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EDITORIAL

Mycobacterial Enigmata

Much has been written in recent years on the effect of tuberculosis infection, and particularly BCG, in converting a negative to a positive lepra reaction. Presumably, though we still lack positive proof, resistance to leprosy may also be raised at the same time.

Dr. McFadzean's paper in this issue raises the question as to whether there is any action in the reverse direction—does leprosy infection in the community have any effect in causing the low-grade tuberculin positives common in some countries but not in others.

In a report on a BCG campaign under WHO in Pakistan, abstracted on p. 122 of the last issue, mention is made of the frequency of low-grade tuberculin reactions in Eastern Pakistan, as compared with its comparative infrequency in Western Pakistan. Could this be due, at least in part, to infection of the community with leprosy, which is much more common in the former region?

On the other hand, is there not a possibility that some sub-pathological, or at least sub-clinical, mycobacterial agent may be at work in the body causing a low-grade sensitivity and/or resistance to the pathogenic mycobacteria. Repeatedly cultures of acid-fast or facultative acid-fast organisms have been cultured from biopsies of leprosy patients, the material having been taken from well under the surface of the skin, and with all precautions to avoid surface infection. Not infrequently it has been claimed, though without general support, that these cultures represent *Myco. leprae*. It has even been claimed that injection of some of them can convert the negative lepromin reaction to positive. Are similar organisms to be found in non-leprous subjects? If so, are they more frequent in some countries than in others? Can their presence be correlated with low-grade positive tuberculin or with positive lepromin reactions? Can they spread from the soil or from one person to another, and if so through what channels? These are matters worthy of investigation.

If BCG administered orally is able, without causing any recognisable symptoms, to convert a negative lepromin or tuberculin reaction, why should not other unrecognised organisms, belonging to or allied to the mycobacterial group, have similar effects?

Dr. Brown's paper approaches the question from the genetic angle. First a case is propounded in which a mother and her child are infected, the child having lepromatous leprosy, and the mother

a single tuberculoid lesion. Of the three methods of infection mentioned, the first seems the most likely. If both were not infected by an unknown person with concealed lepromatous leprosy, then it seems likely that the child was infected in that way and then, when his lesions had become infectious but were still concealed, he infected his mother, but only mildly, as she had comparatively high resistance.

The question of susceptibility varying according to genetic rules as set forth in the paper is of considerable interest. It might be useful to test its application in circumstances such as those in Culion, where children brought up under chances of repeated infection either escaped the disease or recovered without treatment (p. 164).

TUBERCULOSIS AND LEPROSY — FURTHER IMMUNOLOGICAL STUDIES

The Late JOHN LOWE, C.B.E., M.D., F.R.C.P.
Medical Secretary, British Empire Leprosy Relief Association
and
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Introduction

The idea that infection with *Mycobacterium tuberculosis* may produce cross sensitivity to the allied organism *Mycobacterium leprae* has been the subject of numerous papers during the last fifteen years, particularly from South America and from France. In three recent papers[1, 2, 3] one of us (J.L.) discussed the literature of the subject and presented recent work. It was concluded that there was strong evidence that natural tuberculous infection, as revealed by a positive tuberculin test, and also BCG vaccination, could and usually did sensitize a person to the leprosy bacillus, as shown by a positive lepromin test; and that this sensitization might possibly be accompanied by relative immunity to leprosy.

The possibility of the reverse phenomenon was also considered, i.e. infection with either *M. leprae* or some allied organism inducing sensitivity to tuberculin, but evidence on the matter was lacking.

While these articles were in the press there appeared the reports of Edwards *et al.*[4, 5] suggesting that in certain countries there appeared to be a non-specific factor which could cause a positive tuberculin test. They found that in certain areas, particularly in India and in Egypt, there were two types of response to tuberculin, (a) a high grade sensitivity (fairly strong reaction to a weak dose of tuberculin, 5 or 10 T.U.) which indicates the

specific response to tuberculous infection, and (b) a low grade sensitivity (small reaction to a small dose of tuberculin which appears as a larger reaction to a strong dose such as 100 T.U.) which is considered to constitute a non-specific response.

Regarding the nature of the factor producing the non-specific response, Edwards *et al.* make the following statements. "The cause is still unknown." "It is more prevalent in rural than in urban areas." "The hypothesis was offered that it is due to infection with an organism antigenically related to *M. tuberculosis*."

It has been shown that *M. leprae* is antigenically related to *M. tuberculosis*. Moreover, in all the areas mentioned by Edwards *et al.* (the south-eastern United States, Egypt, India) leprosy is endemic. On the other hand in the south-eastern United States leprosy is rare, while the non-specific factor is reported to be prevalent there, and also leprosy is endemic in Mexico where the non-specific factor is not common. It seems most unlikely that in all the countries where the non-specific factor is found to operate it could be caused by infection with *M. leprae*. It is, however, possible that, in certain areas, leprous infection might be one factor tending to produce sensitivity to *M. tuberculosis*.

The objects of the work here reported were (1) to obtain more data on cross sensitivity between tuberculous infection and leprous infection; (2) by using the methods of Edwards *et al.* to obtain evidence whether, in Nigeria, a non-specific factor influenced the result of the tuberculin test, and if so (3) to get evidence whether, in Nigeria, this non-specific factor could be infection with the leprosy bacillus.

General Background of the Work

In the work reported previously [1,2] from the Uzuakoli area of Eastern Nigeria, most of the healthy persons studied were adolescents and adults, with high tuberculin-positive and lepromin-positive rates; correlation between the two tests, though definite, was incomplete. In the present investigation it was planned to study healthy children, with lower tuberculin-positive and lepromin-positive rates, which might facilitate more accurate correlation.

By collaboration with the local mission school authorities it was arranged to test all the children, both boys and girls, in two large day schools. The ages of the children were between 5 and 16 years.

The schools were situated near Uzuakoli, East Nigeria, an area where clinical tuberculosis of any kind appears uncommon, where there are practically no cows (because of trypanosomiasis), where

milk in any form is not a common article of diet, and where tuberculosis of bovine origin apparently does not occur.

On the other hand, leprosy is common in the area, the incidence in the past having been of the order of 5 per cent of the population; many of the cases, however, were mild and " closed." In addition, there is in the near vicinity a leprosy settlement with several hundred patients, mostly open cases, who have a certain amount of contact with surrounding markets and villages.

Methods

621 children between the ages of 5 and 16 were tested with PPD 5 Tuberculin Units and, if the reaction was less than 6 mm., they were tested again with 100 Tuberculin Units. At the same time, all the 621 children were given an intradermal injection of lepromin prepared and standardized biologically at the Leprosy Research Unit, Uzuakoli.

Methods of Reading

TUBERCULIN. Measurements were made of definite areas of oedema.

LEPROMIN. For the present purpose, only the late (Mitsuda) reaction is considered, for in the dark-skinned Africans the reading of the early reaction is often not easy, and the late reaction is found more reliable. The late reaction consists of the formation of a nodule at the site of the injection of lepromin, the nodule usually appearing within two weeks and being at its maximum size about 21 days after the injection. The readings here recorded were made on the 21st day.

Those with no nodule formation, or a tiny one only just palpable, were recorded as *Negative.*

Those with a small but easily palpable nodule, usually measuring 2-3 mm. in diameter, were recorded as *Weak Positive.*

Those with a larger and very easily palpable nodule were recorded as *Strongly Positive.*

Results

The results of the simultaneous tuberculin and lepromin tests are given in Tables I-III together with the X^2 values. Table I gives the results for the entire age group 5-16 years, Table II the results for the age group 5-8 years, and Table III the results for 9-16 years.

The anlyisis of these results was kindly undertaken by Dr. Ian Sutherland of the Medical Research Council's Statistical Research Unit, who states that each of the three values of X^2 is undoubtedly significant, confirming the reality of the association between the results of the two tests. As regards the results in the 9-16 years

sub-group, inspection of the individual values contributing to X^2 $[(O-E)^2 \div E]$ shows quite a smooth relationship between the two tests, i.e. the stronger the tuberculin reaction, the stronger is the lepromin reaction. There are no breaks which might suggest non-specificity of weak tuberculin reactions. However, in the 5-8 years sub-group it appears that the *strong* reactions, i.e. those greater than 10 mm. to 100 T.U. and greater than 5 mm. to 5 T.U., behave as a group and show no association with the lepromin reaction. This pattern, if real, is difficult to interpret; the numbers, however, are small and the effect may only be due to chance.

Discussion

I COMPARISON OF LEPROMIN AND TUBERCULIN REACTIONS

A significant correlation has been demonstrated between the lepromin and tuberculin reaction in the group investigated. This, however, could have resulted from the simultaneous exposure of the population to leprosy and tuberculosis. It is felt that further elucidation of this relationship could best be made by undertaking simultaneous tuberculin and lepromin studies

(a) in an area in which tuberculosis occurs but not leprosy,
(b) in an area in which leprosy occurs but not tuberculosis, and
(c) in an area in which neither tuberculosis nor leprosy is found.

The first is easy to find, but it is doubtful if the latter two exist.

II. EVIDENCE THAT THERE IS A NON-SPECIFIC FACTOR TENDING TO CAUSE A POSITIVE TUBERCULIN TEST

If such a factor operated in Nigeria, it might be expected to reveal itself in two ways: (a) by a tuberculin-positive rate too high to be explained by the known prevalence of tuberculous infection, and (b) by the high frequency of cases in which tuberculin sensitivity was of low or moderate degree and revealed only by large doses of tuberculin, as described by Edwards *et al.*

(a) *The tuberculin-positive rate in Eastern Nigeria*: The tuberculin-positive rates in our present studies in children and adolescents are influenced by the standards applied in reading the tuberculin test. If the rather rigid standard is adopted to a 6 mm. or more response to 5 T.U., and 11 mm. or more response to 100 T.U., the positive rate was 44.3 per cent. If less rigid standards are adopted, and 6 mm. or more reaction to 5 T.U. or 100 T.U. are considered significant, the positive rate rises to 68.3 per cent. How do these figures fit in with the known incidence of tuberculosis in the area?

(ii) A statistically significant correlation was demonstrated between the lepromin and tuberculin reaction in the group investigated. However, this could have resulted from the simultaneous exposure of the population to tuberculosis and leprosy.

(iii) The tuberculin-positive rates appeared to be higher than could be explained by the incidence of tuberculosis in the area.

(iv) The results of the tuberculin tests with the two different doses of P.P.D. were similar to those reported by Edwards *et al.* in parts of the world where they consider that a non-specific factor is responsible for reactions to 100 Tuberculin Units in those negative to 5 Tuberculin Units.

(v) No conclusion could be drawn as to whether or not the leprosy bacillus is a factor in producing a positive tuberculin test.

TABLE I

Tuberculin and Lepromin Reactions in Age Group 5-16 Years

Result of Lepromin Test	Result of Tuberculin Test: 6 mm. or more to 5 T.U.		Reaction to 100 T.U. in those with 0-5 mm. to 5 T.U.			
	11 mm. or more	6-10 mm.	11 mm. or more	6-10 mm.	0-5 mm.	Total
Strong +	33	30	8	15	4	90
Weak +	43	43	35	42	28	191
Negative	24	27	32	92	165	340
Total ...	100	100	75	149	197	= 621

X^2 = 162.83 with 8 degrees of freedom.

TABLE II

Tuberculin and Lepromin Reactions in Age Group 5-8 Years

Result of Lepromin Test	Result of Tuberculin Test: 6 mm. or more to 5 T.U.		Reaction to 100 T.U. in those with 0-5 mm. to 5 T.U.			
	11 mm. or more	6-10 mm.	11 mm. or more	6-10 mm.	0-5 mm.	Total
Strong +	0	3	1	4	3	11
Weak +	10	10	16	9	15	60
Negtive	14	8	14	53	127	216
Total ...	24	21	31	66	145	= 287

X^2 = 51.93 with 4 degrees of freedom
(combining the strong and the weak positive lepromin reactors)

A few cases of tuberculosis have been seen among the leprosy patients and in the general population, but a definite opinion has been formed that tuberculosis is not common in the area where this study was made. It seems unlikely that the present incidence of tuberculosis alone can explain the tuberculin-positive rates here recorded in children and adolescents. Moreover, in a previous study in this same area 80.2 per cent of adults were found to be definitely positive to 50 T.U.

(b) *Comparison of our findings using PPD tuberculin with those of Edwards et al.*: The results reported by Edwards *et al.* are given below, followed by our findings in Nigeria presented in a comparable form:—

	Response to 5 T.U. (or 10 T.U.)					Response to 100 T.U. in those showing 0-5 mm. response to 5 T.U.				
Age	No.	0-5mm.	6-10mm.	Over 10mm.			0-5mm.	6-10mm.	Over 10mm.	
Age	No.	0-5mm.	6-10mm.	Over 10mm.			0-5mm.	6-10mm.	Over 10mm.	
INDIA		5 T.U.								
5-10	1206	85%	9%	6%		674	40%	22%	38%	
				15%					60%	
11-15	924	77%	12%	11%		447	32%	20%	48%	
				23%					68%	
5-15	2130	81%	11%	8%		1121	37%	21%	42%	
				19%					63%	
EGYPT		10 T.U.								
5-8	1119	64%	17%	19%		558	50%	41%	9%	
				36%					50%	
9-12	1403	47%	22%	31%		556	38%	45%	17%	
				53%					62%	
13-16	490	32%	17%	51%		123	29%	47%	24%	
				68%					71%	
5-16	3012	51%	20%	29%		1237	43%	43%	14%	
				49%					57%	
MEXICO		10 T.U.								
5-8	499	75%	2%	23%		351	89%	6%	5%	
				25%					11%	
9-12	913	63%	2%	35%		536	84%	11%	5%	
				37%					16%	
13-15	315	46%	1%	53%		130	70%	18%	12%	
				54%					30%	
5-15	1727	63%	2%	35%		1017	84%	10%	6%	
				37%					16%	

DENMARK			10 T.U.					
5-8	1636	97%	1%	2%	1507	91%	1%	2%
			3%				6%	
9-12	5600	95%	1%	4%	5098	95%	4%	1%
			5%				5%	
13-16	2606	90%	2%	8%	2269	94%	5%	1%
			10%				6%	
5-16	9842	95%	1%	4%	8874	94%	4%	2%
			5%				6%	

NIGERIA								
			Response to 5 T.U. P.P.D.				**Response to 100 T.U. in those showing 0.5 mm. response to 5 T.U. or 10 T.U.**	
5-8	287	84.1%	7.3%	8.3%	242	59.9%	27.2%	12.9%
			15.6%				40.1%	
9-12	262	58%	21.8%	20.2%	152	31.6%	46.0%	22.4%
			42%				68.4%	
13-16	72	37.5%	30.5%	32.0%	27	14.8%	48.2%	37.0%
			62.5%				85.2%	
5-16	621	67.8%	16.1%	16.1%	421	46.8%	35.4%	17.8%
			32.2%				53.2%	

A study of these figures shows that the findings in Nigeria are comparable to those recorded in India and Egypt, where the non-specific factor is considered to operate; our findings are widely different from those recorded in Mexico and Denmark where the non-specific factor is considered to operate little, if at all. The prominent feature is the high proportion of persons showing a feeble reaction to 5 T.U., but showing a much greater reaction to 100 T.U. Therefore, if the hypothesis of Edwards *et al.* is correct, it is possible that there is a non-specific factor present in the population studied in Nigeria.

III. THE POSSIBILITY OF LEPROSY INFECTION CONSTITUTING A NON-SPECIFIC FACTOR CAUSING A POSITIVE TUBERCULIN TEST

No conclusions can be drawn from the present investigation as to whether or not the leprosy bacillus is a factor in producing a positive tuberculin test.

Summary

(i) 621 children between the ages of 5 and 16 years in Eastern Nigeria were tested with 5 Tuberculin Units of P.P.D., and if the reaction was less than 6 mm. they were tested again with 100 Tuberculin Units. At the same time all the children were given an intradermal injection of lepromin, and the late reaction (Mitsuda) read.

TABLE III

Tuberculin and Lepromin Reactions in Age Group 9-16 Years

Result of Lepromin Test	Result of Tuberculin Test: 6 mm. or more to 5 T.U. — 11 mm. or more	6 mm. or more to 5 T.U. — 6-10 mm.	Reaction to 100 T.U. in those with 0-5 mm. to 5 T.U. — 11 mm. or more	6-10 mm.	0-5 mm.	Total
Strong +	33	27	7	11	1	79
Weak +	33	33	19	33	13	131
Negative	10	19	18	39	38	124
Total ...	76	79	44	83	52	= 334

$X^2 = 70.70$ with 8 degrees of freedom.

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SUSCEPTIBILITY AND RESISTANCE IN LEPROSY

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Infection with leprosy as a result of contact with a patient is not necessarily the whole story, because:—

(a) Conjugal infection is relatively uncommon.
(b) Only a fraction of the children of infected parents develop leprosy.
(c) The majority of people living in constant relationship with patients do not develop the disease; others do so after contact so brief or trifling as to pass unnoticed.
(d) Some of the evidence suggests that a child can infect a parent who had hitherto failed to contract the disease.

In illustration of (d) *P.N.* is an intelligent woman now in the employment of a leprosarium in Uganda. Her circumstances are well known to the authorities. She is aged 41, married with one child—a boy born when she was 22. Her husband has never had leprosy. None of the grandparents had leprosy. There is no history of leprosy in the family. She and her husband lived in a typical East African house, separated by farms from their neighbours. There were a few cases of leprosy in the district but none was obviously lepromatous. The nearest patient had tuberculoid

leprosy and lived more than 100 yards away. When the boy was 8, he developed hypopigmented macules on the forearms and the backs of the hands, which disappeared without treatment after two or three years. One year later a tuberculoid macule appeared on the mother's right cheek. This was confirmed by biopsy. A year later, when the boy was 13, he was admitted to the settlement with widespread lepromatous leprosy.

There are various alternatives—none of them easy:

A. *The boy's original macules were leprotic* and the boy infected his mother, or the mother infected the boy before she had any signs of tuberculoid leprosy.

B. *The original macules were not leprotic* and the mother infected her son with lepromatous leprosy from a bacteriologically negative tuberculoid patch, or before her patch appeared.

C. *Both mother and son were infected by the same source* but any contact was trivial and not remembered, and no different from what the mother and her neighbours normally experienced—e.g. seeing an occasional patient in the market.

" C " is quite feasible, but why in the same environment should the mother not have been infected before when she was younger? It is more probable that the boy's macules were lepromatous and that his presence in the family made all the difference. Both mother and child were susceptible, but the boy more so than his mother.

This is not the only case where the child has appeared to bring the infection into the house. In some instances the parent has shown the first signs several years after a son or daughter has developed leprosy. It is not always easy to delve into the history of patients as deeply as one would wish, but most of the anomalies listed above are explicable on the basis of inherited susceptibility or resistance.

Susceptibility and its converse, resistance, presuppose a factor *X*, the presence or absence of which decides whether the disease will develop and what form it will take.

If " A " is the descendant of a marriage where both parents have *X*, and " B " of a marriage where neither have *X*, " A " can be represented by *XX* and " B " by *OO*. If " A " and " B " marry, their children will belong to one of the groups *XX*, *OO* and *XO*—i.e. there will be resistant and susceptible children and others at a " neutral " point *XO* somewhere between.

(i) *If XX and XO marry,* the majority of their children
Will be resistant (*XX*);
Some will be " neutral " (*XO*);
None will be susceptible (*OO*).

(ii) *If OO and XO marry,* the majority of their children
Will be susceptible (*OO*);
Some will be " neutral " (*XO*);
None will be resistant (*XX*).

(iii) *If XO and XO marry,* the pattern will be the same as if *XX* and *OO* married—
Some will be resistant (*XX*);
Some will be " neutral " (*XO*);
Some will be susceptible (*OO*).

Where people of the Resistant group (*XX*) are in the majority and, therefore, marry only Resistants (*XX*) or Neutrals (*XO*), casual contact may take place in each of several generations without producing clinical leprosy. When, however, two Neutrals (*XO*) marry, a susceptible child is possible who will develop leprosy, and the more serious type, after only a brief exposure to infection. If this susceptible child escapes because it is not exposed to any risk, and eventually marries a person who has become susceptible by the same process or even an individual belonging to the Intermediate or Neutral group, a family may be created with a bias towards susceptibility, and the infection of one member may rapidly lead to leprosy in some of the others producing " Household Infection."

In endemic areas where marriages follow the brother and sister pattern, or are confined within a small tribal group because of language and cultural differences, the incidence of leprosy may be higher.[1] Under such circumstances, the probability of the pairing of Neutrals or Susceptibles is greater. As the marriage circle extends the chances are less, especially if " *X* " is not a single factor or gene, but a combination of several.

It is known that a negative lepromin can be converted to positive by BCG. It is thought that sensitization to tubercle may protect against leprosy, but a positive lepromin does not protect against tubercle. It is improbable that " *X* " is anything but a compound factor.

Suppose that *O* represents the absence of one component related to leprosy;
,, ,, *X* ,, the presence of that component;

,, ,, *T* represents the absence of one component related to tuberculosis;
,, ,, *Y* ,, the presence of that component.

The possible combinations of *O*, *X*, *T* and *Y* are much greater than when only *O* and *X* are considered. If *OXTY* is paired with *XXYY*, the following may result:—

(a) *OXTY* " neutral " to leprosy and tuberculosis;
OXYY " neutral " to leprosy but resistant to tuberculosis;
XXTY resistant to leprosy but " neutral " to tuberculosis;
XXYY resistant to both.

Similar patterns may be worked out for other combinations, e.g.—

(b) *OXTY* and *OXYY* one neutral to both diseases, the other neutral to leprosy, resistant to tuberculosis.
OOTY susceptible to leprosy but neutral to tuberculosis;
OOYY susceptible to leprosy but resistant to tuberculosis;
OXTY neutral to leprosy and tuberculosis;
OXYY neutral to leprosy but resistant to tuberculosis;
XXTY resistant to leprosy but neutral to tuberculosis;
XXYY resistant to leprosy and tuberculosis.

(b) *OXTY* and *OXTY* are paired—both neutral to both diseases they may produce:—
OOTT susceptible to leprosy and tuberculosis;
OOTY susceptible to leprosy, neutral to tuberculosis;
OOYY susceptible to leprosy, resistant to tuberculosis;
OXTT neutral to leprosy, susceptible to tuberculosis;
OXTY neutral to both diseases;
OXYY neutral to leprosy, resistant to tuberculosis;
XXTT resistant to leprosy, susceptible to tuberculosis;
XXTY resistant to leprosy, neutral to tuberculosis;
XXYY resistant to both diseases.

To obtain a complete picture one would have to include any other components that may behave genetically, such as the blood group, the hormone pattern, the susceptibility to disease in general. There may indeed be others. It would be necessary also to know what the relationship is between the various immunological tests and the different genes, whether direct or indirect, specific or non-specific; what is the effect of small or massive infections, and what

causes conversion in the absence of infection or vaccination. There are benign cases of leprosy which have a very positive lepromin suggesting marked resistance but producing progressive and severe deformities; there are others in which bacilli may come and go and the lepromin reaction fluctuate round the ± level, whilst the disease runs a more hectic course but leaves less permanent damage in its trail. One sees tuberculoid patients with briskly positive Mantoux and lepromin reactions, and similar patients with a negative Mantoux and a strongly positive lepromin. On the other hand, there are the lepromatous patients with negative lepromins, some of whom are Mantoux positive, but others negative. The infiltration and ulceration following injections of lepromin also vary widely in patients who appear clinically to be almost identical.

These notes are put forward not as a complete solution but because it is believed that along such lines it may be possible to harmonise the various types of leprosy and their frequency, their different immunological reactions, the influence of other infections, and to explain why, and by what method, leprosy disappears from some communities after changes in the sex, age and type incidence.

(N.B.—The word " neutral " is not used to suggest that the resistant gene inherited from one parent is cancelled by the absence of that gene in the other parent. It is used only in an attempt to simplify what is probably a difference in degree.)

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THE PATTERN OF SENSORY LOSS IN LEPROSY AND ITS SIGNIFICANCE IN THE PATHOGENESIS OF LEPROTIC NEURITIS

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The pathogenesis of the neuritis of leprosy occupied the attention of workers at the turn of the century and has recently again come into the foreground as a result of the work of Khanolkar in the early changes in the nerve terminals resulting from the leprosy bacilli.[9] Although in general it may be said that the theory of Gerlach and Dehio [4] has found the most wide acceptance of all the theories, there yet remains some doubt in the minds of observers that it is not the whole picture.

The possibility of the lesion being in the spinal cord as suggested by Danielssen and others, has been denied by Woit.[11] The degenerative changes in the cord being regarded as secondary by Woit and subsequent workers. The discovery of bacilli in lesions which were previously considered negative (by standard methods of investigation) brought discredit to the theory that the neuritis of leprosy was centrifugal, or that the patch in leprosy was the remote effect of a proximal nerve lesion at the " site of predilection " as occurs in herpes (Virchow and others quoted by Dharmendra).[5]

In brief the theory of Dehio and Gerlach states that the primary involvement of the nerve fibre occurs at the periphery in the skin patch. The sensory nerves which supply this patch become invaded by bacilli and are surrounded and subsequently destroyed by the cellular infiltration which is the host's response to the presence of the bacilli. This inflammatory reaction ascends up the nerve fibres involving in its course a greater and greater number of nerve fibres until finally, when the sensory nerve joins the motor nerve, there is destruction of the whole mixed nerve trunk, resulting in the paralyses which are commonly seen.

In the material available to us in the orthopaedic unit we have attempted to study the absolute pattern of sensory involvement and also to correlate this with the pattern of motor paralysis.

Material

A total of 186 charts of patients who had attended the orthopaedic research department were examined. This covered a period of five years, from 1951 to 1955. The pattern of sensory loss had been a routine investigation for the upper extremity in each case. This pattern was drawn into a printed outline of the hand and forearm. Two modalities were noted: pain, as tested for by a pin or a bit of wire; and light touch, as tested for by a feather or a nylon thread. The sensory tests had been done by two observers, and the method was feather and pin in the earlier cases and nylon thread and wire (blunt pin) in the later cases.

In order to determine the error to these methods two observers assessed the same case using the two different methods for the touch and pain tests. The diagram Fig. 1 represents the result of this test. It will be seen that the pattern is the same but the measurements are different. This indicates that there is quite a margin of error but that the general pattern is comparable.

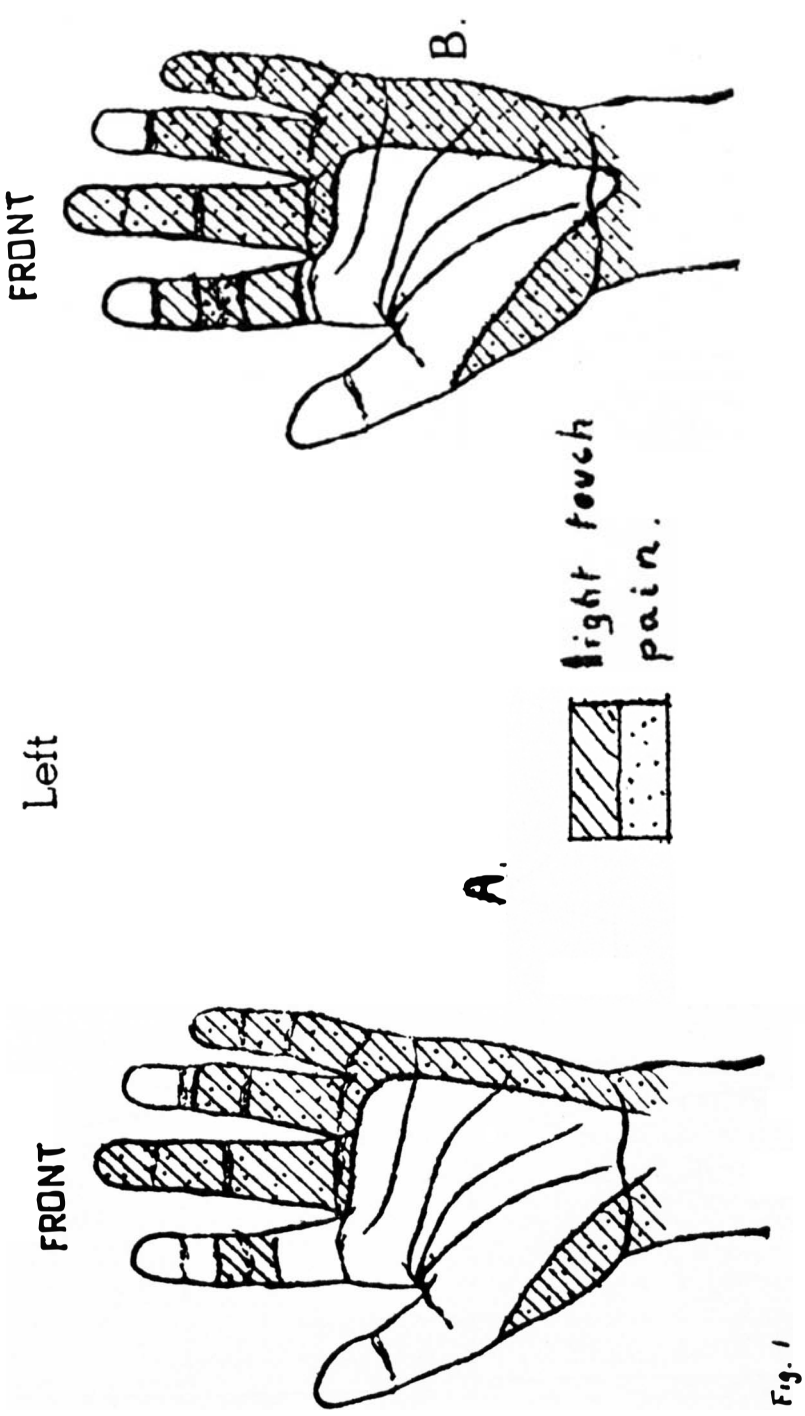
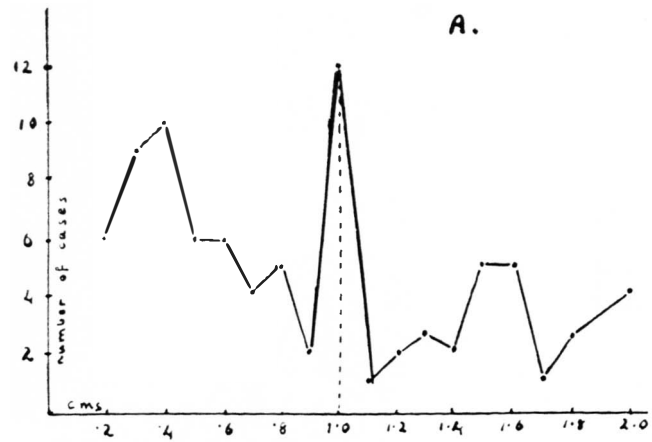


Fig. 1

Fig. 2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number of cases
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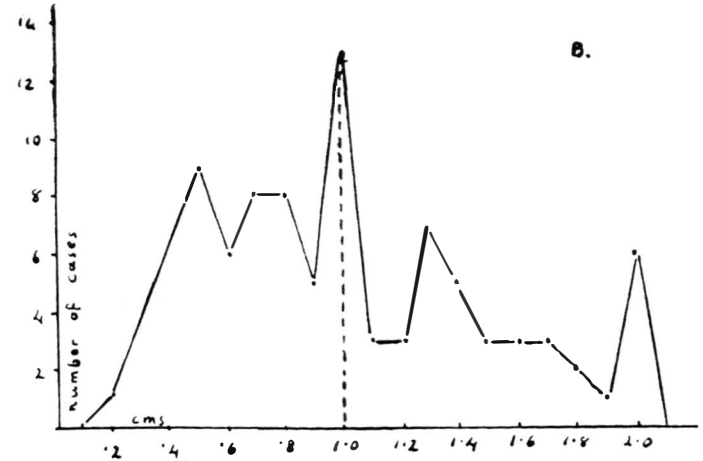
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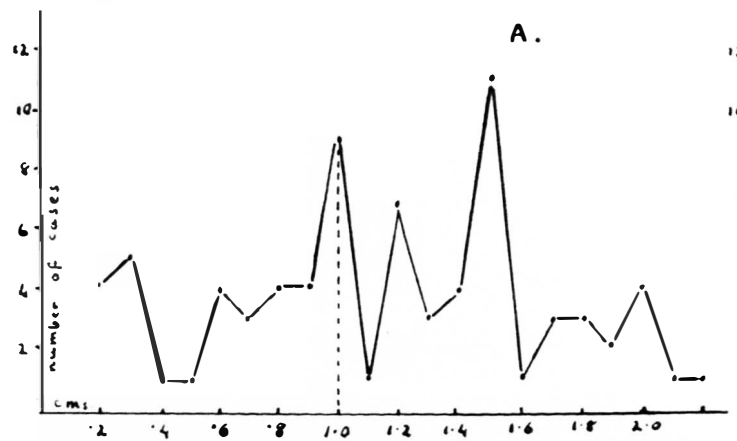
Fig. 3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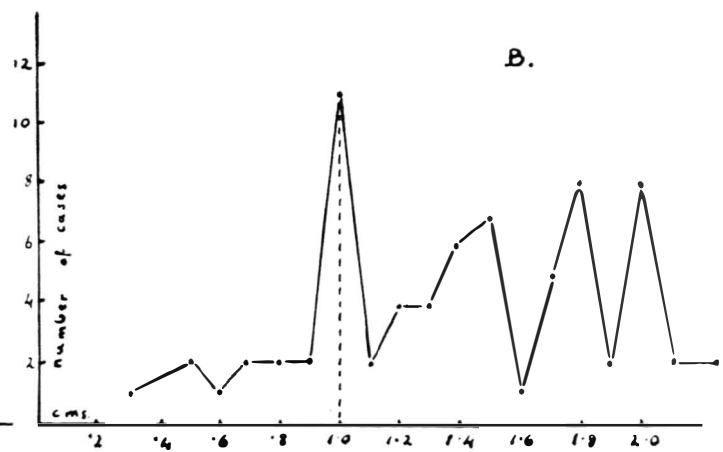
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FIG. 1. *Two sensory assessments of the same hand, done by different methods and two different assessors.*
(a) Assessment by M.A.F. using a feather and a sharp pin.
(b) Assessment by E.P.F. using a nylon thread and bent pin.

A total number of 304 hand charts were examined with a view to determining the percentage of incomplete sensory involvement (Table I). All the hands examined did not necessarily have a motor lesion, since in some cases only one hand had a motor lesion and both hands had a sensory deficit. In these cases the unparalysed hand was also included in these figures.

TABLE I

	Total	Complete Anaesthesia	Incomplete Anaesthesia	
Palmar aspect ...	304	181	123	(59.5%)
Dorsal aspect ...	304	191	113	(63.1%)

The percentage of incompletely anaesthetic hands (light touch).

The extent of involvement in the incompletely anaesthetic cases was now examined. The distance of the edge of the anaesthetic area from the ulnar border was measured on the diagram, this measurement was noted, and the results plotted as a graph with the distance across the palm plotted against the the number of cases. The results for the two modalities of light touch and pain (pin prick) are given in Fig. 2. It will be seen from the figures that in the majority of the incomplete cases the anaesthetic margin fell within the 1 cm. category (Table II) and that there was a marked peak at the 1 cm. distance. This distance on the diagram represents roughly the anatomical edge of the ulnar supplied skin area. None of the cases showed anaesthesia involving only the radial border of the hand, and in a few cases (due to the presence of a tuberculoid lesion on the hand) the anaesthesia was irregular and corresponded only to the area covered by the lesion.

On the dorsal surface the picture is less significant and no such clear cut peak could be demonstrated (Fig. 3). The number of cases falling within the ulnar border was considerably less than the number of cases beyond this (Table II).

FIG. 2. (*a*) *Pain* (*pin prick*); (*b*) *light touch.*
Graph showing the extent of the anaesthesia across the palmar surface of the hand from the ulnar border.

FIG. 3. (*a*) *Pain* (*pin prick*); (*b*) *light touch.*
Graph showing the extent of the anaesthesia across the dorsal surface of the hand from the ulnar border.

TABLE II

	Modality	Incomplete Anaesthesia	Within 1 cm.	Beyond 1 cm.
Palm ...	Touch	86	50	36
	Pain	85	60	25
Dorsum ...	Touch	71	21	50
	Pain	73	35	38

Table showing the number of cases in which the anaesthesia was limited to the ulnar area as compared with the number where it extended beyond.

Relationship between motor and sensory paralysis

A study of the pattern of sensory loss as compared with the pattern of motor loss was made (Tables III and IV). It is seen that in the majority of cases the anaesthesia corresponds to the motor loss but this is not uniformly the case. Thus, 10 cases out of 96 cases of ulnar motor paralysis still had some areas of sensation in the ulnar supplied area. And in 121 cases of ulnar and median paralysis there were 6 who had sensation in the ulnar area and 19 in the median area. This would seem to indicate that it is not necessary for the sensory component of the nerve to be completely destroyed before the motor component is affected.

TABLE III

Motor Lesion	No.	Complete hand anaesthesia	Incomplete anaesthesia
No motor lesion ...	25	0 (0%)	25
Ulnar	96	40 (42%)	56
Ulnar Median ...	121	97 (80%)	24
Ulnar Median Radial	2	2 (100%)	0

Table showing the percentage of cases in the different categories of motor involvement who show complete anaesthesia.

TABLE IV

Motor Lesion	No.	Sensation still present in part or whole of Ulnar area	Median area	Radial area
Ulnar	96	10	58	44
Ulnar Median ...	121	6	19	12
Ulnar Median Radial	2	0	0	0

Table showing the areas which are incompletely anaesthetic in the three motor nerve lesions.

The difference in the behaviour of the nerves in the two main sub-divisions of leprosy led to a study of the paralysis in these two sub-divisions. Table V gives the classifications of the 186 cases composing the series.

TABLE V

Leproma	57
Indeterminate	64
Tuberculoid	19
Dimorphous	15
Maculoanaesthestic	7
Unclassified	24
	186

Classification of the cases composing the series. The relatively large number of cases unclassified is due to the rapid turnover in the orthopaedic wards. Some of the patients were discharged before the chief leprologist had seen them.

Dividing these cases into the two categories Leproma and Non-Leproma, the charts were again examined and the result noted in Table VI.

TABLE VI

Class	No. of Hands	Complete Anaesthesia	Incomplete Anaesthesia	Areas showing incomplete involvement Ulnar	Median	Radial
Lepromas	99	67 (67%)	32 (32%)	11	33	23
Non ,,	145	72 (50%)	73 (50%)	5	44	33

Among lepromas there is seen to be a slightly greater tendency for the anaesthesia to involve the whole hand. In the non-lepromatous cases the proportion is almost exactly equal.

Discussion

In Figures 2 and 3 and Table II we see that in cases which show incomplete sensory involvement of the hand the largest number show a sensory deficit corresponding to the ulnar sensory area. This area is always involved at least in part, and, except in the case of a tuberculoid lesion which involves the hand, there were no cases seen where the anaesthesia involved areas of the hand other than the ulnar and left this latter area with sensation. This finding in itself indicates that the ulnar nerve is for some reason peculiarly susceptible to paralysis. In its motor component, too, this nerve is with very rare exceptions the first to be involved. This susceptibility of the ulnar nerve is familiar to everybody. Its site of predilection is, as was pointed out by the very earliest workers, just above the medial epicondyle of the humerus. The next most susceptible nerve appears to be the sensory terminals of the radial, although this is not often completely involved. In its motor component, however, the radial is the least commonly involved. The median nerve stands next to the ulnar in frequency of motor involvement, and is very often the last to be completely destroyed in its sensory component.

Thus, in the ulnar nerve the motor and sensory components are equally liable to damage. In the median nerve the motor component is more susceptible, and in the radial nerve the sensory elements are by far the most susceptible. There is, however, no provision in the hypothesis of Gerlach and Dehio to account for these irregularities. Why should the bacilli preferentially select for their entry into the nerve terminals, those of the ulnar nerve? Why is it that a lesion of the median nerve without involvement of the ulnar nerve is such a rarity? Why is a motor lesion of the radial nerve without involvement of the ulnar and median nerves never seen? Even in the cases of a single nerve trunk, the infiltration is not continuous but intermittent, leaving untouched the more deeply situated parts of the nerve. The sensory terminals may be the first affected, but the involvement of the motor branches is not in the ascending order of their joining the main trunk of the nerve but occurs as a whole, and very often is accompanied by obvious clinical signs at the site of predilection.

In their original papers Dehio and Gerlach point out that the parts of the ulnar nerve which lie deep in the forearm and hand are not infiltrated, whereas those parts which lie superficially above

the elbow and on the dorsum of the hand are heavily infiltrated. They fail to point out, however, that the anaesthesia is not due to the perineural infiltration of the terminal fibres, but to the proximal involvement of the nerve trunk at the site of predilection. Our anaylsis has demonstrated a regularity of pattern of anaesthesia and a conformity to nerve trunk distribution in most cases that could not be due to terminal infiltration. Moreover, it is well known that all affected skin shows the typical perineural accumulation of round cells, even where there is no clinically demonstrable sensory deficit in the area from which the biopsy has been taken.

Why, then, should certain portions of the nerve be relatively immune to involvement? In the case of the median nerve the site of predilection is the part of the nerve just proximal to the flexor retinaculum. Where the nerve lies under cover of the retinaculum it again assumes a relatively normal appearance, only to become involved again in its terminal cutaneous and muscular branches. Why does the radial nerve enjoy a relative immunity to affection —except in its terminal cutaneous branches, where it curves around the radius? Examining these commonly affected sites and the relatively immune sites we see that the nerves in these areas have some factors in common, viz:—

(a) They are superficially situated and not covered by layers of muscle. At operation it is so often seen that the nerve begins to assume a normal size and appearance at the point where it becomes covered by a layer of muscle fibres.

(b) They are in relation to bone or fascia: the ulnar nerve just proximal to the ulnar groove under the medial epicondyle of the humerus; the median nerve proximal to the anterior surface of the carpals; the radial nerve (terminal sensory elements) just as it winds around the lower end of the radius. And when this nerve is affected totally the lesion is in the part of the nerve which is in relation to the lateral intermuscular septum in the upper arm. (In this position the nerve is more deeply situated than the corresponding sites of predilection of the ulnar and median nerves. Does this factor account for the infrequency of its involvement?)

Regarding factor (a) Brand[1] has pointed out that the more superficial tissues of the body are at a lower temperature; the nerves

lying in the subcutaneous fat are at a lower temperature than nerves lying under a layer of muscle; the nerves of the extremities are at a lower temperature than the ones in the trunk; and the nerve terminals in the skin will have the lowest temperature of them all. It is an established fact that certain varieties of mycobacterium require a lower temperature for optimum growth. Thus *M. ulcerans* isolated from an indolent ulcer on human skin will not grow at 37° C. but when first isolated only grows at 33° C.[7] Even though, in modern times, leprosy is commoner in the tropics, the clinical manifestations of the disease are usually more severe in the colder climates.[3]

Regarding factor (b) the proximity to bone and fascia, especially when these lie near the surface, obviously renders the nerve more liable to minor trauma. Moreover, these structures are also relatively avascular and therefore likely to be at a lower temperature than highly vascular structures such as muscle. It is to be noted that Cochrane[3a] states that in the Mongolian or European, alopecia following leprotic infitration of the scalp affects the entire scalp with the exception of the areas in immediate relation to the superficial temporal artery or the occipital artery. Is the factor in this case also the temperature, which will, of course, be higher in the immediate vicinity of the artery?

It must be accepted that the bacilli probably find their way into the system via the peripheral nerve terminals,[9] that there the damage they do is great, that the bacilli then " migrate " either up the axon filaments,[9, 8] or along the lymphatics accompanying the nerve or along the vasa nerorum.[6] These bacilli can, however, only do damage in those parts of the nerve where conditions are favourable.

Summary

1. A study of 304 hand charts was made and the pattern of sensory loss examined in each case.
2. Of the incomplete cases the commonest pattern was ulnar loss. And the area which retained sensation most commonly was the middle of the median area. This suggests that the anaesthesia is not due to peripheral nerve fibre destruction but to the proximal nerve trunk lesion at the site of predilection.

3. These findings were related to the motor paralysis of the same hand. Thus, in the ulnar nerve motor and sensory involvement is commonest; in the median nerve motor involvement is slightly more common than sensory involvement; in the radial nerve motor involvement is rare, sensory involvement is common.

4. The possibility of the temperature of the nerve fibre being a factor in determining the site of predilection of the nerve is discussed.

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ROUTINE TREATMENT OF LEPROSY WITH DDS

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A comparison is made between injections of DDS suspended in hydnocarpus esters, and DDS tablets taken orally. A comparison is also made between inpatient and outpatient treatment in 11 groups of patients over a period of 3 years, improvement being estimated by the bacterial index (B.I.). This is based on careful examination of smears from at least five smears taken from areas where bacilli were considered to be most numerous.

The results are tabulated, giving the numbers in each group (inpatients or outpatients), the weekly average dosage of DDS (injected or orally), the average initial B.I., the percentage reduction of the B.I. after 3 years' treatment, the average distance outpatients had to travel to reach the clinic, and the average interval between attendances.

IMPROVEMENT IN THREE YEARS' TREATMENT

	Number of outpatients	Number of inpatients	Average mgm. per week DDS Injections	Average mgm. per week Tablets	Initial B.I.	% reduction of B.I.	Average miles from clinic	Average week interval attendance
1. ...	28	—	159	—	4.0 to 3.1	57.6	13	2.31
2. ...	12	—	148	—	3.0 to 2.1	65.6	13½	2.5
3. ...	8	—	130	—	2.0 to 1.1	81.5	12½	3.0
4. ...	7	—	164	—	1.0 to 0.1	84.1	8½	2.5
5. ...	—	39	330	—	4.0 to 3.1	55.6	—	—
6. ...	—	14	332	—	3.0 to 2.1	72.5	—	—
7. ...	—	17	335	—	2.0 to 1.1	80.0	—	—
8. ...	—	9	346	—	1.0 to 0.1	75.3	—	—
9. ...	—	39	—	592	4.0 to 3.1	58.3	—	—
10. ...	—	12	—	627	3.0 to 2.1	54.9	—	—
11. ...	—	12	—	700	2.0 to 1.1	53.5	—	—

Discussion

Note that the improvement in the Bacillary Index is over 50 per cent in all groups, the greatest improvements being in the out-patients (groups 3 and 4) on DDS injections, and having original indices of 2.0 to 1.1 (81.5 per cent) and 1.0 to 0.1 (84.1 per cent). The small average weekly doses in these two groups, 130 and 164 mgm., are of particular interest and the results compare favourably with group 11 where the B.I. improvement is only 53.5 per cent, although the weekly oral dosage averages 700 mgm. On the whole the improvement appears to have been more rapid under injections than under oral administration.

It should be noted that apparent improvement is slowest among the injected patients in the groups with the highest indices, namely Nos. 1 and 5, with B.I. 4.0 to 3.1. This is because these represent a massive infection, often many times that represented in 3.0 to 2.1.

The greatest contrast lies between group 7 and 12, both with an initial index of 2.0 to 1.1, the former showing an improvement of 80 per cent and the latter of only 53.5, and this in spite of the fact that the average weekly dose in the latter was nearly twice that in the former.

The average intervals between attendances of outpatients varies from 2.31 and 3 weeks, but even with the longer period an improvement of 81.5 per cent is shown.

In group 5 the average fall in the B.I. increased from 55.6 per cent at the end of 3 years' treatment to 83.6 per cent at the end of 5 years; in group 6 from 72 to 88 per cent. In group 7, 10 patients out of 17 patients had become negative by the end of the 5th year, and in group 8, out of 11 there were 6 negatives, 2 more not being available for examination, and the three others being only very slightly positive.

The number of new outpatients attending from 1949 to 1955 showed a yearly average of 709, rising from 560 in the former year to 900 in the latter, but of these 30 per cent did not return sufficiently often to benefit, while another 15 per cent were irregular in attendance.

REVIEWS

International Journal of Leprosy, Vol. 23, No. 4, Oct.-Dec., 1955.

The original articles are as follows:—

C. B. Lara and J. O. Tiong write on the *Problem of the Negative Inmates in the Culion Sanatorium.* Recent investigation

showed that 440 of the patients, about 25 per cent, had been clear of infection sufficiently long to allow them safely to return to the community-at-large. These are divided into adults (268) brought from different parts of the country, and those born in the institution (172). Of the first category 81 per cent are not ready or are unwilling to go home. Of the latter category many would like to be discharged, but they have no relatives to whom they could be sent. Of the first class of 268 there are 171 with deformities, 91 of them being males. Of the latter, 61 per cent would be able to support themselves if Government help were withheld, and the same proportion of female patients with deformities would be similarly able. Almost all those without deformities would be able to support themselves. Out of the 1,700 inmates in the sanatorium only about 40 per cent were taking treatment, some complaining of unconfirmable subjective symptoms and others unwilling to become negative for fear of being discharged from the sanatorium where they have security and a Government allowance. Of the 172 born in Culion 44 were orphans and, with the exception of one girl who had taken diasone, all had recovered from leprosy spontaneously with only very rarely a tendency to relapse, thus showing high resistance. Of those born in Culion and who were of age, almost all would be able to earn their living. The following recommendations are made to provide for the immediate and eventual relief of the inmates: an experienced and sympathetic social worker to maintain liaison with relatives at home; means to secure the property of inmates; means to improve the crops and livestock of the inmates; better education; and security of land tenure. Lastly, apprehension is expressed at the danger of many negatives remaining in the sanatorium and unrestricted opportunity to marry with the patients, which would eventually lead to a large number of births and infection of fresh generations of children.

The "Acute Infiltration" Reaction of Lepromatous Leprosy is the subject of a paper by I. T. Tahiri. He describes two forms of reaction in tuberculoid leprosy, one milder and the other more acute (*"acuter Schub"*). He describes three forms of reaction in lepromatous leprosy: (1) an acute reactivation caused by an increase of bacillus-containing cells; (2) *erythema nodosum leprosum* most frequently in the resorptive phase under sulphone treatment; (3) an acute infiltration syndrome, which is the subject of this article. It occurs abruptly in lepromatous cases, sometimes overnight, with an erysipelas-like eruption. "The acute infiltration lesion itself consists mainly of infiltration of lymphocytes, plasma cells and epithelioid cells, sometimes with Langhans or atypical

giant cells. Young lepra cells may be present in the new lesions, but older lipid-containing cells are usually absent. Seldom, if ever, are they found anywhere except outside the newly developed lesions, as if they had been pushed aside by the infiltration. Needless to say, lipid-containing cells are seen in the sites of resorption of old lepromatous lesions." In 22 of the 24 cases described the Mitsuda test was positive, and this may be temporary or more permanent. The differences between this and the reactional tuberculoid are that in the former the Mitsuda reaction is less strong, and the reaction occurs in patients with the lepromatous and not the tuberculoid type. It is suggested that, as in the border-line form the tuberculoid turns into the lepromatous, so this reaction may be a sign of the reverse process: the lepromatous (formerly borderline) regaining active resistance and returning to the tuberculoid.

Observations on the Morphology of Mycobacterium leprae by Ordinary Optics, Phase Microscopy, and Electron Microscopy, by K. R. Chatterji, N. N. Das Gupta and M. L. De. Material for examination was obtained from untreated active disease by trituration of fat-free biopsy material in physiological saline, and centrifugation. The material for the electron microscopy was fixed with osmic acid vapour, 2 per cent, for 10 minutes. The living bacilli were examined by phase microscopy. Comparable forms were found by all three methods: a short oval type of cell with 1 or 2 polar condensations; elongated types with double polar condensations; very long types with alternate light and dark zones; homogeneously dark elongated types. " From the study of these different variants it seems to us that there possibly exist two phases of the growth cycle—a slow phase of multiplication resulting in solid homogeneously dense forms, and a rapid phase resulting in forms possessing alternate light and dark regions." The various forms seen are illustrated by photomicrographs.

E. Gehr writes on *The Mitsuda Reaction with the Dharmendra Antigen in Various Groups of Healthy Persons in Surinam.* In all 1,499 persons were tested with Dharmendra's antigen. These are divided into 8 different racial groups. The positive reactions in non-leprous people are numbered as percentages as follows in these groups: Medical and nursing staff working at leprosy clinic and leprosarium 94, Creole contacts 64, Creoles in coastal zone 49, Creoles in mental hospital (all adults) 59, East Indians (descendents of immigrants from India and Pakistan) 21, Indonesians 25, Bush Negroes 70, and American Indians 26. The remarkable feature is the comparatively large percentages of positive reactions in Bush Negroes among whom leprosy is un-

common compared to that among the Creoles. Tuberculosis is not likely to be the cause as it is uncommon among the Bush Negroes who live at a long distance from the town.

In a paper entitled *Nephrotic Syndrome in Leprosy; Dysproteinaemia,* G. Tarabini-Castellani studies the blood protein in 10 nephrotic cases of lepromatous leprosy. In proportion to the advance of leprosy he found a decrease in the total proteins. The albumen fraction is always decreased, but if this loss is compensated by an increase of total proteins then function is maintained and the prognosis is not so bad. The alpha 1 globulin almost always increases, but irregularly, and the beta globulin always increases. The same blood protein picture is found in lepromatous cases without renal symptoms, and it is therefore concluded that it is not proteinuria which causes it, but leprosy.

B. Gozsy and L. Kátó write, in their series of studies on the effects of phagocytic stimulation, on the *Action of Chaulmoogra Derivatives on Endothelial Cells of Skin Vessels.* With a view to studying the effect of chaulmoogra oil on the cellular defence mechanism in leprosy the following experiments were carried out. Twelve different substances including histamine bihydrochlorate, chaulmoogra oil and 9 chaulmoogra derivatives were applied to the depilated abdomens of white mice. The histamin was applied in a 0.5 solution in 70 per cent ethyl alcohol, and the chaulmoogra and derivatives in linseed oil. Immediately after the application (to 4 mice each preparation) 0.5 cc. of india ink mixture was injected intravenously in the tail of each animal. The degree of phagocytic activity was assessed by the degree of pigmentation of the skin. This was read off after 2 and 24 hours against a control of linseed oil alone, degrees of pigmentation being marked from zero up to 4 plus. The chaulmoograte of sodium, magnesium and barium and the control gave negative results. The histamine salt gave 4 plus, as did chaulmoogra oil (20 per cent) and its various esters after 24 hours. Histamine, 20 per cent chaulmoogra and its 1 per cent benzyl ester gave 4 plus at the 2-hour reading. "This similarity of action of both histamine and chaulmoogra derivatives on the endothelial cells of skin vessels, and the fact that for both cases the induced phagocytosis could be inhibited by an antihistaminic, permits us to suppose that this action on the part of the chaulmoogra derivatives is brought about as a consequence of the liberation of histamine. However this hypothesis remains to be proved by direct quantitative methods."

The same authors also write on the *Action of Chaulmoogra Oil on the Reticuloendothelial System.* Following on the evidence in the

last-described experiments that chaulmoogra derivatives acting like histamine stimulate the activity of endothelial cells in the skin, a further trial was made to find out whether chaulmoogra derivatives also resembled histamine in slowing the disappearance from the body of india ink injected into the vein. Out of 250 albino rats 50 were injected with histamine subcutaneously, 50 with Benadril, and 50 on two occasions with chaulmoogra oil. India ink suspension was injected intravenously in the first 2 groups 30 minutes after the previous injection, and in the third 1 hour after the latter chaulmoogra injection. Controls consisted of another 100 rats which were given only the solvents previous to the india ink. The ink disappeared in the controls within 39 to 68 minutes. The disappearance was delayed in the antihistamine group and accelerated in the histamine and chaulmoogra groups. This gives further evidence that chaulmoogra acts in leprosy in stimulating the cellular defence mechanism of the host to increased activity.

G. L. Fite and H. W. Wade review the known facts about *Albert Neisser's participation in the discovery of Hansen's Bacillus and the establishing of it as the cause of the lesions of the disease of leprosy.* In 1874 Hansen observed and described the bacillus; but his observation was very defective and he was unable to convince others, including Danielssen his chief, as he had not at that time the technique and the stains necessary for showing the bacillus clearly. In 1879 Neisser went to Bergen and obtained material from Hansen and applied new methods of staining according to Koch's method, which showed up the bacilli clearly. Thus Neisser confirmed and expanded the work of Hansen. Meanwhile Hansen had also obtained the later staining methods from Koch, but was slower than Neisser in publishing the results. While Neisser was a bacteriologist, Hansel was a leprologist, and did far more for the campaign against leprosy than discovering the etiological agent.

REPORTS

Dr. A. McKelvie writes from the Gold Coast: " I am now sending you statistics of people under treatment for leprosy at the end of 1955. From these, you will see that the work grows. The biggest increase in the number of people under treatment has taken place in the Northern Territories where the number of patients has increased from 6,909 to 12,986 within the year. This has been due to the efforts of Mr. D. G. Turner and Mr. R. Boteler, Leprosy Control Officers who were recruited for the Gold Coast by BELRA

" There are now treatment centres 10-15 miles apart along

all the main and most of the secondary roads of the Gold Coast and we are continuing to open centres along dry season roads. These last will be served by junior staff travelling on bicycles and will be supervised by senior staff when roads are passable. If Landrovers are forthcoming from the United Nations Children's Fund, this will make our work more effective.

" The results of twice weekly DDS therapy continues to be satisfactory. On a visit last month to a group of clinics in the Northern Territories run by a missionary society, I discharged 373 out of the 1,436 persons examined, about 26%. The discharge rate for persons who had received treatment for 18 months or more and suffered from the non-lepromatous types of the disease must be about 90%. In the part of the country I am referring to, one cannot yet speak of leprosy control being established because so many immigrants come over the border from French Territory, being attracted partly by the availability of free treatment partly by the better economic conditions which prevail in the Gold Coast. In addition, a number of nomads cause even greater confusion to the would-be statistician.

" The unsatisfactory feature of leprosy work here is the lack of doctors. Until a few months ago, I was the only doctor engaged in leprosy work in the 91,800 square miles of the Gold Coast. Now there is a second man available for the time being. Consequently, more travelling is being done and the thousands of people who are more than ready for discharge will soon be examined and receive their certificates."

LEPROSY TREATMENT CENTRES IN THE GOLD COAST

Region	Clinics Open	Lepromatous Patients Total	Non-Lepromatous Patients Total	Grand Total
Colony East	48	507	3133	3640
Colony West	59	434	1994	2428
Ashanti	47	413	4194	4607
Trans-Volta Togoland ...	38	214	905	1146
Eastern Northern Territories	82	884	7859	8743
Western Northern Territories	41	539	4704	5243
Unclassified	5			168
TOTAL	320	3018	22789	25975

Kuching Leprosy Settlement, Sarawak

In the Annual Report of the Kuching Leprosy Settlement, the number of patients at the beginning of the year is given as 449.

Of these 113 were discharged on " symptom-free parole leave," and 69 more patients were admitted. Of the 398 at the end of the year, 156 were Sea-Dyaks, and 146 Chinese. Mr. Hamish Macgregor, the Superintendent, worked for many years in the Leprosarium at Itu, Nigeria. The following abstracts from the Report are of particular interest:—

" Perhaps the most interesting point in regard to the admissions is that a number of patients came along for treatment of their own accord, and without waiting to be sent. In addition to this a number of others were brought in by relatives or friends from the settlement who were home on parole leave, and either saw or heard of them and persuaded them to come for treatment.

" The number of discharges, 113, represented 25% of our population, and is the highest in the history of the settlement; a further proof of the efficacy of the sulphone drugs. This figure compares with 59 in 1953 and 34 in 1952."

This year the patients sent a present to Sir Winston Churchill of a beautifully beaded forest palm hat and a hunting knife.

Annual Report of the Medical Department, Tanganyika for 1955.

There was no indication that there was any alteration in the incidence of the infection during 1955. Admission to leprosaria followed the pattern of previous years, and although more patients came under out-patient treatment, this was the result of expansion of facilities and greater attention being paid to the disease, rather than to any increase in incidence.

In several districts the development of organised out-patient treatment with sulphones made good progress. This was especially so in the Southern Province with the development of the treatment centres in Newala, Masasi and Mtwara Districts based on the Mkunya Leprosarium. In Morogoro also good progress was made and substantially more patients were brought under treatment.

Out-patient treatment is readily organised in areas where communications are good and populations are concentrated. Where the population is scattered as in such districts as Singida and Kilosa, it is not such a straightforward matter, and in these districts progress has been less marked.

Report on Public Health, S. Rhodesia, 1953

Leprosy

Information regarding the patients under treatment in the two leprosaria is given in Table A of the Appendix. An the end of 1953, for the first time for very many years, there were no non-

African patients under treatment in these institutions. The admission and discharge figures of African patients for the past five years are of interest:

	1949	1950	1951	1952	1953
Admissions	314	330	367	330	295
Readmissions	101	104	118	119	102
Discharged cured or arrested ...	208	253	207	384	448
Deserted	52	71	66	38	94
Died	54	56	29	33	28

Admissions have not varied greatly but there has been a big improvement in cases discharged cured and arrested. The overcrowding has therefore been greatly eased. The success of the sulphones in treatment is already providing much encouragement to indigenous patients to come forward voluntarily for treatment, since cured and arrested cases return to their homes and, from the knowledge they spread, other sufferers come in for treatment. In any case a high proportion of the cases come from neighbouring territories; at Ngomahuru, of 132 male admissions only 48 were Southern Rhodesians. In fact a number of alien cases are known to have come into the Colony ostensibly to seek work, but in fact to seek admission for treatment of leprosy.

All patients are now on DADPS therapy and making good progress. The present routine is one tablet (100 mgm.) daily six days a week for six weeks, and thereafter a maximum dose of two tablets daily, six days a week. Reactions are infrequent and of a mild nature. Ferrous sulphate is also given as a routine.

Report on Leprosy in the Sudan

The results of a survey done by a lay worker of BELRA in Central District of Equatoria during 1951/52 and 1952/53 became available. Over 27,000 persons were examined. The incidence of leprosy was 44 per 1,000. This may be compared with the figure of 52 per 1,000 reported by Abbott amongst the Azande and that of 20 per 1,000 resulting from a less extensive BELRA survey in the Moro district. The Medical Officer, Li Yubu, considered that the incidence in the district was as high as 65 per 1,000.

The total number of lepers in settlements in Equatoria was 1,329. This figure was less than 12 per cent of the total known lepers in the province, while the surveys indicated that the actual number of cases in the province is certainly greater than the total known cases.

A majority of cases of leprosy in Equatoria is of neural type. In the Central District survey only 10.2 per cent of cases were classified lepromatous.

BCG VACCINATION

[In view of the possible rôle of BCG in increasing resistance to leprosy, the conclusions reached in a recent paper appearing in the British Medical Journal, Nov. 12th, 1955, on *Protection of Infants against Tuberculosis*, may be of interest to readers.]

That infants are at risk and that this cannot be prevented. That vaccination at birth is safe, practicable, and effective, though it does not obviate the need for segregation and for general hygiene and education in hygiene. That it would appear that vaccination is effective in protecting throughout the danger period of infancy, though we do not know whether revaccination may not be necessary at 10-12 years; this may well depend on any exposures in the intervening years. That, of all the vaccines we have used, quickest conversion is afforded by the standard Danish BCG. That a half-strength vaccine—that is, 0.375 mg. per ml.—is preferable for this work in infants, as it allows for more accurate dosage. That the complications are mainly directly proportional to the antigenic potency of the vaccine and to the dose given. That until the tuberculous antigen is isolated, vaccination with live attenuated bacilli offers a sound means of protection, and, because of the urgent need of this protection from the earliest moment, vaccination may most profitably be undertaken in the newborn period despite the slightly increased risk of glandular complication at this early age, provided the vaccine is given in accurate dosage, by an experienced person.

That when potent freeze-dried vaccine is available generally the problem of "shortevity" and temperature control will no longer cause difficulty in distribution. A freeze-dried vaccine, free from clumping when dissolved, and containing a known number of evenly distributed and living bacilli, allowing of accurate controlled dosage, will be a great step forward.

That in view of the need for careful follow-up and the difficulty of getting sufficient trained personnel it would not be practicable to carry out mass vaccination in the newborn at present even if that were desirable. Nevertheless, where facilities exist—that is, in maternity hospitals and where there is experienced paediatric supervision—we believe that vaccination in the newborn period offers a very real contribution to the protection of infants against tuberculosis, and we consider that it might with advantage become an accepted part of the "care of the newborn" in maternity hospitals. But, apart from such hospitals, we think that it should at present be reserved for infants who are known to come from, or be going to, tuberculosis households, and in these it

should be a routine procedure as soon after birth as possible—that is, during the first week of life. Many such infants are, of course, born at home or in district maternity homes, and not every district has a paediatrician, but there are maternity and child welfare doctors in all areas, and one or more of these from each area could be trained to undertake this special work without its adding greatly to his or her existing clinical commitments, and so all infants at known risk could in fact be satisfactorily vaccinated.

I would conclude with the warning that with the changing pattern of tuberculosis and the rapidity of introduction of new antituberculosis chemotherapeutics our future programme may well differ significantly from that which I have outlined.

ABSTRACTS

Wound Healing, by **Paul Brand, F.R.C.S.:** New England Jl. of Med. (1956), 254, p. 64.

No properly controlled experimental study of wound healing in leprosy has yet been carried out. In this clinic, however, over 800 operations on the hands and feet of patients with leprosy have been performed, and the healing process has been followed with some care and attention. In the same clinic many orthopaedic and plastic reconstructions have been carried out on the hands of non-leprous patients.

It has been my impression that although most wounds in leprosy patients heal normally, delay in union may result from a poor nutritional state, which is common in these patients. Also, anaesthesia of the wound area deprives the patient of normal protection. It is good practice, therefore, to leave the skin sutures in place for two or three extra days. Gross sepsis has occurred in only one case, although stitch abcesses and other superficial infections have been about as common as in the hands of non-leprous patients.

Skin flaps and tubes have been transferred, the same rules as apply to healthy persons being used, and with at least as good results. Free grafts have taken with more regular success than in other cases. " Tendon suture and free tendon grafting have been performed in hundreds of cases and with more regular success than in any similar series that I have seen of traumatic or other injuries." This success is probably due in part to the fact that post-operative exercises are not inhibited by pain.

*Tropical Diseases Bulletin, Vol. 53, No. 1, January, 1956.

On the Advantage of Using Diluted Lepromins, by **H. Floch.** Bull. Soc. Path. Exot. 1955, V. 48, No. 3, 367-71.

The author shows by means of a curve a diminution of about 17 per cent. of positivity by using in the Mitsuda test an antigen diluted 1/750 as compared with one diluted 1/20, but with a dilution of 1/1000 it was diminished by 40 per cent. On the other hand with diluted antigen (1/750) he obtained 90 per cent. of positivity in 46 tuberculoid type children, and 59 per cent. in 58 undifferentiated type children; whereas with the undiluted he obtained 93 per cent. and 43 per cent. in 43 tuberculoid and 85 undifferentiated respectively. Using the diluted antigen (1/750) there were found 78 per cent. of conversions of negative to positive Mitsuda reactions after intradermal inoculation of BCG, whereas in Brazil they obtained 100 per cent. of conversions using massive oral doses of BCG and testing with undiluted lepromin antigen. The conclusion is to recommend, chiefly with a view to conserving an antigen difficult to obtain, the use of 2 dilutions: one 1/150 and the other 1/750.

*Tropical Diseases Bulletin, Vol. 53, No. 2, February, 1956.

Radiological Appearances of Lesions of the Hands seen in Leprosy Patients in the Congo, by **J. Chardome** and **M. Lechat.** Ann. Soc. Belge de Méd. Trop. 1955, June 30, No. 3, 267-78.

The hands of 126 patients in the Yonda leprosarium were radiographed, and the lesions found are described in the text and by means of 9 photographs. The bone changes were: resorption especially at the ends of the fingers and not different from those found in other neurotrophic troubles such as psoriasis, syringomyelia, Charcot's disease and Raynaud's disease; decalcification and osteoporosis present in 28 cases; osteitis was rare and was considered to be the result of injuries and other infections; cavities in the carpus were found in 17 cases, and these were not confined, as other authors had found, to the lepromatous type; blocking of the nutrient canals especially when there was resorption of the terminal phalanx; changes in the interphalygeal joints; and changes in sesamoid bones of the thumb. An attempt is made to classify the frequency of the lesions found according to the type and the duration of the disease. (There is no discussion of the mechanism of the bone changes.)

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***Tropical Diseases Bulletin, Vol. 53, No. 3, March, 1956.**

Isoniazid in Leprosy, by **R. F. R. Scragg.** Trans. Roy. Soc. Trop. Med. & Hyg. 1955, Nov., V. 49, No. 6, 548-54 [11 refs.].

Five lepromatous type patients and 1 tuberculoid, who had responded poorly to sulphones or had suffered from repeated reactions under sulphone treatment, were placed on isoniazid treatment alone for 17 to 26 months. The daily dosage ranged from 3.9 mg/kg. to 9 mg/kg. of body weight. In 4 of the 5 lepromatous cases there was clinical improvement, but none in the tuberculoid. There was an improvement in the bacteriological findings of the 5 lepromatous cases, the mean index diminishing from 6.4 to 0.5. Two of the patients developed lepra reactions during treatment, and 2 of them had severe neuritis. The results obtained differ from those of other workers who have noted no improvement. The author attributes this to the larger doses used. (As there were no controls there is nothing to indicate to what extent the good results were attributable to delayed action of the sulphones given previously.)

***Tropical Diseases Bulletin, Vol. 53, No. 5, May, 1956.**

The Leprosy Problem in Brazil, by **H. C. de Souza-Araujo.** Mem. Inst. Oswaldo Cruz, 1954, June v. 52, No. 2, 427-41, 22 figs. on 7 plates.

The author first described leprosy in Brazil in 1925, when efforts at control were first being initiated. There were then 1,963 cases in primitive leprosaria, 9,002 known cases, and a total estimate of 24,000. From 1946 to 1950 there were 22,245 new patients recorded, of which 56.5 per cent. were open cases of leprosy. At the end of 1950 there were 60,623 known cases, 1.12 per thousand of the population. Of these there were 21,917 in the State of Sao Paulo, and 13,591 in Minas Gerais. There were in 1952 23,421 patients in leprosaria, of which 7,901 were in the State of Sao Paulo, and 5,465 in Minas Gerais. The total amount spent by the Union on leprosy in 1951 was the equivalent of U.S. $1,800,000. There are also 29 preventoria for children of patients, housing in 1952 some 4,045 children. There are also 93 leprosy clinics. At least 200 dispensaries are needed for follow-up work, the treatment of closed cases, and after treatment of discharged patients. There are 4 centres for research and training. Many details of the incidence in the various States are given; in some of them leprosy is considered a very serious problem.

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*Tropical Diseases Bulletin, Vol. 53, No. 6, June, 1956.

Hydnosulfone in the Treatment of Leprosy, by **Dharmendra** and **K. R. Chatterjee.** Leprosy in India, 1955, Vo. 27, No. 4, 230-33.

Hydnosulfone is a condensation product of hydnocarpic acid with DDS. It is a slightly yellowish powder with a molecular weight of 716, and a melting point of 122°C. It is insoluble in water and ether. It is similar to Chaulfone, a French preparation, but differs in retaining the original saturated group. It is well tolerated, does not produce any toxic effects, and does not break down into its component parts in the body when taken orally. It was found too irritant for intramuscular injection. In only 4 out of 26 patients treated was there a slight initial reduction in eryhrocytes and haemoglobin. There was, after treatment varying from 30 to 100 weeks' duration, complete or almost complete clinical subsidence in 8 of the 18 lepromatous patients, 3 of them becoming bacteriologically negative. In 6 others there was considerable clinical and bacteriological improvement. In 7 tuberculoid cases there was subsidence of erythema and thickening. Most of the patients included in this trial had previously been treated without any, or lasting, effect. It is concluded that Hydnosulfone is of definite value in the treatment of leprosy, particularly in patients who cannot tolerate, or do not make satisfactory progress with, some other drugs, such as DDS, isoniazid and thiosemicarbazone.

The Value of Vaccination with BCG in the Prophylaxis of Leprosy, by **V. Pardo-Castello, F. R. Tiant** and **R. Ibarra Pérez.** Bol. Soc. Cubana de Dermat. y Sifil. 1955, Sept. v. 12, No. 3, 144-53.

Twenty-two children with negative Mitsuda reactions were given 100 mgm. of BCG by mouth once a week for 3 weeks. Three weeks after the vaccination the Mitsuda reaction was positive in 9 and negative in 13. Of these 22 children, 15 were the children of These 15 were given another similar 3-week course of BCG. Three weeks after this revaccination the Mitsuda reaction had become positive in five who had been negative before; in four who had been positive or doubtful before the reaction had become negative; and in the other six cases the reaction remained the same.

It is acknowedged that these results are not as good as those obtained by some workers, though they correspond to those found

by others. The authors still consider that BCG increases resistance to leprosy, and uphold its universal use where leprosy is endemic.

Regarding Sulphone Treatment of Leprosy, by **L. Swerts.** Ann. Soc. Belge de Méd. Trop. 1955, Dec. 31, v. 35, No. 6, 785-800.

The author working at the Red Cross of the Congo anti-leprosy centre at Nepoko reviews the treatment of 2,500 tuberculoid cases of leprosy and 800 lepromatous cases over a period of 3 years. As regards the tuberculoid macules, these disappeared more or less completely in 90 per cent. But the thickened nerves were more difficult to assess as to their improvement, as the thickness might diminish without any corresponding improvement in sensation. There was, however, a gradual improvement in about 40 per cent. of cases over the 3 years. Much better results as regards function were obtained in patients with only a single tuberculoid lesion than in those with multiple lesions. With the lepromatous type there was 90 per cent. of definite improvement in the skin lesions, though the nerve lesions were little improved. The bacteriological findings became negative in 11.24 per cent., and very much reduced in 37.85 per cent. at the end of the 3 years.

Tests with Tuberculin and Lepromin in the Makoda Chiefdom by **L. Swerts.** Ann. Soc. Belge de Méd. Trop. 1955, Dec. 31, v. 35, No. 6, 801-4, 1 chart.

Preliminary to BCG vaccination in the Makoda Chiefdom, tuberculin and lepromin tests were made of the population. For the Mantoux test a dilution of 1/1000 was used and readings made after 48 hours. Positive readings were with 10 to 20 mm. infiltration and upwards. For the lepromin an antigen was used composed of a suspension of 0.4 gm. of dried leproma per 100 cc. of saline. Positive reactions were when there was a nodule formed of over 4 mm., the reading being made after 3 weeks. The Mantoux and Mitsuda reactions are compared, and also males and females in each of these categories. The results are shown in curves. With the tuberculin the curve increases at first very slowly up to the 6-10 period, accelerates for the 10-15 period, then rises abruptly at 16-20. Up to 15 years the curve of the 2 sexes is practically the same, but after that the male curve rises more quickly than the female. They stop rising at 50 to 60 per cent at the age of 36 to 40 years, but the curve for the men rises higher than that for the women. The lepromin curves are quite different. There is not the initial latent period, and they rise much more abruptly, the 2 curves overlapping till a maximum is reached of 65 to 75 per cent. at the age of 16 to 20 years.

The two main differences between the tuberculin and the lepromin reactions are: (1) The absence with the lepromin of the initial latent period before the curve starts to rise, and (2) The more abrupt rise of the lepromin curve, so that it reaches the maximum earlier and at a higher percentage level. This is explained by a supposition that the people in this region are impregnated with leprosy at an earlier age than they are with tuberculosis. The fact that the lepromin reaction is converted earlier than the tuberculin does not support the idea that the conversion of the lepromin reaction is due to Koch's bacillus.

Induced Immunity in Leprosy, by **P. C. R. Pereira,** *et al.* Arquivos Mineiros de Leprologia, July, 1955, p. 172. (Authors' Summary.)

The authors, working as a team, established a plan of study and have vaccinated with the bacillus of Calmette and Guerin thousands of contacts and of other persons resident in leprotic areas. A clinical examination of the patients is made, the possibly infected cases separated and the others receive Mitsuda's antigen.

All the negative cases are treated with BCG and after a period of from 30 to 6 days are again treated with Mitsuda. The great majority (93.4 per cent) showed the Mitsuda change. The authors hope to keep these patients under constant observation during a period of from 5 to 10 years to determine whether they have really become immune to leprosy.

The authors make a large number of observations as regards the data obtained and suggest the continuation of the study of this interesting subject.

The experiments were begun more than 3 years ago (about 1953) and the paper is illustrated by various tables and graphs, showing the index of positive Mitsuda reaction before and after BCG.

Dr. de Souza Lima writes in the Memoirs of the Third Panamerican Conference on Leprology on the *"Pseudoexacerbation" Reactional State of Leprosy.*

The term "pseudo" is used because the reaction of this nature results not in an actual exacerbation of the disease, but in improvement and sometimes in complete clearing up. It occurs in patients of the lepromatous type under sulphone treatment, and yet it has the "clinical appearance of the reactional tuberculoid eruption." Histological examination of the lesions may reveal

tuberculoid and frankly lepromatous features side by side, and there may be a reactional tuberculoid histological picture without the corresponding clinical aspect. The lesions of the pseudo-exacerbation may replace those of the lepromatous type, mask them, or coexist with them. The aftercourse may be that of the reactional tuberculoid or that of the lepromatous type. The auhor describes two groups of patients. Group 1 consisted of 29 patients. The reaction appeared suddenly and without affecting the general condition, though in some there might be slight fever lesions might appear, and there might sometimes be severe neuritis followed by muscular atrophy. The duration was usually 3 to 6 months. There was complete clearing up of all the cases in this group although in 22 of them the disease had been more or less advanced. In the second group of 61 patients lepromatous and tuberculoid lesions coexisted, but in spite of this the pseudo-reaction was beneficial, leading to clearing up much more rapidly than usual, so that 44 could be transferred to outpatient clinics. In all but two cases the lepromin reaction remained negative, and in them it was only slightly positive.

CORRESPONDENCE

[In reply to the editorial request in the last issue for evidence for or against drug resistance to sulphones in leprosy, the following letter was received.]

Dear Sir,

Out of 20,000 odd patients treated since April, 1950, we have not seen a single case which shows the normal signs of drug resistance—a steady improvement, followed by lack of response, followed by a recrudescence of the disease while still under treatment, and unable to be controlled by the drug in question. The patient mentioned under " Drug resistance " in my article (*Lep. Rev.*, Vol. 27, p. 58) has not shown any further deterioration, and in fact his last smear was improved.

I find several patients of this nature starting with a large number of bacilli. The number is fairly rapidly reduced, but there seem to be occasional pockets of acid-fast dust, often in the form of globi. These are commonly in the ear lobes, but can be elsewhere. Being small pockets, and often showing no clinical evidence of activity, I presume that it is rather a matter of chance whether the pockets are found on bacteriological examination—hence the variable smear results.

The pockets are very persistent and I have found globi of acid-fast dust nearly two years after smears have become negative in other parts of the body. Dr. Davey is of the opinion that they are probably dead bacilli, not cleared away. We have discussed the significance, from the points of view of both infectivity and fitness to stop treatment.

I fear that the proof can only await the successful culturing of bacilli or a lab. animal which can reliably be infected, or clinical experience of at least two decades.

Some evidence in favour of these bacilli being dead is the fact that defaulters have, after leaving our care with acid-fast dust, returned after a period of time, with none. Unfortunately we cannot take as certainly true, their assertion that they have had no private treatment.

Compared with this we have many reports of drug-resistance after periods of two years or less of treatment with Thiacetazone and Izonaizid.

Another question that may be asked is whether discharged patients whose leprosy has recurred show drug-resistance. In my experience, and that of my colleagues, these patients improve about as rapidly after being placed again on treatment as would be expected of a similar group of patients with untreated leprosy.

There are a few patients who have had fairly severe reactions at a time when it appeared that all signs of the disease were past. They have not, however, shown signs of progressive worsening of the disease, as would be expected if drug-resistance were present.

To sum up, I can see no evidence at present to believe drug-resistance has occurred in leprosy under dapsone treatment, though the period of treatment of very large numbers has already been nearly three times that required for drug resistance to be produced under other drugs. On the other hand, it seems too good to be true that dapsone is the one exception in drugs, and leprosy in diseases, to a rule which is so distressingly wide in its application.

Yours sincerely,
ARTHUR GARRETT.

Oji River,
E. Nigeria.

Dear Sir,

Your editorial remarks and reference to Dr. J. A. K. Brown's article in *Lep. Rev.* of April 1956 raise, as you say, an important

problem. Lepromatous, Borderline and Tuberculoid cases in reaction are all " open " forms of leprosy. You point out that all these may show large numbers of bacilli to routine methods of examination and therefore, presumably, may pass on the infection. I suggest the inability to trace all cases of leprosy to patients falling into these categories is due to the long incubation period and, particularly in parts of Africa, to the changing population. Where there are large areas of country with no obvious lepromatous cases some of us may overlook the fact that many of the cases of leprosy found may AT ONE TIME have been Borderline or Tuberculoid cases in reaction even if no longer exhibiting such features? Further, as you point out, " concealed " (or undiagnosed) cases may spread the infection. I believe many, if not most, cases of diffuse lepromatous leprosy are contagious, perhaps for a long time, before the disease declares itself sufficiently to be recognised by a casual observer or even by the patient himself.

Yours truly,

J. T. WORSFOLD.

Chitokoloki Leprosy Settlement,
Balovale,
Northern Rhodesia.
1st August, 1956.

VINOBA BHAVE AND REHABILITATION OF LEPROSY PATIENTS

On 25th February, 1956, the Dy. Minister for Revenue, Mr. Hanmanth Rao, accompanied by the District Health Officer and Dr. Bhagwan Rao Tandade, Medical Officer, Leprosy Control Pilot Project Scheme at Narayanpet, called on Vinobaji and discussed with him a scheme for the rehabilitation of leprosy patients. Dr. Bhagwan Rao explained the purpose of the scheme and indicated that at an average of 5 acres per patient, at least 250 to 300 acres of land would be required under the scheme. After acquainting himself with every aspect of the plan, Vinobaji agreed to allocate the required acreage under the Bhoodan scheme. This plan when it gets into operation, would considerably alleviate the miserable plight and distress of those unfortunate victims of leprosy to whom society attaches a " stigma " even to-day.

LEPROSY REVIEW. VOLUME XXVII. (1956)

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