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LEPROSY REVIEW.

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CONTENTS

Edited by DR. E. MUIR, Hon. Acting Medical Secretary of the British Empire Leprosy Relief Association, 8 Portman Street, London, W.1, to whom all communications should be sent. The Association does not accept responsibility for views expressed by the writers.

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OBITUARY

We announce with great sorrow the death of Dr. John Lowe. It is little more than a year since he left Nigeria and became Medical Secretary of B.E.L.R.A. Among his duties was the editorship of this journal. Dr. Lowe's long medical experience of every form of anti-leprosy work made him an ideal man for this post, and as far as impaired health permitted, he threw himself enthusiastically into all the activities of the Association.

Born in Birmingham in 1895, after service in the first world war, in which he was awarded the M.C., he studied medicine at Birmingham University, graduating in 1922. In the following year, he joined the Methodist Missionary Leprosarium at Dichpalli, Nizam's Dominions, where he worked for 8 years. In 1931 he was invited to join the Leprosy Research Department at the Calcutta School for Tropical Medicine. There he worked for some 15 years, chiefly at leprosy research, but during the second world war he devoted himself to more general medicine, editing the Indian Medical Gazette, and acting as Professor of Tropical Medicine and Director of the School.

While in Dichpalli he became Doctor of Medicine with a thesis on malaria. Later he was awarded the K.I.H. gold medal for his work on leprosy.

In 1946 Lowe returned to England, where after some further study he obtained the M.R.C.P. In the following year he was appointed Director of the Leprosy Research Unit at Uzuakoli in Nigeria, being encouraged to renew his leprosy work by the reports of success with the sulphone drugs. He was the first to introduce the oral treatment of leprosy with diaminodiphenylsulphone (DDS), and after preliminary trials he arranged for mass treatment throughout Nigeria with this comparatively simple and inexpensive drug, which is now regarded almost universally as the treatment of choice.

While in Nigeria, Lowe was awarded F.R.C.P. and last year the C.B.E. In April, 1954, he returned to England, having been offered the post of Medical Secretary to B.E.L.R.A. While still in Nigeria, there had been warnings of heart trouble, and this increased during the closing months of last year, making it

inadvisable, to his great regret, for him to undertake a visit overseas to acquaint himself at first hand with the work of B.E.L.R.A. in East Africa.

Dr. Lowe had the true scientific spirit, critical of his own work, he was slow to come to conclusions. But once he had taken up a project, he threw himself wholeheartedly into its solution, though often facilities were scanty and he had to improvise and work in what many would have considered impossible conditions.

Leprosy has so far attracted but few first class research workers, and the loss of Lowe has removed one of the best of these few. It is hoped that his life and example will act as a call to young medical graduates to enter this very needful and now much more hopeful line of research.

. . . . . .

Until a permanent successor to Dr. Lowe is appointed, Dr. E. Muir, Hon. Medical Advisor, will discharge the duties of the Medical Secretaary of B.E.L.R.A., including editing of "Leprosy Review." He will be glad to receive useful and interesting contributions—original articles, news items, correspondence, etc.—for publication.

ISONIAZID ALONE AND COMBINED WITH THIOSEMICARBAZONE

W. S. DAVIDSON, M.B., CH.B., D.P.H.

In a previous paper[1] I described how a small series of cases improved at the end of six months on INH; improved at a slower rate for the next three months and then deteriorated when the INH was combined with sulphetrone. These cases improved again when put on INH alone.

Subsequent follow-up has, however, indicated that this improvement was not maintained. Consequently, a further series of 11 cases has been tested on INH alone. At the end of six months the group improvement index, calculated by awarding marks for clinical and bacteriological improvement as previously described[2] was 0.36. By the end of twelve months, however, this index had fallen to 0.18. Three cases had improved, three were worse than when treatment started and five had not altered.

To whatever extent conclusions are justified on such small series of cases, it would appear that INH produces its maximum improvement between the sixth and ninth month of treatment and

thereafter the cases deteriorate, presumably from the development of resistance. The onset of resistance may be delayed by the exhibition of other anti-leprosy drugs.

As a pilot series of INH and Thiosemicarbazone[1] had shown more promise than INH and sulphetrone, 47 cases were put on the combined treatment of INH and Thiosemicarbazone (Neustab).

These 47 patients were all suffering from lepromatous leprosy of considerable duration. They had all had previous extensive treatment with sulphones and some also with thiosemicarbazone. They had reached a stage of chronicity in their disease where no further improvement appeared to be taking place.

The result of ten months' treatment with combined INH and Thiosemicarbazone (Neustab) is shown in the table.

The two fatal cases have been ignored when calculating the index of progress. They died during a severe influenza epidemic and the mortality in the series was not significantly different from the general mortality in the leprosarium at that time. Their deaths were not in any way attributable to the treatment and they were improving up to the time of their fatal illness.

Considering the chronicity of the disease in all cases and the lack of further improvement in previous treatments, the index of progress of 1.42 must be considered as very good. Eleven cases became bacteriologically negative, a further 22 cases showed improvement, ten cases remained static and two cases deteriorated. Several cases shown as static in actual fact show improved smears but the improvement is not yet sufficient to be recorded in our counting system.

It will be noted that the improvement in cases not previously treated with thiosemicarbazone is slightly greater than those so previously treated. An element of the total improvement is therefore due to the Neustab alone, but there is a substantial improvement in the cases previously treated with Neustab suggesting that the combination of INH and Neustab had remedial properties over and above the mere summation of the effects of the individual drugs.

The daily adult dosage was 350 mg. INH and 200 mg. Neustab. This dosage was reached in suitable cases in 2 months. It was given for six days a week and there was a week's rest after every two months' treatment.

The dosage, however, requires careful adjustment to the reaction of the patient and can only be given to patients under constant supervision. In all, 22 cases suffered from toxic or reactive phases requiring a break or modification of treatment.

The main clinical impressions are that INH increases the incidence of neuritic reactions and thiosemicarbazone produces an anaemia which is slower in recovery than that associated with sulphone treatments.

In the 22 toxic cases, there were eight cases of neuritis and fourteen of anaemia. Several also suffered from dermatitis, albuminuria, pyrexia, and erythema nodosum.

Toxic manifestations occasionally required cessation of treatment but in general modification of dosage was sufficient.

SUMMARY AND CONCLUSION

The present paper continues a previous examination and report on the use of INH alone and in combination with other anti-leprosy drugs.

There are indications that INH alone reaches its maximum usefulness between the sixth and ninth month of exhibition and thereafter cases fail to improve, or deteriorate.

INH and sulphetrone in a small series did not show good results.

INH and thiosemicarbazone (Neustab) in a series of 47 cases gave very promising results over a 10-month period.

INH plus thiosemicarbazone in full dosages may give rise to toxic manifestations. This treatment should therefore be given in an institution under medical supervision.

TABLE SHOWING RESULT OF TEN MONTH'S TREATMENT OF INH AND NEUSTAB

Previous Treatment	No. of Cases	Became Negative	Improved but still Positive	Not Improved	Deteriorated	Died	Index of Progress*
Sulphones and Neustab	15	4	5	6	0	0	1.2
Sulphones only ...	32	7	17	4	2	2	1.53
Total	47	11	22	10	2	2	1.42

* The two fatal cases omitted from Index.

ACKNOWLEDGEMENTS

The above patients are inmates of Derby Leprosarium. To Dr. Peter Graham, Mother Alphonsus and Staff of that institution I am indebted for the daily care of the patients and the maintenance of records forming the basis of this paper.

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EXPERIENCE WITH THE TREATMENT OF TROPHIC ULCERS BY PLASTER CASTS

CYNTHIA FISHER, M.B., B.S.
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Ulcers on the sole of the foot present a very big problem to the leprosy worker, both in the Settlement and in local Segregation Villages. Their treatment entails endless work for dressers, and many patients despair of ever being able to walk about without bandaged feet. It is extremely difficulty to keep bandages clean, and secondary infection is common. The ideal treatment is complete rest for the foot, but here our difficulty must be a common one—our patients feel well in themselves and hate staying in bed. Many patients have been very co-operative and have stayed in bed, only to find on their discharge from hospital that their ulcers have quickly reappeared. Such patients are, not unnaturally, unwilling to embark on another round of hospital treatment.

In the past all types of dressing have been in use here—daily eusol, flavine, peroxide, dettol or cod liver oil; or weekly medicated dusting powder, flavine emulsion or dry dressings and strapping. All these have the disadvantage that the application is time-consuming and entails frequent disturbance of the healing surface.

In order to deal with these difficulties, we have been using plaster-of-paris casts (below knee walking plasters) in the treatment of ulcers.

The treatment of chronic ulcers by plaster casts is a well-known and old-established method, but there is little reference to it in the literature on leprosy.

The importance of rest has been stressed by Khan[5] but even though he used plaster of paris for immobilising the limb, the ulcers were still dressed through windows in the cast, and although the patients were allowed to walk about they did not lead normal lives.

Small plasters of salicylic acid resin have also been used (Haythornthwaite[4]) but these were more in the nature of a hard-wearing dressing than a form of immobilisation.

Most writers (Maynard[1], Muir[2] and Lowe[3]) are agreed on the necessity for the removal of necrotic bone before the ulcer is able to heal.

Treatment with plaster casts has the following theoretical advantages:—

(a) Complete rest for the foot is ensured.

(b) Very little time is required in treatment as the cast takes only a short time to apply, and maintenance is easy.

(c) The patient is mobile throughout treatment, and there is thus no temptation to stop treatment before the scar has consolidated.

SELECTION OF CASES

Almost all ulcers below the knee were found suitable for this form of treatment, and age was no bar.

All sequestra were first removed, and we found that a week's course of daily eusol dressing was very useful in clearing up mild infections.

If the leg was oedematous, the patient was given two days' complete rest in bed to allow the swelling to subside.

A few patients had ulcers in congenitally deformed feet (e.g. talipes equino varus) but these were all fitted with casts which enabled them to walk comfortably.

TECHNIQUE

No suitable stockinette or walking irons were available.

Dry dressings were applied to the ulcer, and the limb bandaged very carefully from toes to knee with cotton bandages in lieu of stockinette. It is very important that the bandages should be applied without wrinkles as these encourage plaster sores, which, if neglected, are nearly as difficult to heal as the original ulcers.

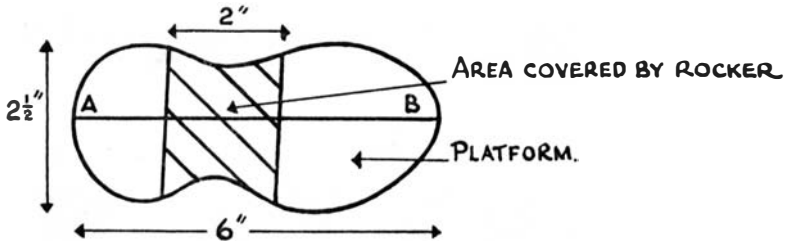
Three six-inch and one four-inch plaster bandages were first applied for each cast over the cotton bandages, from below the knee to the base of the toes. This made a light firm plaster which could withstand patching without becoming unwieldy.

Small wooden rockers of a suitable size were made by our carpenter, and these were fixed to the plastered feet with two four-inch plaster bandages. Thus a total of three six-inch and three four-inch plaster bandages was used on each cast.

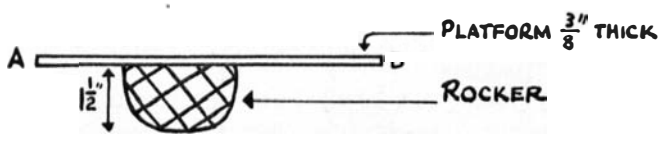
The patient was put to bed in the ward for forty-eight hours so that the plaster was thoroughly hard before he walked, and was seen again after twenty-four hours to ensure that the plaster was comfortable.

The *rocker is an essential* part of the treatment as it is necessary to keep the plaster dry by raising the foot from the ground. Wear of the rocker may be minimised by covering it with a layer of rubber.

PLAN OF ROCKER



SECTION ALONG LINE AB



MAINTENANCE

Constant supervision was necessary as the casts tended to wear through, and we have found a weekly plaster clinic most useful. A register of all patients with plasters was kept, and everyone reported to the clinic so that the Medical Officer could supervise repairs. Patients were encouraged to report to the Medical Officer at any time when their plaster cracked, so that an immediate repair could prevent the formation of a plaster sore. Those patients who for any reason (e.g. deformed hands) were unable to look after themselves, were not permitted to return to their normal houses in the Settlement, but were accommodated in two " plaster houses." These house four patients each, and are situated near the hospital so that the patients may be fed from the hospital kitchen.

LENGTH OF TREATMENT

The optimum time for treatment appears to be between three and four months. Most of our patients wore the same plaster during the whole of this time, and this was true even in the rainy season. Sometimes the children needed their plasters replaced after six weeks or so.

RESULTS

(a) *Healing of Ulcers.*

The results of the first fifty cases inspected are shown in the table below.

TABLE

RESULTS OF PLASTER TREATMENT

Condition of Ulcer on Removal of Plaster	No.	%
Total (plasters removed)	50	
Healed and ready for weight-bearing ...	25	50
Healed	11	22
Improved	9	18
No change	5	10
Worse	Nil	
Number still in plaster	15	
Total treated	65	

All the plasters were applied and the results assessed by the writer, so that the standards applied throughout were uniform.

" Healed and ready for weight-bearing " indicates that the foot needed no protective dressing after the plaster was removed.

" Healed " means that there was no raw area, but the ulcer was not strong enough for the patient to walk barefoot, and dry dressings were worn for two or three weeks for protection.

" Improved " means that the ulcer was reduced in size.

All except two of those ulcers classified as healed have remained so till the time of writing (at least three months and some for as long as six months). Both these relapses occurred on congenitally deformed feet, and the ulcers were very small, and have now healed. They could have been prevented if the plaster had been left on for a few more weeks.

Of the sixty-five patients, forty-four had received treatment in hospital for their ulcer for varying periods, but the length of time in bed did not affect the results of plaster treatment. Eleven of these patients had been in bed for six months or longer, and it was the failure of other forms of treatment which induced the application of plaster.

(b) *Others Effects.*

(1) Most patients were able to live a more or less normal life, and requests for plaster treatment are multiplying.

(2) The perennial problem of having too many patients for

too few hospital beds has been solved. We have released thirty of our eighty hospital beds for more important surgical cases.

(3) Many patients whose leprosy had been quiescent for as long as two years, but who had been detained because of ulcers, have been discharged and have now returned home.

(4) The number of staff required for dressing ulcers is considerably reduced, and we aim to close down our ulcer dressing room completely.

(5) Clinic patients can be admitted to the Settlement for treatment and returned to their Segregation Villages when their ulcers are healed.

CONCLUSIONS

We have found this method of treatment simple and practical, and, with proper safeguards, to be commended.

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ACKNOWLEDGEMENTS

I wish to express my thanks to the Director of Medical Services Eastern Region, Enugu, for permission to publish this paper, to Dr. T. F. Davey, Senior Specialist, Nigeria Leprosy Service, who suggested this work and gave encouragement and advice, and to all the patients who have co-operated so willingly.

LEPROMIN NOT INACTIVATED BY LEPROMATOUS SERUM

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and

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INTRODUCTION

In his interesting paper on ' The Significance of Antibody in the Pathogenesis of Leprosy,'' Ridley (1954) reported experiments which seemed to indicate ' that in many lepromatous sera there is present an antibody which reacts with lepromin to fix complement; and that some lepromatous sera are capable of neutralising

lepromin so that a mixture of the two does not elicit a response in the skin of tuberculoid patients.'"

Regarding the presence of lepromin complement fixing antibody it will suffice to say that sera from lepromatous cases have long been known to fix complement in presence of antigen prepared from a variety of acid-fast bacilli including the tubercle bacillus. This property of the lepromatous serum is therefore not likely to be due to the presence of a specific lepromin complement fixing antibody.

His finding regarding the inactivation of lepromin by incubation with lepromatous serum is a very interesting one, and if confirmed, would satisfactorily explain the negative reaction to lepromin in lepromatous cases. According to Ridley a negative reaction in such cases is caused by the neutralisation of the injected lepromin by a circulating antibody so that the antigen is not available for reaction with fixed antibody of the tissues to produce a positive reaction. In view of the important bearing that it may have on the matter, attempts were made to repeat Ridley's work regarding the inactivation of lepromin by lepromatous serum, and the results are reported herein.

METHODS AND MATERIAL

In the work reported here sera from three active and advanced cases of leprosy of lepromatous type were used (designated in the tables as A, B and C). The sera were freshly drawn and were not inactivated by heat. Following the method used by Ridley, mixture of equal parts of serum and lepromin was incubated at 37° C. for half an hour and then put in the refrigerator for about 24 hours before being used for the test. As controls were used mixtures of: (i) lepromin and tuberculoid serum; (ii) lepromin and physiological saline; (iii) lepromatous serum and saline; (iv) tuberculoid serum and saline; all the controls were prepared and incubated in the same way as the mixtures of lepromin and lepromatous serum.

As antigens two different preparations were used—the refined antigen prepared by the chloroform method and designated here as Dharmendra's antigen; and Mitsuda's antigen prepared by Wade's method and designated here as Wade's antigen.

For the test a total number of 24 patients of the tuberculoid type were used, all with thick, raised, bacteriologically negative lesions, i.e. persons who were expected to produce well-marked early and late reactions to lepromin.

The patients were divided into 4 groups of 6 each. Each group was injected with a mixture of lepromin and lepromatous serum, together with 2 or 3 control mixtures, as indicated later. These injections were given intradermally into the arms of the patients. Early results (24 to 48 hours) were read in all the 4 groups, while late results up to 5 weeks were read in 2 groups.

RESULTS

The results in the 4 groups of cases with various mixtures are included in the accompanying tables and are summarised below:—

The results in Groups I and II can be taken together since the only difference between the two groups is in the lepromin used, Dharmendra's antigen in one case (Group I) and Wade's antigen in the other (Group II). Serum from the same lepromatous case (A) was used for mixing with the antigens, and all other conditions were also similar. As controls were used the mixture of the antigen with: (i) physiological saline, and (ii) serum from a tuberculoid case. Both early and late reactions were read.

Regarding the early reaction, it may be said that in none* of the cases and with neither of the antigens used was early reaction less marked with the antigen mixed with lepromatous serum than with the other two preparations. On the other hand, in all the cases it was more marked with this particular preparation than with the other two preparations.

The late reaction, as was to be expected, was weaker with the refined antigen (D) than with the crude antigen (W). However, when the different preparations of the refined antigen are compared, it will be noted that the lepromin-lepromatous serum mixture has always given a little stronger reaction than the other two preparations. As compared with the refined antigen, the crude antigen (W) gave a stronger late reaction, and in this case there was not much difference in reaction to the 3 preparations, all of them giving reactions of about the same strength.

The results in Groups III and IV can be considered together as the two groups differ only in respect of the source of the lepromatous serum (from patient B in Group III and patient C in Group IV), and the dose of the various preparations (0.1 c.c. in Group III, and 0.2 c.c. in Group IV). Only the refined antigen was used, and only early readings (24 to 48 hours) were taken. As controls to the lepromin-lepromatous serum mixture were used

* In one case with one of the antigens (W) the reaction at 24 hours was slightly more marked with the antigen-saline mixture than with the antigen-lepromatous serum mixture, but at 48 hours the reaction to the latter mixture was greater. In all other cases the reaction to the antigen-lepromatous serum mixture was greater at both the 24 and 48 hours readings.

equal amounts of mixture of saline with: (i) lepromin, (ii) lepromatous serum, and (iii) tuberculoid serum.

A perusal of the results will show that as in the previous two groups the early reactions to the lepromin-lepromatous serum mixture were in no case less marked, but on the other hand were almost always more marked than those with lepromin-saline mixture. As was to be expected, the mixture of lepromatous and tuberculoid sera with saline generally gave negative results. However, one of the lepromatous sera gave rise to doubtful reaction in 2 cases and a definite but weak positive reaction in another case. Reaction with this serum (C) in the other cases and with the second serum (B) in all the 6 cases were negative. A similar reaction was seen in 1 of the 12 cases with the serum from a tuberculoid case. It is difficult to say anything about the significance of these reactions to the serum. It may, however, be said that it is not likely that these slight reactions were caused by any contaminating organism, since after incubation and storage in the refrigerator there was no gross turbidity seen in the serum-saline mixtures, nor did smears from them show any contamination.

DISCUSSION

The findings reported here do not lend support to those of Ridley regarding the neutralisation of lepromin after it has been mixed and incubated with serum from a lepromatous case. Sera from 3 lepromatous cases were included in the study under report and 24 cases of tuberculoid type were tested with intradermal injections of mixtures of lepromin with these sera alone with some other mixtures similarly prepared and incubated. In none of the 24 cases was the addition of lepromatous serum to lepromin found to abolish or reduce the actiivty of lepromin. On the other hand, it was found to increase the activity since reaction to the lepromin-lepromatous serum mixture was almost always stronger than that to an equal amount of lepromin-saline mixture prepared and treated in an identical way.

In addition to the early reaction, late reactions (not reported on by Ridley) were observed in 12 of the 24 patients. As in the case of early reaction, the addition of lepromatous serum to lepromin did not inhibit the late reactions also.

The hypothesis advanced by Ridley regarding the cause of negative lepromin reaction in lepromatous cases therefore cannot be upheld. According to his hypothesis the negative reaction to lepromin in such cases is caused by the presence of circulating antibody which neutralises the antigen before it can reach and react

with the fixed antibody in the tissues. If confirmed this would have provided a plausible explanation for the little understood negative reaction to lepromin in lepromatous cases.

It has been stated above that the lepromin-lepromatous serum mixture, far from giving weaker reactions, gave stronger reactions than mixtures of lepromin with saline or tuberculoid serum. The reason for this is not clear. It may have been caused by the fact that lepromatous serum diffuses less rapidly from the site of injection thereby resulting in more marked induration which is maintained for a longer period. However, this is only a surmise. injection of lepromatous and tuberculoid sera (mixed with saline) did not provide any clear-cut evidence on this point, although they provide some indication to this effect.

SUMMARY

The addition of lepromatous serum to lepromin was not found to neutralise or reduce its strength as judged by the reaction produced by intradermal injection of the mixture in cases of leprosy of tuberculoid type. The findings of Ridley in this connection are therefore not confirmed.

In view of these results Ridley's explanation regarding the cause of a negative lepromin reaction in lepromatous cases cannot be supported and does not appear to be correct.

REFERENCE

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GROUP I

Serum A

Early and Late Reactions to Dharmendra's Antigen and Controls

(Dose of mixture injected 0.2 c.c.)

Case No.		Early Reactions (E/I in mm.) Lepromin with Saline	Lepromin with T. Serum	Lepromin with L. Serum	Late Reaction (Nodule in mm.) Lepromin with Saline	Lepromin with T. Serum	Lepromin with L. Serum
1	...	15/3	20/3	26/3½	2	2	2½
2	...	22/3½	23/3½	27/7½	2	2	2½
3	...	37/5	33/4½	39/6	3½	4	4
4	...	28/6	34/5	38/8½	3 Ulcer	3½	4 Ulcer
5	...	23/3½	34/7	40/12	2	3	4
6	...	35/5½	37/6½	41/13½	4	4	5

GROUP II

Serum A

Early and Late Reactions to Wade's Antigen and Controls

(Dose of mixture injected 0.2 c.c.)

Case No.		Early Reactions (E/I in mm.) Lepromin with Saline	Lepromin with T. Serum	Lepromin with L. Serum	Late Reaction (Nodule in mm.) Lepromin with Saline	Lepromin with T. Serum	Lepromin with L. Serum
7	...	28/6	25/5	30/5	Absent at 3rd & 4th week readings		
8	...	23/3½	22/3½	26/3½	5½ Ulcer	5	5½ Ulcer
9	...	19/3½	20/2½	23/5	3½	4	4
10	...	16/4	18/2½	21/3½	7	7	7 Ulcer
11	...	17/2½	25/3½	28/5	6	5	5
12	...	31/5	29/3	39/5½	7	7 Ulcer	6½

GROUP III

Serum B

Early Reactions to Dharmendra's Antigen and Controls

(Dose of mixture injected 0.1 c.c.)

Case No.	Lepromin + Saline 24 hrs.	48 hrs.	Lepromin + Lepromatous Serum 24 hrs.	48 hrs.	Lepromatous Serum + Saline 24 hrs.	48 hrs.	Tuberculoid Serum + Saline 24 hrs.	48 hrs.
1	16/2½	15/3	13/2½	19/3	Slt EI	Nil	Slt EI	Nil
2	19/2½	17/2½	29/3	28/2½	5/2	6/0	6/2	6/0
3	21/3½	19/3	21/4½	20/3½	5/0	Slt EI	5/0	Slt EI
4	28/4	25/4	22/4	24/3½	Slt EI	Nil	8/2½	12/2½*
5	18/3½	18/3	24/5	19/4	Slt EI	Nil	Slt EI	Nil
6	15/2½	17/2½	24/3½	22/3	5/2	Nil	Slt EI	Nil

* This reaction read as ±, all other reactions with the serum-saline mixtures were read as negative.

GROUP IV

Serum C

Early Reactions to Dharmendra's Antigen and Controls

(Dose of mixture injected 0.2 c.c.)

Case No.	Lepromin + Saline 24 hrs.	48 hrs.	Lepromin + Lepromatous Serum 24 hrs.	48 hrs.	Lepromatous Serum + Saline 24 hrs.	48 hrs.	Tuberculoid Serum + Saline 24 hrs.	48 hrs.
7	21/5	16/3½	20/5	19/4	7/2½	Nil	7/2½	Nil
8	17/4	16/4	25/5	21/5	14/3†	16/3†	6/2	Slt EI
9	22/5	18/4½	30/6	22/5½	8/2	Nil	5/2½	Nil
10	33/10	30/8	30/6	38/4½	19/2*	22/2*	6/0	Nil
11	20/2	19/2½	30/5	23/3	Slt EI	Nil	Slt EI	Nil
12	22/6	21/6	24/7	28/6	12/2½*	14/2½*	Slt EI	Nil

† Reaction read as +.

* Reaction read as ±.
All the other reactions with serum-saline mixture read as negative.

THE SEVENTH SESSION OF THE WHO REGIONAL COMMITTEE FOR SOUTH EAST ASIA

The Seventh Session of the WHO Regional Committee for South-East Asia was held in New Delhi from 21st to 25th September, 1954. At this session leprosy featured prominently in both the Sixth Annual Report of the Regional Director and the Regional Programme and Budget Estimates for 1956, as also in the discussion on these two documents.

In his report the Regional Director referred to the recommendation of the Regional Committee at its Sixth Session in 1953 regarding the need for augmenting efforts in the field of leprosy. He stated that almost all countries in the Region have been giving increasing attention to this problem, and outlined anti-leprosy activities in the various countries with WHO aid.

In Burma the Leprosy Control Project was started in August, 1952, following the report of the WHO Leprosy Consultant, who made a survey in 1951. The WHO Leprosy specialist provided to the Government of Burma in 1952 terminated his activities towards the end of 1953 and the project is being further developed by the national staff. Outstanding developments are the: (1) establishment of a Central Leprosy Institute in Rangoon to direct and guide the national campaign as well as conduct research and training activities, (2) opening of a new State Sanatorium for the care of patients, (3) introduction of methods for early detection and treatment, and (4) utilisation of auxiliary personnel in the expanded programme.

In Ceylon, the Leprosy Control Project was based on the report of the WHO specialist who made a survey in 1951. A leprologist is in the country since June 1954 and an occupational therapist will be recruited shortly.

In India, a national control campaign has been instituted, and plans have been made for the early organisation of modern anti-leprosy units in the different States in endemic areas and gradual increase of such units throughout the country. This plan is also under discussion for UNICEF and WHO assistance.

In Indonesia, plans for an intensive anti-leprosy programme, with the possible assistance of UNICEF supplies and equipment, are being made. WHO has been requested to provide the services of a leprosy consultant for a period of 3 months in 1954 to carry out a survey and it is proposed to provide this. Subsequent assistance will depend on his findings.

In Thailand, a leprosy survey has been conducted in 1953 by a WHO leprologist and his report has been submitted to the government. At a conservative estimate the total number of cases of leprosy in Thailand is at least 100,000. The existing anti-leprosy services are inadequate to meet the need for adequate case finding, treatment, and follow-up. Following are some of the recommendations made by the consultant: (1) The anti-leprosy work in Thailand be done on a regional basis. (2) There be a colony and hospital in each region under the charge of a trained medical graduate, who may also act as Regional Leprosy Officer. (3) A strong anti-leprosy unit be set up at the centre in the Public Health Department

to co-ordinate leprosy work throughout the country and to arrange for the training of all the necessary personnel of various categories. (4) Intensive sulphone treatment of all cases through leprosy clinics or dispensaries, with selective and discriminate isolation of highly infectious cases in leprosy colonies or settlements of simple and cheap type, and the health education of the public in general. The Government of Thailand are considering these recommendation and, to help carry them out, they have requested the services of a WHO leprosy expert.

During a discussion of the Regional Director's Report, the delegates from India, Ceylon, Indonesia and Thailand commented on the leprosy control situation in their countries. The delegates from Ceylon raised the question of a possible conference or study group on leprosy in view of some difference of opinion in his country on modern methods of leprosy control. The Regional Director suggested that he would welcome proposals for a group of leprosy workers from Ceylon to visit countries where modern leprosy control methods were in operation, or to send consultants from outside Ceylon or arrange for holding of a seminar. After some discussion, it was agreed that it was not yet opportune to hold a conference but to await a concrete request from Ceylon Government.

The Regional Programme and Budget Estimates for 1956, together with revised programme of work for 1955, were approved with some minor changes made by the Programme Sub-Committee. In introducing the proposed programme and budget estimates, the Regional Director remarked that these had been developed in such a way as to greatly reduce the possibility of dislocation on Regular Budget of any fluctation in the funds from Technical Assistance and other extra budgetary sources. He pointed out that the proposed programme reflected the increasing integration of WHO assistance in national long-term programmes, Leprosy Control Programme being amongst such long-term projects. The proposals and budget estimates for leprosy for 1955 and 1956 in the various countries of the region are as under: In Burma, where the leprosy control project has now been taken up by the Government, there is a provision for $1,300 under regular budget during 1955, and none during 1956. In Ceylon, where the leprosy project was commenced in 1954, it is proposed to continue it during 1955 and 1956; the provision under regular budget for the two years is $16,320 and $10,220 respectively. In Indonesia under the regular budget there is a provision for a leprologist during 1955 and 1956, with equipment and supplies, possibly from other extra-budgetary funds. The regular budget estimate for the two years is $10,830 and $11,400 respectively: under other extra-budgetary funds there is a provision for $25,000 and $20,000 for the two years respectively.

For Thailand there is provision for a leprologist during 1955 and 1956, and it is hoped to secure equipment and supplies through other extra-budgetary funds. Under the regular budget there is provision for $12,200 and $10,820 for the two years respectively: under the extra-budgetary funds the provision is for $30,000 and $25,000 respectively.

There is reported in *Hindu*, a Madras journal, a statement made at the opening of a leprosy clinic in the Cuddalore District of Madras State, that " in that district there were 2,315 villages with a population of 27 lakhs [2,700,000], of which 150,000 were affected." This, if correct, means that about 5½ per cent of the population are suffering from leprosy. It is proposed to have one treatment centre for every ten villages, the expense to be met largely by public donations.

REVIEWS

International Journal of Leprosy. Vol 22, No. 3, July/Sept. 1954.

The original articles are as follows:

N. Souza Campos and P. R. de Souza write on " Reactional States in Leprosy." They apply the term " lepra reaction " to only " erythema nodosum leprosum " which occurs in lepromatous leprosy. They distinguish between (a) tuberculoid reactivation (tuberculoid lepra reaction), (b) reactional tuberculoid leprosy, which they hold to be a distinct variety, and (c) the *limitantes* or " border line " condition. The clinical and histological features of these conditions are described and illustrated. (The reviewer did not find it easy to understand this paper.)

R. S. Ginto, J. A. Doull and Laurentino De Guia write on " The Mortality of Persons with Leprosy prior to Sulphone Therapy," based on accurate studies carried on in the Cordova, Talisay, Cebu Provinces, Philippines, from 1933 till 1951. Sulphone treatment was not used at all commonly until 1951; most of the patients had been treated for some years with hydnocarpus injections. Standardised death rates are combined for the total population and for persons suffering from (a) lepromatous and (b) non-lepromatous leprosy. The mortality in lepromatous patients was five times that of the general population; in non-lepromatous leprosy the mortality rate was little higher than in the general population.

In the period 1941-48, covering the Japanese occupation, the mortality in the general population rose by 64.5 per cent, but in the leprosy patients it was less, probably because many returned

to the leprosarium and were thus safeguarded from malaria, malnutrition and other hazards.

The study should give valuable data for comparison with similar data collected during the post-sulphone era from 1951 onwards.

J. L. Buyers and R. R. Wolcott write on " M. leprae in skin and nasal scrapings during Sulphone Treatment.' 146 cases were studied. They found that within one year, the nasal scrapings became negative in 50 per cent and in 5-10 years over 90 per cent. The skin scrapings became negative in less than 10 per cent of patients treated from one to ten years. The nasal scrapings were studied because, from the public health point of view, the nasal mucosa may be more important than the skin which is usually free from ulcers.

Montestruc et al. write on " Child Leprosy in Martinique." They record a high incidence in children, and a high proportion of lepromatous cases in children. The difficulty in tracing the source of infection, frequently extrafamilial, is discussed. The limitations of chaulmoogra treatment are emphasised; it has been abandoned. Sulphones are now in general use. It is proposed that all new-born babies and all anergic leprosy contacts shall be compulsorily vaccinated with BCG, and compulsory hospitalisation of all lepromatous children is recommended.

J. H. Hale, B. D. Molesworth, D. A. Russell and L. H. Lee write on " Isonicotinic Hydrazide in the Treatment of Leprosy." Their own summary is as follows:

1. A trial on isonicotinic hydrazide treatment of leprosy in Malaya is reported.

2. Only 26 patients out of a total of 83 showed any improvement at the end of an eight-month period.

3. Eighteen of the patients showing improvement were from the group classified as atypical. Since spontaneous changes take place frequently in cases of this group, it is difficult to assess the effect of the drug.

4. Because only 24 per cent of the whole series became progressively worse under the treatment, we believe that the drug is not without some limited therapeutic effect. It is not, however, comparable in efficiency to the sulfones.

J. Convit and E. Rassi write on " Lepromin and Tuberculin Tests in Venezuelan Leprosy Foci and the Induction of Lepromin Reactivity by BGC Vaccination." Their own summary is as follows:

The reactions to tuberculin and lepromin were studied in 8,353 inhabitants of leprosy foci in rural areas in Venezuela. Of this general group, a total of 1,356 (16.2 per cent) proved to be negative to the Mitsuda test, while 5,205 (62.5 per cent) were Mantoux-negative. In the group of Mitsuda-positive persons 44.8 per cent were also Mantoux-positive, while in the Mitsuda-negative group on 3 per cent were Mantoux-positive.

In the Mantoux-positive group 99.5 per cent were Mitsuda-positive, while in the Mantoux-negative group 74.4 per cent were Mitsuda-positive.

Considering that 16.2 per cent of the persons examined were found to be without protection against leprosy in the first tests, and taking into account that almost all lepromatous and indeterminate cases come from the lepromin negatives, which form the endemic matrix of the disease, there can be no doubt but that BGC vaccination will be an effective phophylaxis, at least in rural areas, when it can reduce the percentage of Mitsuda negative from 16.2 to 1.3 per cent.

Dharmendra, N. Mukerjee and P. N. Khoshoo write on " A Comparative Study of three Antigens for the Lepromin Test," (a) the Wade modification of the Mitsuda antigen, (b) the original Dharmendra antigen (long extraction with chloroform and long grinding), and (c) modified Dharmendra antigen (1 hour's chloroform extraction and short grinding, 5 minutes. Tests with all three antigens were made on 110 patients, some being lepromatous, and tuberculoid and some intermediate. Early (24-48 hours) reactions and late reactions, 2-5 weeks, are recorded. With all three antigens the findings are summarised:

The Wade-Mitsuda antigen produced the highest number of positive late reactions and a fair number of early reactions. The Dharmendra original caused the highest number of positive early reactions, and the lowest number of late reactions. The Dharmendra variant gave results which were intermediate.

In the lepromatous cases both the early and the late reactions were negative with all the antigens. In the maculoanesthetic cases the early reactions was positive in 82 per cent and 56 per cent with the Dharmendra and the Wade antigens, respectively, and the late reaction was positive in 34 per cent and 96 per cent with these antigens. In the tuberculoid cases the corresponding figures were 95 per cent and 92 per cent for the early reaction, and 74 per cent and 100 per cent for the late one.

In general the early reactions with the Dharmendra antigen and the late reactions with the Wade-Mitsuda antigen were in agreement. There was, however, disagreement in 5 of the 91 cases—4 of the 29 maculoanesthetics and 1 of the 36 tuberculoids.

The Dharmendra variant gave stronger late reactions than the Dharmendra original, but this increase was far below the strength and frequency of the late positive reactions with the Wade Mitsuda antigen. A better way of attempting to remove the disagreement between early reaction with Dharmendra's antigen and late reaction with Wade's antigen may perhaps be to try to increase the potency of the former.

J. Emibl, et al., report failure in an attempt to demonstrate multiplication of Hansen's bacilli in the yolk sac of the duck embryo.

Sister Hilary Ross writes on ' An Evaluation of the Maillard-Gagliardo Complement Fixation Test in Leprosy ' in 100 cases. This test uses sheep red cells sensitized with old tuberculin, and, as a complement, reconstituted dried guinea-pig serum. The test is characterised by the hemolytic reaction resulting from the fixation of complement. The summary is as follows:

1. The Maillard Gagliardo complement fixation test was performed on sera obtained from 100 cases of leprosy in which a diagnosis of tuberculosis had been excluded.

2. Normal controls showed titers up to 10.

3. Titers above normal were found in 44 (44 per cent) of the 100 cases of leprosy.

4. Titers above normal were found in 33 (58.9 per cent) of the 56 bacteriologically positive cases of lepromatus leprosy.

Y. T. Chang reports on studies of Nicotinamide and Pyrazinamide (Aldinamide) on Mouse Leprosy. His own summary is as follows:

A brief review of the pharmacological actions of nicotinamide and allied compounds in relation to the mycobacterial infections has been presented.

The activity of nicotinamide and pyrazinamide (Aldinamide) in mouse leprosy has been studied, employing the intraperitoneal route for infection. Compartive studies of the activities of these compounds with that of the known effective drugs—isoniazid, streptomycin and DDS—were made in animals treated immediately after inoculation or after delays of one or two months. The duration of the experiments was three months.

Both nicotinamide and pyrazinamide were found to be highly effective in the suppression of the leprous infection. Nicotinamide was found to have a degree of activity similar to that of pyrazinamide and of isoniazid, and superior to that of streptomycin; DDS was the least active.

All these five compounds were most effective when the administration was started immediately after the inoculation and continued for three months, proportionately less effective when the treatment was delayed for one or two months. Only nicotinamide, pyrazinamide and isoniazid possessed any significant activity when the treatment was delayed for two months and then carried out for only one month.

The Editor (H. W. Wade) discusses bacteriological improvement in the chemotherapy of leprosy. He mentions the fabulous number of bacilli present in untreated lepromatus lesions, the shrinkage in the size of the lesions produced by treatment, as well as the reduction in the density of the bacilli. He thinks that considering the tremendous number of bacilli that were present at the outset to be disposed of, and considering the resistance of even dead acid-fast bacilli to such deleterious influences as exist in the tissues of normal animals, let alone lepromatus patients, the wonder is that leprosy lesions ever clear up at all. At any rate, we should seriously ask ourselves if the current attitude that the bacilli in the lesions do not decrease as they should under sulfone treatment should not be reconsidered. The situation may be viewed with an attitude less discouraging to the physician and to the patient.

The Editor also discusses certain differences of opinion between research workers about cellular reaction to microbacteria. This discussion is difficult to summarise; the finding of Suter that monocytes cultured from immune guinea-pigs were able to inhibit multiplication of tubercule bacilli, whereas normal monocytes could not, has been challenged and discussed by Mackaness and others.

No More Leprosy, by J. A. Kinnear Brown, B.Sc., M.D., D.T.M.

This booklet is written to inform and stimulate the interest of all in Uganda who have to do with the welfare of the people. Dr. Brown did an important piece of work in Nigeria some 25 years ago, founding the leprosarium at Uzuakoli and initiating the system of clinics and village isolation centres which have proved so useful in bringing leprosy under control in Eastern Nigeria. Now that he has returned to Africa as the Leprosy Control Officer for Uganda, he is initiating a leprosy control system along similar lines. The booklet is divided into thirteen chapters, each of them dealing with some practical aspect of leprosy control. Most emphasis is laid on sample surveys. Already 56,000 people have been examined and 900 patients discovered. By this it is calculated that in the 5 million people of Uganda there may be 80,000 with leprosy. A hopeful sign however is that only 1 in 10 suffer from the severe lepromatous form of the disease, and that only 1 in 5 is under the age of 15. As to the method of control, he says: " The only way in which people can be safe is by not having contact with people with leprosy. The simplest and kindest way to be certain about this is to arrange for the lepers to have their own villages near dispensaries."

The booklet is well illustrated by photographs of patients. We publish with permission a reproduction of an interesting and useful propaganda poster given at the end of the booklet (see pp. 124-5).

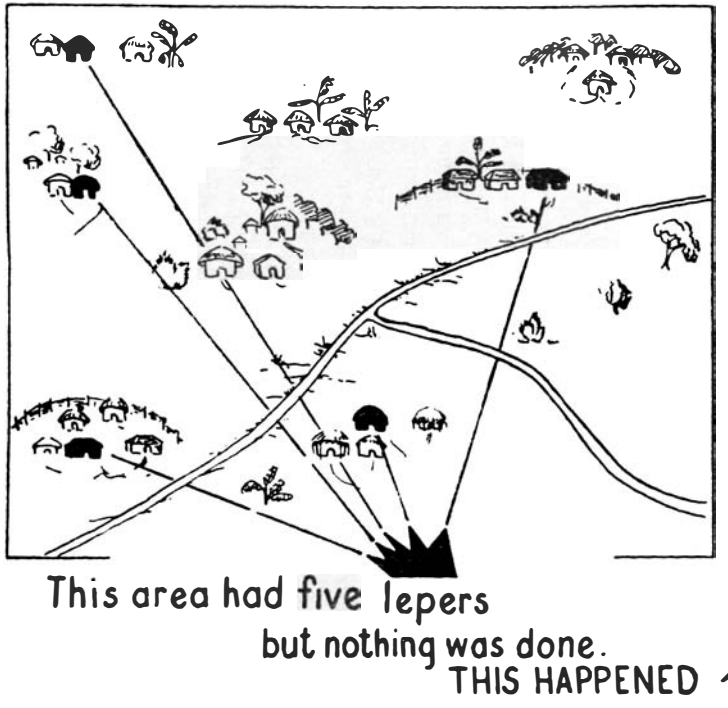
Leprosy in India. Vol. 27, No. 1 (Jan. 1955).

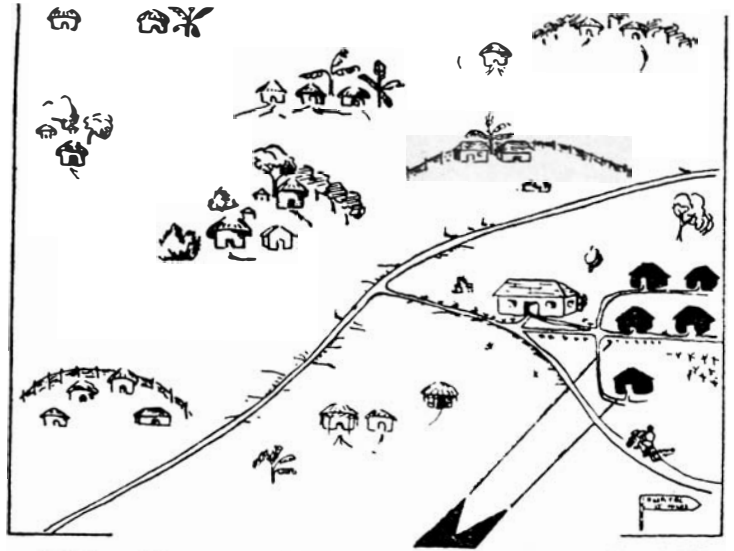
In Editorial Notes on classification the modified classification drawn up by the Indian Association of Leprologists for its second bi-annual meeting, to be held in Jamshedpur in March, 1955, is given. This corresponds largely with the views of Dr. Wade. Six main forms of leprosy are included: Lepromatous (L), Tuberculoid (T), Maculoanaesthetic (MA), Polyneuritic (P), Borderline (B), Indeterminate (I). These again are condensed into three main groups as follows:

Nonlepromatous (*N*)	*Intermediate* (*N/L*)	*Lepromatous* (*L*)
Tuberculoid (T)	Borderline (B)	Lepromatous
Maculoanaesthetic (MA)	Indeterminate (I)	
Polyneuritic (P)		

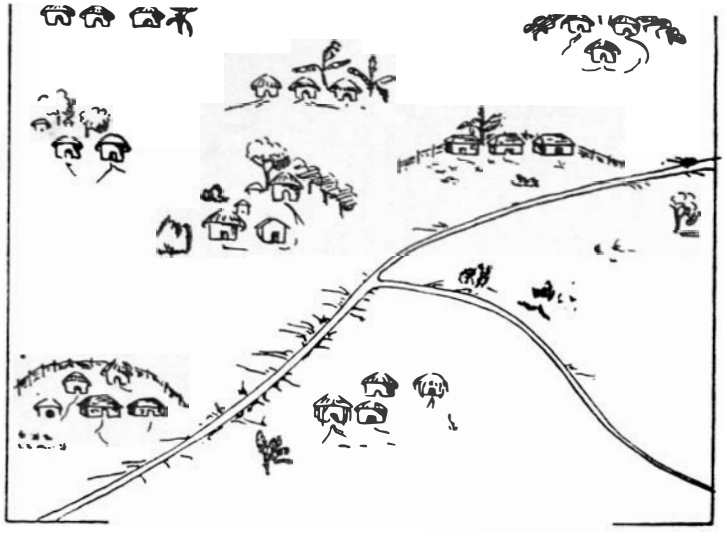
The following comment is made on the classification recommended at the International Leprosy Congress in Madrid in October, 1953:

HOW MANY LEPERS IN YOUR AREA





This village put its lepers in ONE PLACE and medicine was sent every week.



The lepers were cured and returned home and all danger disappeared.

THE MEDICINE IS READY!

WHAT ARE YOU GOING TO DO IN YOUR AREA ?

Ask your District Commissioner, Medical Officer, Health Inspector or your Chief for advice.

" One finds that when making actual recommendations regarding primary classification it is really histopathological criterion that has carried more weight with the committee, otherwise how could one explain the recommendation of the committee to include flat hypopigmented patches generally known as ' simple macular ' or ' maculoanaesthetic ' under the type ' Tuberculoid ' which term implies, and is usually applied to, thickened patches with varying degrees of elevation. The inclusion of the two morphologically different lesions under the term ' Tuberculoid ' can be justified only when histology is considered the basis of primary classification, since low grade tuberculoid changes are generally found in a large number of these flat patches. In this respect the recommendations of the WHO Expert Committee are preferable as the ' simple macular ' or ' maculoanaesthetic ' cases were included under a class separate from the red, thick and elevated lesions of the Tuberculoid type."

REPORTS

Itu Leper Colony, Nigeria. Report for 1954.

In 1954 the number admitted to the colony was 642, whereas in several previous years the number had been 900. This drop is in spite of the fact that the local authorities are taking steps to see that all who suffer from leprosy will come for treatment. This makes it clear that leprosy treatment is having its effect.

" 1955 is going to bring new problems for the colony. There is much to be done, and it has to be done with fewer able-bodied workers than ever before. Partly owing to the opening of treatment centres in the Cameroons, the total number of those receiving discharge at the end of the year was just under 2,000.'

Gratitude is expressed for donations received from the British Empire Leprosy Relief Association and the Child Adoption Scheme. Dr. and Mrs. Macdonald, the founders of the colony, retired in September. The new hospital was completed in July, 1954—a standing memory to the amazing work which they have accomplished and to the cause for which they have laboured so long.

The Report of Lake Bunyonyi Leprosy Settlement for 1954 mentions:

" Medical safaris in outlying parts of the district show that leprosy continues on the decline in Kigezi. Ankole patients, who speak the same language as that of Kigezi, are continuing to seek admission for treatment. These cases are in a very poor condition indeed nutritionally, and in an advanced state of the lepromatous disease. The condition of these cases suggests that there is a considerable incidence of leprosy in certain counties of Ankole."

Annual Report of the Medical Department, Tanganyika, for 1953.

The report mentions that cases of leprosy in government and missionary institutions at the end of 1953 numbered 976 and 3,792 respectively. The Interterritorial Leprologist has estimated that there are 100,000 cases of active infection. In order to make the best possible use of valuable acommodation in leprosaria, the

burnt out or untreatable cases are being discharged to their homes to make room for open infectious cases. There are 17 leprosaria in Tanganyika, where specific anti-leprosy treatment is given. Five of these are administered by government and the other twelve by missions. The BELRA staff work at the two principal government leprosaria at Makete and Chazi, and at two mission stations, Mngehe and Lulindi. The two largest leprosaria are those run by the Benedictine Mission at Perambho and Ndanda.

The Medical Department employed two medical officers on whole-time leprosy duties during 1953. Increasing emphasis is being given to encourage the development of out-patient leprosy services. The most notable progress in the development of out-patient facilities was in the Tanga District. Here to begin with few patients attended, as they were afraid they would be interned in the government leprosarium. When it became clear that sulphone treatment was effective, there was a dramatic increase in the number of patients, many of them coming from places where it had not been known formerly. There seems to be a widely held belief that a person suffering from leprosy should eat only such food as decayed meat, porridge made from old grain, and wild vegetables. Fresh corn as well as sugar and oil are denied him, and this abstinence frequently causes malnutrition. The progress of the work has been greatly helped by the interest and assistance of nursing sisters employed by various sisal estates; also African dressers from various native authority dispensaries assist in bringing in new patients. By the end of the year approximately a thousand patients were attending for treatment.

Proceedings of a Conference on Leprosy, Westfort, Pretoria, October 19th-21st, 1954.

A duplicated typed copy of the report of this Conference (78 pages) has been received. It is full of interesting material. Twenty doctors attended. The Conference was opened by Dr. le Roux, Secretary for Health and Chief Health Officer of the Union, in a speech in which he outlined the history of anti-leprosy work in the Union. Cases were recorded and hospitals opened in the 18th and early 19th centuries. In 1874, home segregation of patients was recommended. Laws for segregation were passed in the period 1884-1909, the leprosy institutions being under the Department of the Interior, together with the prisons. In 1924 the institutions came under the Health Department, leprosy boards were formed, and arrested cases discharged. In 1947 chemotherapy was started with gratifying results. In 1954 1,931 cases of leprosy

were reviewed by Leprosy Boards and 875 (45 per cent) were recommended for discharge. The incidence has fallen to 0.7 per thousand, and some leprosy institutions were so empty that they were being taken over for tuberculosis patients. The speech ended with the following paragraph: "The policy of compulsory segregation had repeatedly been attacked in all parts of the world. Wild statements had been made that the policy would cause patients to hide their disease. In actual fact it is now found that only 3.4 per cent of patients admitted to an institution do not come forward willingly for treatment."

Papers and demonstrations on the classification of leprosy were presented by Drs. A. R. Davison and H. Mostert; on histopathology by Dr. R. Kooij; on erythema nodosum leprosum by Dr. A. R. Davison; and its histopathology by Dr. R. Kooij; on blood chemistry by Dr. A. A. Kinnear; on thiosemicarbazone treatment (he found that it was usually inferior to sulphone, and that sulphone and semicarbazone given together was no better than sulphone alone), on very small doses of A.C.T.H. in complications, on atebrine in treatment of leprosy, all by Dr. Davison; on serological tests for syphilis and treatment of latent syphilis, by Dr. H. F. Schiller; on surgical treatment for leprous neuritis (" neurolysis ") by Dr. I. le Roux*, who recorded immediate and permanent relief of pain; on the lepromin test by Dr. R. Kooij; on the significance of " very scanty " positive smears (he points out that 10,000 bacilli per cubic centimetre is about the smallest number that will give positive results in routine examinations); on the effect of sunlight on reducing the acid-fast staining of leprosy bacilli, and on staining methods by Dr. R. Kooij (he stresses the importance of alcohol fastness in leprosy bacilli, and of not diagnosing leprous infection on positive smears without clinical findings); Dr. Kooij also reported on primary pigmentation of the skin caused by long wave ultra-violet rays of sunlight (two kinds of skin pigmentation produced by sunlight are mentioned, one being the well-known sun tan, preliminary erythema followed by secondary pigmentation, and the other being immediate pigmentation produced by rays of 300-460 m/u, the latter being produced in coloured races and even in pale leprous patches, and being produced by actual increase in pigment in the basal layer). Dr. Kooij also reported the effect of ultra-violet light in diminishing acid-fastness of leprosy bacilli recorded by Dharmendra and Mukherjee; and on electron microscopy of the leprosy bacillus. Dr. R. Davison also

* Dr. le Roux statement that this is a fairly new line of treatment necessitated by sulphone treatment is inaccurate. Dr. Lowe performed dozens of these operations between 1925 and 1930 and published his results.

presented two final papers, one producing evidence to show that delay in recognition of leprosy in the Union is rarely caused by fear of isolation, and the other discussing infection of the staff in leprosy institutions, recording cases in two European doctors and three European overseers who had worked in leprosy institutions in South Africa, two of the five persons having been born and grown up in England. In addition, 10 cases are recorded in Bantu persons who had worked in leprosy institutions.

CORRESPONDENCE

The Editor,
" Leprosy Review."
Dear Sir,

With regard to your editorial on dapsone in dermatitis herpetiformis in the April issue of the " Leprosy Review (page 56), we should like to remark that the authors mentioned in the editorial aforesaid made use of diasone and sulphetrone in this condition. As far as we know we have been the first who recommended diaminodiphenylsulphone in dermatitis herpetiformis (Dermatologica 1953, 16, 387).

Yours faithfully,

E. E. Kruizinga,
H. Hamminga.

Groningen (The Netherlands).
May 26th, 1955.

ABSTRACTS

The Determination of the Chronaxy in the Diagnosis of Leprosy by **A. Dubois.**

The author has applied the method of chronaxy, that is to say the period of latency between the application of an electrical stimulus to a muscle and the contraction of the same, to the diagnosis of early cases of leprosy. He finds it a useful method of confirming diagnosis and considers that it may possibly also furnish elements useful in prognosis and in understanding the pathology of the disease.

The Effects of Cortisone and ACTH upon the Eye Symptoms in Leprosy by **Einosuke Shionuma, Masao Arai** and **Nobuko Ito.**

The authors found in 65 patients that local treatment with cortisone produced very remarkable effects upon iridocyclitis, upper

scleritis due to reactions in lepromatous leprosy. It was of no use however in keratitis. ACTH was also useful in three patients in these same conditions. This fact is taken to confirm the assumption that erythema nodosum leprosum is an allergic inflammation.

A Comparison of Vaccination with Vole Bacillus and B.C.G. Vaccines. British Medical Journal, Jan. 1955, p. 133. By **H. W. O. Frew, J. R. Davidson** and **J. T. W. Reid.**

This report describes an investigation designed to compare vaccination with B.C.G. and vole bacillus vaccines, and records the prelinimary results after one and two years. The rate of conversion after vole vaccination is a little slower than after B.C.G., but the percentages (95 per cent after six weeks and 100 per cent after twelve weeks—are very satisfactory and compare closely with those obtained with B.C.G. Induration developed in a proportion of cases receiving vole vaccination by the multiple-puncture method, and later lupoid reactions occurred in the vaccination area. These occurred in 5 per cent after the first year and 15 per cent after the second year. This result occurring in arms regarded as healed at the first yearly inspection was unexpected and unfortunate, and has resulted in temporary suspension of the use of the vaccine.

The Effect of Pyridoxine on the Action of Isoniazid by **J. Ungar, E. G. Tomich, K. R. Parkin** and **P. W. Muggleton.**

Both in man and in experimental animals side reactions have been reported after Isoniazid treatment. It has been found that pyridoxine in suitable doses appears to counteract those side effects in animals. Pyridoxine does not interfere with the direct antituberculous activity in isoniazid in vitro, or in infected mice. The authors suggest that pyridoxine could be tried without adverse effect in the isoniazid treatment of patients who show intolerance to this drug.

Attempts to make positive the lepromin reaction in lepromatous cases. Le Semana Medica, **106**, p. 401. By **S. Schujman.**

The author claims that by repeated injections of various acid-fast bacilli (tubercle bacillus, rat leprosy bacillus, etc.) in lepromatous cases of leprosy, it is possible to sensitise the patient to lepromin so that the late lepromin reaction becomes positive. He claims to have converted negative lepromin reactions into positive in 55 per cent of cases.

Evolution des Méthodes de Prophylaxie de la Lepre. Annales de la Société Belge de Médecine Tropicale, Vol. XXXIV, 5, 1954, 144, p. 565. By **A. Dubois.**

Dr. Dubois comments on the hopeful prospects of bringing leprosy under control. At the Red Cross station Népoko, in the Belgian Congo, in 1919, there was a rate of 4 per cent of leprosy. It is calculated that there are 150,862 patients with leprosy in the Begian Congo, of which only about 6.41 per cent were lepromatous. The method employed formerly was to isolate in leprosaria not only those who were highly infectious lepromatous cases, but also those less infectious. With the advent of sulphones in 1946 the situation was considerably changed. Dr. Dubois states that it is now possible to change highly infectious cases into only slightly infectious ones, and to treat those with only few bacilli as out-patients. There is also no doubt it is possible to render negative or nearly so all open cases. It is now possible to envisage with confidence the rapid diminution of leprosy in the same way that in the last century sleeping sickness has almost disappeared. The two factors in ridding the country entirely of leprosy are active treatment, and at the same time raising the standard of living and hygiene.