | | | |
|-----------------------------|---|---------------------|----|----------------------|-------|
| (1) Anaesthesia | | (2) Bent fingers | ∪∪ | (3) Hypopigmentation | ===== |
| (4) Erythema and thickening | ■ | (5) Thickened nerve | — | | |

S. M. Case No. 8990. Figure 2 (a)

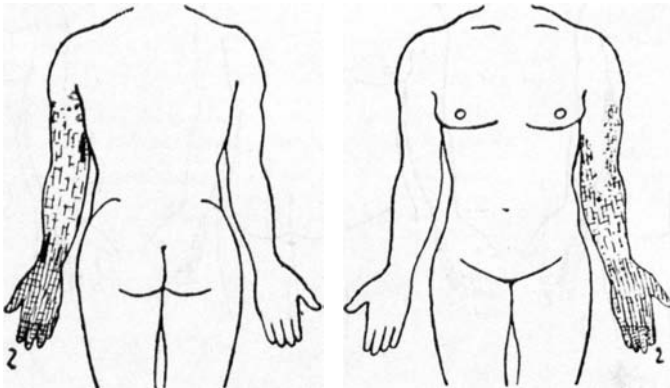


Before Treatment.

N. B.—Symbols used for different signs :—

- | | | | | | |
|-----------------------------|---|---------------------|----|----------------------|-------|
| (1) Anaesthesia | | (2) Bent fingers | ∪∪ | (3) Hypopigmentation | ===== |
| (4) Erythema and thickening | ■ | (5) Thickened nerve | — | | |

S. M. Case No. 8990. Figure 2 (b)



After Treatment.

N. B.—Symbols used for different signs :—

- | | | | | | |
|-----------------------------|---|---------------------|----|----------------------|-------|
| (1) Anaesthesia | | (2) Bent fingers | ∪∪ | (3) Hypopigmentation | ===== |
| (4) Erythema and thickening | ■ | (5) Thickened nerve | — | | |

CONCLUSIONS.

Deformity of the hand is a serious complication of leprosy as it may make the patient an invalid and unable to earn his livelihood. Therefore it should be the endeavour of all leprosy workers to prevent this complication or try to correct it as much as possible by all means. As the claw hand is caused by involvement of ulnar nerve the pathology of the process should be clearly understood to administer proper treatment. In this paper we have emphasised the benefit which may be derived by giving injections of hydnocarpus oil, massage, exercise and decapsulation etc. Other methods of treatment like diathermy application, stimulation of muscles by galvano-faradic current, massage by specialists or special baths, have not been discussed here because, although useful, only a small number of patients in big institutions can have the benefit of these treatments. The methods advocated in this article are simpler, do not require any special apparatus or a specialist, and can be easily carried on in any outdoor clinic. If properly carried on with patience and perseverance for a long time patients will benefit considerably as will be evident from the cases illustrated here.

REVIEWS

Leprosy in India. Vol. XXIII. No. 1. January, 1951.

This is a special issue devoted to the Third All-India Leprosy Conference held in Madras in October, 1950. The Conference was called by *Hind Kusht Nivaran Sangh*, the body which has taken the place of the Indian council of the British Empire Leprosy Relief Association. The latter was in existence for 25 years, and an interesting paper was read describing the work done and the progress made during that period. The previous two conferences were held at Wardha in 1947 and at Calcutta in the end of 1948. The subject that was most stressed at the conference was the need for anti-leprosy workers who will be willing to live in the villages and work among the people. Without this even the new treatment with the sulphones will not bring leprosy under control. Much time was given to discussion of the sulphones, and especially of the relative toxicity of DDS. Those with most experience of this drug

were able to show that in small non-toxic doses it is as effective as when larger amounts are given.

Another subject discussed was *Rehabilitation of Patients*, and a lecture given on plastic surgery by Dr. Bland was of special interest.

A paper which raised a good deal of interest was on *Positive Bacillary Findings in Neural Leprosy*. Using a special method for examination, 39.7 per cent of neural cases were found to be positive on the first occasion, and of those found negative 48.1 per cent. were found positive after an average period of three years. Possibly this unusually large number of positives depends to a certain extent on the ambiguity of the word "neural."

The report of the conference is given very fully; it is full of items of interest, and is worthy of careful study by all who are interested in the subject of leprosy in India. E. MUIR

Leprosy in India. Vol. XXIII. No. 2. April, 1951.

Of the two original articles, the first, by Dr. Muir, on *Bacteriological Changes under DDS Treatment of Leprosy*, has already been reprinted in the last number of Leprosy Review.

The second article is a review of the three All-India Conferences that have been held in 1947, 1948 and 1951. It traces the indigenous movement to deal with leprosy both in its medical and sociological aspects, taking its origin from the interest and impetus given by Mahatma Gandhi. Another sign of rising interest was the formation of "The Indian Leprologists' Association" at the last of these conferences held in Madras. E. MUIR

Leprosy in India. Vol. XXIII. No. 3. July, 1951.

Treatment of Leprosy with Novotrone, by Drs. Dharmendra, S. N. Chatterji and N. Sen. Novotrone is a sulphone with a chemical composition similar to that of sulphetrone, and it is produced in India. To 15 cases the drug was given intramuscularly and to 12 cases orally. After an average period of 8 months trial the authors report as follows: "As judged by laboratory tests and by the results of treatment of patients with leprosy, novotrone appears to be similar to sulphetrone. Novotrone is quite effective in the treatment of leprosy, and is free from toxic effects. Its continuous use for 8 to 12 months has not produced any appreciable fall in the haemoglobin and R.B.C. values of the blood. It can be given by mouth in daily doses of 1 to 3 gm. or by intramuscular injection of a 50 per cent watery solution twice weekly in doses

of 0.5 to 2 gm. As in the case of sulphetrone, oral treatment is not economical because of the poor absorption of the drug from the intestines. While definite clinical improvement has been produced within a year, bacteriological improvement has been less evident. It may be said that novotrone shares both the advantages and limitations of the other sulphones in the treatment of leprosy.

A Review of the Relative Activity of the Sulphones by Dr. J. M. Mungavin. The relative potencies of the five sulphones: DDS, 2196, Diasone, Promin and Sulphetrone, when given orally to mice against streptococci and other organisms, were respectively 100, 43, 18, 16, 1.

From this and other findings it is concluded that: (a) the soluble sulphone derivatives are partly converted to DDS, probably in the stomach, before being absorbed, and that their therapeutic action reflects their degree of conversion to DDS and the blood concentrations of free DDS; (b) the soluble derivatives are partly converted to DDS, probably in the stomach before being absorbed. The therapeutic action of a soluble derivative depends on the degree of conversion to DDS and the blood concentrations of free DDS produced. When sulphetrone is given intravenously it is almost all excreted very rapidly in the urine and can have very little therapeutic action. The same applies in varying lesser degrees to the other soluble derivatives; (c) 2196 is the only one that is converted to DDS in appreciable amounts in the biological fluids. Indeed it is the only one almost quantitatively converted to DDS *in vivo*. It is therefore likely to be useful in cases where parenteral administration is necessary.

DDS is both effective and cheap, and its manufacture is not protected by patents. It is to be preferred therefore to the more expensive proprietary derivatives. E. MUIR

Leprosy in India. Vol. XXIII. No. 4. October, 1951.

Differentiation of Human and Rat Leprosy Bacilli by Irradiation by Dr. A. Mukerji.

The author found that smears of *Myco. leprae*, when irradiated for five hours in sunlight, in ultraviolet rays for two hours or for half an hour in roentgen rays (42r) from a 150 K.V.P. plant in some cases lost its acid-fast staining property entirely or appeared beaded with alternate white and red bands. Under the same conditions rat leprosy bacilli are not similarly affected.

Lepromatous Leprosy with Exclusively Localized Macular Lesions. Eight cases were examined and followed up over a period of

years. When first seen all the cases had purely localized macular lesions which were clinically not typical of the lepromatous type. However later after varying periods the lesions became generalized and typically lepromatous. As judged by the results of repeated bacteriological, immunological and histological examinations it would appear that the lesions were lepromatous even when first seen. It can be concluded that in certain cases of the lepromatous type the lesions may remain exclusively localised for a considerable period before becoming generalized, and may during that period simulate tuberculoid lesions in being well circumscribed, the presence of anaesthesia and thickened nerves making the resemblance all the more marked. They are however smooth, soft and succulent, their edges are not as clear cut, bacteriologically they are strongly positive, and the lepromin test is negative. The histological findings may not be definite in the early stages.

E. MUIR

International Journal of Leprosy. Vol. 18 (1950) July-Sept.

Preliminary Report on 4:4' Diaminodiphenylsulphone (DDS) Treatment of Leprosy—Ernest Muir.

In this paper the writer describes the results of a year's trial of the parent sulphone DDS on 94 patients at the Purulia leprosy home, India, commencing April, 1949. The ordinary uncomplicated case—lepromatous or tuberculoid—was found to tolerate 4 mgm/kilogram body weight given orally as a 2.5% suspension. Two important signs of intolerance were anaemia and lepra reaction. If reactions occurred treatment was suspended for 2-3 weeks. As a rule patients were better after each reaction. There was rapid clinical improvement in the majority of patients, especially in the healing of ulcers, blocked nose, eye conditions and lepra reactions. Bacteriological improvement was slow. He believes that in DDS we have an effective, easily administrable, and, with reasonable precautions, safe drug.

Studies of the Absorption, Excretion and Distribution in the body of the Sulphones used in the Treatment of Leprosy.—Sister Hilary Ross.

In this study of a small group of patients in Carville who had had sulphone therapy for from 4 months to 7 years, the absorption, excretion and distribution of promin, diasone, promacetin and sulphetrone is reported. It was found that the drugs were retained in the body up to 14 days after cessation of treatment and occasionally as long as 4 weeks. There are little differences in distribution

regardless of whether the sulphone is given orally, as with diasone, promacetin and sulphetrone, or intravenously as with promin. Diasone, promacetin and sulphetrone are not completely absorbed. Approximately 50% of sulphetrone is absorbed even after one year's therapy. Excretion via the kidney is relatively rapid. The skin concentrations were about the same in most cases, whether the drug was given orally or intravenously. Post mortem studies showed that the liver, spleen, kidneys, skin and nerves serve as organs of storage of the sulphones.

Effects of Sulphetrone Treatment in Fiji—Austin, C. J.

A report on the treatment of 444 patients—317 males and 127 females—with sulphetrone. Amongst the various racial groups the Indians showed the most marked improvement. The most striking result was the emptying of the wards of all the familiar chronic ulcerated lepromatous patients. 413 of the patients treated were lepromatous and 31 tuberculoid. Sulphetrone was found to be markedly toxic when full doses (3.0-6.0 gms) were given daily in spite of one week's rest in four. Forty-five patients were unable to stand the full dosage because of lepra reactions or desquamative dermatitis. Some patients who did well for six months began to get reactions from which they found it difficult to recover. In spite of these drawbacks the writer feels that results have been highly gratifying.

Visceral Tuberculoid Leprosy—Jorge Campos, R. de C., and Marino Molina, S.

Sections of liver were obtained by laparotomy from 7 patients with tuberculoid and undifferentiated types of leprosy. Histological sections were studied. In all the 5 tuberculoid cases and one of the 2 undifferentiated, characteristic tuberculoid follicles were found which the writers attribute to the action of leprosy bacilli. 'The existence of these visceral granulomas in tuberculoid cases,' they consider, "permits the conclusion that in this type of leprosy the infection is not confined to the skin, nerves and superficial lymph nodes, as has been maintained by almost all authors.'

Leprosy and Leprosy Work in East Africa—Innes, J. R.

Eight leprosy surveys were made by the writer in Uganda, Kenya and Tanganyika during the 3 years 1947-1950. Out of 361,943 people examined, 6,107 cases of leprosy were found, i.e. 16.8 prevalence per thousand, giving an estimated total of 215,210 cases. Only 20% were lepromatous.

A Note on Leprosy in Liberia—Poindexter, H. A.

' There is no hospital in Liberia set aside for the treatment of leprosy, and segregation is not enforced. There are, however, three leprosy colonies or villages with 200-300 residents, and several smaller stations where patients may come for treatment once or twice a week.'

Reactions to Tuberculins in Leprosy—A Review. Wade, H. W.

In his summary of this long and careful review the author says

" When Koch's Old Tuberculin was given by subcutaneous injection in treatment, it often induced lepra reaction. It is not known whether they were tuberculoid cases which reacted in that way, or whether tuberculin can by non-specific effect induce reactions in lepromatous cases which lepromin cannot provoke. Diagnostic skin tests employing Old Tuberculin by the von Pirquet method have given results which afford no evidence that leprosy infection may give rise to false positive reactions. In all cases where control data on normal-population groups are given, the results are closely comparable. The results of diagnostic tests by the Mantoux method with O.T. are mostly of like tenor. When purified protein derivatives have been used in the Mantoux test there is evidence of a tendency to lowered frequency of reaction in lepromatous leprosy than in other forms. These products are less prone to cause non-specific reactions than is O.T. In lepromatous cases which have recovered there is a suggestion of a tendency to recover, of activity to various antigens. The indications seen in one report that children living in contact with lepromatous cases may be more reactive to tuberculin than contacts of tuberculoid cases is also of interest. Further investigations with various antigens in varying doses and in different ways might be profitable."

G. O. TEICHMANN.

International Journal of Leprosy, Vol. 18 (1950) Oct.-Dec.

Thiosemicarbazone (TB1) in the Treatment of Leprosy. Preliminary Contribution—Vegas, M., Convit, J., Medina, J. A. and de Blomefield, E.

This preliminary report on the use of conteben in the treatment of 42 patients with lepromatous leprosy for 3-6 months indicates that the drug has marked therapeutic activity. All the patients showed clinical improvement. No manifestations of intolerance were noted, apart from lepra reactions which subsided after reduction of dose. The daily dosage for adults—beginning with 25 mgm and increasing weekly by 25 mgm. The maximum daily dose received by one patient was 900 mgm (18 tablets). Children began with 5 mgm and gradually increased to 25 mgm.

The Sulphone Treatment of Tuberculoid Leprosy—Lowe, John.

The writer considers that sulphones constitute the therapy of choice in tuberculoid as well as lepromatous leprosy. This report is on 50 cases of tuberculoid leprosy with active lesions. After a preliminary phase of focal reactions in the skin lesions, signs of activity slowly subsided, and usually within 6 months the lesions became inactive and "residual." Thickening, tenderness and pain in nerves subsided much more slowly.

G. O. TEICHMANN.