Division.	District.	Total Population on which the Leper-ratios have been calculated.	Total number of Lepers.	Proportion per 10,000 of population.	Remarks.
	Bombay—*contd.*				
Deccan	Khandesh	1,460,851	1,699	11·6	
	Nasik	843,582	425	5·0	
	Ahmednagar	888,755	695	7·8	
	Poona	1,067,800	1,078	10·0	
	Sholapur	750,689	607	8·0	
	Satara	1,225,989	1,488	12·1	
	Total for Deccan	6,237,666	5,992	9·6	
Western Karnatic	Belgaum	1,013,261	338	3·3	
	Dharwar	1,051,314	214	2·0	
	Kaladgi or Bijapur	796,339	331	4·1	
	Kanara	446,351	19	·4	
	Total for Western Karnatic	3,307,265	902	2·7	
Sind	Kurrachee	564,880	55	·9	
	Hyderabad	918,646	58	·6	
	Shikarpur	915,497	74	·8	
	Thar and Parkar	298,203	7	·2	
	Upper Sind Frontier	174,548	15	·8	
	Total for Sind	2,871,774	209	·7	
	City and Island of Bombay	821,764	369	4·4	
	Total for Bombay Presidency (British Territory*)	18,857,044 (18,901,123)	10,186 (10,187)	5·4 (5·3)	* Exclusive of Aden. The figures in brackets denote the numbers and ratios inclusive of Aden.

Division.	District.	Total Population on which the Leper-ratios have been calculated.	Total number of Lepers.	Proportion per 10,000 of population.	Remarks.
	Bombay Feudatory States.				
Bombay Feudatory States.	Cutch	558,415	27	·4	
	Palanpur	645,526	54	·8	
	Mahi Kantha	581,568	65	1·1	
	Kathiawar	2,752,404	426	1·5	
	Rewa Kantha	733,506	319	4·3	
	Cambay	89,722	8	·8	
	Narukot	...	...	...	
	Surat Agency	181,208	51	2·8	
	Jawhar	52,831	36	6·8	
	Janjira	81,780	79	9·6	
	Savantvadi	192,948	35	1·8	
	The Dangs	32,920	3	·9	
	Surgana	12,398	6	4·8	
	Satara Jagirs	131,529	76	5·7	
	Bhor	155,669	143	9·1	
	Akalkot	75,774	23	3·0	
	Jath	79,786	14	1·7	
	Kolhapur	913,131	956	10·4	
	Southern Maratha Jagirs	639,270	227	3·5	
	Khairpur	131,937	6	·4	
	Savanur	16,976	*Nil.*	—	
	Total for Feudatory States	*8,059,298*	*2,554*	*3·1*	
	TOTAL FOR BOMBAY PRESIDENCY, INCLUDING FEUDATORY STATES	26,916,342 (26,960,421)	12,740 (12,741)	4·7* (4·7)†	*Exclusive of Aden. †Inclusive of Aden.

Division.	District.	Total Population on which the Leper-ratios have been calculated.	Total number of Lepers.	Proportion per 10,000 of population.	Remarks.
	Ajmere Merwara.				
Ajmere-Merwara	Ajmere	422,359	11	·2	
	Merwara	119,999	16	1·3	
	TOTAL FOR AJMERE-MERWARA	542,358	27	·4	
	Mysore.				
Mysore	Bangalore*	702,913	239	3·4	*Exclusive of Bangalore City (British portion).
	Kolar	591,030	185	3·1	
	Tumkur	580,786	44	·7	
	Mysore	1,181,814	149	1·2	
	Hassan	514,952	69	1·3	
	Shimoga	527,981	55	1·0	
	Kadur	330,063	17	·5	
	Chitaldroog	413,984	44	1·0	
	TOTAL FOR MYSORE†	4,843,523 (4,943,604)	802 (814)	1·6 (1·6)	†The figures in brackets denote the numbers and ratios inclusive of Bangalore.
Coorg	**Coorg**	173,055	23	1·3	
	TOTAL FOR MYSORE AND COORG	5,016,578 (5,116,659)‡	825 (837)	1·6 (1·6)	‡Figures in brackets inclusive of Bangalore City.

Division.	District.	Total population on which the Leper-ratios have been calculated.	Total number of Lepers.	Proportion per 10,000 of population.	Remarks.
	Rajputana—*contd.*				
East State Agency	Bhurtpore	640,303	89	1·3	
	Kerowlee	156,587	20	1·2	
	Dholpur	279,729	73	2·6	
	TOTAL FOR EAST STATE	1,076,619	182	1·6	
Ulwur Agency	Ulwur	767,786	79	1·0	
	TOTAL FOR ULWUR	767,786	79	1·0	
Jhallawar Agency	Jhallawar	343,601	92	2·6	
	TOTAL FOR JHALLAWAR	343,601	92	2·6	
Harowtee and Tonk Agency	Tonk	380,069	53	1·3	
	Boondee	295,675	69	2·3	
	Shahpura	63,646	1	·1	
	TOTAL FOR HAROWTEE AND TONK	739,390	123	1·6	
Kotah Agency	Kotah	526,267	63	1·1	
	TOTAL FOR KOTAH	526,267	63	1·1	

Division.	District.	Total population on which the Leper-ratios have been calculated.	Total number of Lepers.	Proportion per 10,000 of population.	Remarks.
	Rajputana—*concld.*				
Cantonments	Erinpura	1,859	Leper population of these cantonments is stated to have been included with their respective states.		
	Kherwara	648			
	Kotra	150			
	Abu	92			
	TOTAL FOR CANTONMENTS	2,749	—	—	
	Total for Rajputana	**12,126,655**	**1,708**	**1·4**	
	Central India Agency.*				* Incomplete.
Central India Agency	Bhopawar	978,638	1	·01	
	Indore	372,792	1	·02	
	Gwalior	1,757,509	1	·005	
	Western Malwa	1,619,368	36	·2	
	Bhopal	2,006,859	16	·07	
	Bundelkhand	1,508,053	3	·01	
	Goona	337,973	22	·6	
	Baghelkhand	870,184	—	—	
	TOTAL FOR CENTRAL INDIA AGENCY	9,451,376	80	·08	
	Railways and Cantonments.†				† So far as they have not been included in previous figures—incomplete.
	Aden Settlement	44,079	1	·2	
	Andaman Isles	15,609	1	·6	
	Bangalore (Civil and Military)	100,081	12	1·1	
	Central India Railways	4,448	—	—	
	Central India Cantonments	95,238	12	1·2	
	Total for Railways and Cantonments	**259,455**	**26**	**1·0**	

CHAPTER IV.

Hereditary Transmission and Predisposition.

THERE is as little consensus of opinion as to the hereditary transmission of leprosy from parent to offspring as there is with regard to the contagiousness of the disease. It is not necessary to enumerate the views of the various authors, and only a few references need be made. Dr. Liveing[1] inclines to the belief in an hereditary predisposition rather than an actual transmission of the disease, while Danielssen and Boeck maintain that hereditary predisposition and transmission are the chief causes of the perpetuation of the disease in Norway.[2] It should be remarked that the latter authors speak really of an actual hereditary transmission, and not of a mere predisposition. Leloir[3] and Hansen,[4] on the other hand, altogether deny the existence of such transmission or predisposition. Hansen, as is well known, visited North America to enquire into the condition of the Norwegian lepers who had emigrated to that country. Out of 160 original emigrants he found only seventeen alive, and not one of the descendants of these 160 men had contracted leprosy. This fact is most important. Turning now to Virchow[5] it is found that he says: "The existence of an hereditary tendency has been admitted in all ages and in all countries." But

(1) R. Liveing: Goulstonian Lectures for 1873 (Longmans, Green & Co., London, 1873), pages 84-87.

(2) Danielssen and Boeck: Traité de la Spedalskhed. (Paris, 1848): also R. Liveing loc. cit., page 84.

(3) H. Leloir: Traité Pratique de la Lèpre (Paris, 1886), pages 281—296.

(4) A. Hansen: Virchow's Archives, Vol. CXIV., 3, 1888.

(5) R. Virchow: Krankhafte Geschwülste, Vol. II., pages 503—505.

he asserts distinctly that only the predisposition, and not the disease itself, is transmitted: "without special and specific external relations and causes, the disease will not appear in the offspring of leprous parents." He continues: "If it be true that amongst the lepers who emigrated from Norway to America the disease has disappeared, then we must ascribe a very great importance indeed to these external causes." The proof of this has now been given by Hansen. As regards leprosy in India, Dr. Vandyke Carter,[6] in his well-known work, strongly urges the spread of the disease through hereditary transmission; and Drs. Lewis and Cunningham,[7] in their report on Leprosy in India, held "that the hereditary taint exercises a most important influence in the transmission of the pest." However, they never speak of an actual transmission of the disease, but merely of a predisposition.

There can be no doubt that this difference of opinion is in great part due to the fact that authors have not drawn a clear enough distinction between "hereditary transmission of a disease" and "inherited predisposition to a disease." Even now writers talk loosely of the inheritance of carcinoma and tuberculosis, while a predisposition is evidently all that can be claimed. A second mistake, which apparently has been often committed, is that, in their enquiries regarding the condition of the parents, authors have been satisfied with merely ascertaining that the parent or parents were in a diseased condition. But such information is clearly insufficient. In each case, where family taint is possible as a legitimate ætiological factor, it is necessary to enquire also into the time at which the parent or parents were affected. If a child be born of parents who

(6) Vandyke Carter: On Leprosy and Elephantiasis, page 180.

(7) T. R. Lewis and D. D. Cunningham: Leprosy in India, Calcutta, 1877.

acquire a disease some time after the birth of such child, and the offspring eventually becomes affected also, we cannot ascribe this to inheritance of a specific diseased condition. Such condition can be transmitted to the offspring, only if it were possessed by the parent before the birth of the child. Leloir lays particular stress on this point.

Unfortunately, at the commencement of their investigations, the Commissioners omitted to enquire closely into these circumstances. Thus it frequently happened at that time that no question was asked as to whether or not the parent or parents were healthy at the time of the child's birth. This, as will be seen presently, must materially affect the statistical results, and allowance should be made for this oversight. However, in every case where no statement has been made regarding the condition of the parent at the time of the birth of the offspring, this has been taken as a case of genuine transmission. Nevertheless, even with this admission, the statistical results do not affect the conclusions arrived at by the Commission, as will be fully explained later on.

After due consideration of all the evidence obtained by means of an examination of over 2,000 cases, the Commissioners have come to the conclusion *that leprosy in India cannot be considered an hereditary disease, and they would even venture to say that the evidence which exists is hardly sufficient to establish an inherited specific predisposition to the disease by the offspring of leprous parents to any appreciable degree.* These deductions have been drawn from the following facts, which, as far as the nature of the matter permits, will be given in tabular form.

Before proceeding further a few general remarks should be made on the subject of hereditary predisposition and transmission, and

these will be illustrated by examples derived from the knowledge of tuberculosis. Leprosy and tuberculosis resemble one another in many respects, and the ætiology of the latter disease has been more calmly and carefully considered.

Tuberculosis is essentially a disease of extra-uterine life, and though hereditary in certain instances it is not so as a disease, but as a predisposition. This assertion is not modified in consequence of the two cases published by Landouzy and Martin, Birch-Hirschfeld and Schmorl respectively.[8] For in neither case were any tubercles found in the fœtal organs, though the presence of tubercle bacilli was proved by microscopical observation and animal experiment. The fœtus was still born in either case, and there is thus much room left for speculation as to the ultimate fate of the child, if it had lived. A distinction must clearly be drawn between a congenital or hereditary disease and an infection from the parent through the placental circulation of the child. Instances of congenital tuberculosis have been recorded from time to time, but still are so extremely rare and often also so uncertain as not to invalidate the above statement.

When the influence of heredity in a disease such as leprosy is under discussion, and it is known or proved that the disease itself is not the thing transmitted from parent to child, but only the predisposition to it, great care and caution must be exercised in finding out how far in each case the congenital predisposition has not only been inherited, but also inherited in a legitimate manner. That is, this question must be considered, how far the leprosy of the child is due to the leprosy of its ancestor and how far due to other morbid conditions of the parent, which may have transmitted a congenital predisposition to the child. It must not be forgotten

(8) Supplement to the British Medical Journal, June 6th and 27th, 1891.

that in leprosy as in tuberculosis a predisposition is necessary for the disease to appear at any time. This predisposition may be due to many agents and factors, and it may therefore be quite possible that the factors which produced the predisposition in the parent, quite apart from the resulting leprosy, may be the cause of the congenital predisposition transmitted to the child. This, however, would not be a legitimate inheritance. Now, where practical questions regarding heredity are under consideration, which may result in such measures as separation of husband and wife or interdiction of marriages between the affected, it is of the utmost importance to study the influence of the disease itself in causing a specific predisposition in the offspring.

The fact, acknowledged equally for tuberculosis and leprosy, that the diseases occasionally "skip a generation," speaks strongly in favour of a more general than specific form of inheritance, and hence also for the possibility that a non-tubercular or non-leprous condition of the parent may cause the predisposition in the child, necessary for the development of tuberculosis or leprosy, as the case may be.

"With tuberculosis it is certain that in the congenital and developmental conditions of the offspring there is nothing of a dyscrasia. When in a tubercular family one child after another dies of tubercular arachnitis, this proves the existence of a specific dyscrasia as little as when in another family one member after another becomes insane. From this it can evidently only be argued that the brain and its membranes hereditarily suffer from certain abnormalities. Even the fact that in the same family one child will be affected with tubercular arachnitis, another with tubercular arthritis, and a third with laryngeal phthisis, does in no way prove the

existence of a dyscrasia which in one case attacks the arachnoid, in another the bones, and in a third the larynx. These facts can only prove that the exciting causes select one or other part, and that several areas of the body are in the same predisposed condition."[9]

Mutatis mutandis the same applies to leprosy. And when it is remembered that a predisposition may just as easily be acquired, it is felt that great stress must be laid on the external surrounding conditions. It is therefore necessary to study the effect of a specific hereditary predisposition pure and simple, and carefully exclude the influence of such external relations. It is evidently not sufficient to argue from the fact that a near relation had leprosy, that therefore the congenital predisposition was specifically or legitimately inherited. The congenital predisposition can only be said to have been thus inherited, if the child was actually born of a leprous parent. If born of a parent who some years, and frequently many years, after the birth of the child becomes a leper, this child cannot possibly be supposed to have inherited a specific leprous predisposition in a legitimate manner. The fact that the parent eventually falls a victim to the disease cannot make the inherited predisposition any more specific or legitimate. This can only prove that certain conditions and abnormalities of the parent, which eventually led to the establishment of a predisposition capable of being acted upon by the exciting causes responsible for the development of leprosy, have been transmitted to the offspring. And even this can only be so, if the possibility of an acquired predisposition in either case can be excluded.

In an investigation of this nature, therefore, it is necessary first to enquire into the number of cases and families in which a specific

(9) For these and the preceding remarks also *cf.* Virchow, loc. cit., pages 718–720.

hereditary predisposition may be assumed actually to exist, that is, to enquire into the condition of the children born of leprous parents. Then it must be seen whether such specific inherited predisposition has a greater influence on the development of the disease than a non-specific congenital predisposition. On this must turn the issue of reformatory measures such as forcible separation of husband and wife and interdiction of marriages. If facts do not establish a greater influence for the former, then the solution of the question cannot be sought in such rigorous rules, but must direct itself to the amelioration of the general external conditions.

These considerations have guided all the subsequent arguments in a complex and difficult question.

I.—A case was never seen of a child leprous at the time of birth, or so shortly afterwards, that it might fairly be considered a congenital case. This point has so often been insisted on by other authors and is so well known a fact that it is unnecessary to comment any further on it. Now it is obviously fatal to the conception of an hereditary transmission of a condition acquired by the parent, if it be established that such condition never appears congenitally. And this fact must be kept in view more particularly in the investigation of a disease of bacillary origin, for it is easy to conceive that the ovum or embryo could be invaded by the bacillus.

Though this argument tells forcibly against the hereditary transmission of the actual morbid condition or an infection through the placental circulation, a belief in a transmitted specific predisposition might still be maintained. It might be assumed that some constitutional defect causally related to the leprosy of the parent is set up in the embryo, which subsequently, under favourable conditions, gives rise to the disease. Other facts, however, while still further

disproving hereditary transmission, cast doubt also upon the possibility of an inherited specific predisposition.

II.—Since leprosy cannot in any case be regarded as a congenital disease, an enquiry must be made as to how often it is possible to trace a family taint in the direct line, from parent to offspring, and to this question the discussion in this chapter will be wholly confined.

From collateral relationship little, if anything, can be argued, as here so inextricable a maze of transmissions exists that it is impossible to disentangle one factor from another. If the facts available with regard to family taint in the direct line be not sufficient to establish inheritance, then *à fortiori*, arguments from collateral relationships must be altogether without force. Dr. Vandyke Carter,[10] who collected information from all parts of the Bombay Presidency, gives a most careful and exhaustive table showing the number and proportion of blood relations, and found that about 20 per cent. of lepers acknowledged a family taint. In 14·5 per cent. of these, however, the taint was in the collateral line. As cousins are included in these 14·5 per cent., it may be submitted that with so much admixture of alien blood little can be deduced from the figures. Arguments and deductions from distant relationships, as *e.g.* from grandparent to grand-child are of still less force, and of no real scientific value for the subject under discussion. In fact they argue rather against a specific inherited predisposition. It may be well here to repeat the words of Hjort, quoted by Dr. Liveing:[11] "From time to time the relationship of many hundred lepers to each other has been carefully investigated, and the result has shown that many of

(10) Vandyke Carter: loc. cit., page 184.
(11) R. Liveing: loc. cit., page 85.

them are more or less akin, and hence the conclusion has been drawn that the disease was hereditary in all those lepers who were related." It will be plain to all that this conclusion does not rest on a sound basis, and that a malady can only be inherited when the seeds thereof pass in a direct line from parents to children, while those persons who are only collaterally related cannot inherit the disease one from another. Lastly, when the term "atavism" is used in support of the theory of heredity, and marked "as a form of inheritance worthy of special notice on account of its frequent occurrence," a protest must be raised against the unwarrantable introduction into human pathology of a term which, whatever its place may be in the province of biological speculations, must remain meaningless to the pathologist enquiring into the ætiology of so markedly a general disease as leprosy. In the study of the inheritance of embryological deformities atavism may be of great importance, but if the term is employed to denote the sudden appearance of a constitutional disease after having "skipped one or several generations," it is inapplicable. Atavism can have no place in the ætiology of leprosy.

Collateral and distant relationships may consequently be disregarded, and the present enquiry is confined to the number of instances in which a family taint could be ascertained to exist in direct lines of descent. This will best be given in a tabular form. The first column of the following table specifies the name of the asylum or locality where the lepers were examined. The figures are arranged in two columns, (*a*) number of instances of family taint, regardless of the fact whether the parent or parents were leprous at the time the child was born, (*b*) number of instances of lepers actually born of leprous parent or parents, after the exclusion of

all those cases, where the parent or parents became lepers some time after the birth of the child.

TABLE I.

Family Taint in the Direct Line.

NAME OF ASYLUM OR LOCALITY.	No. of cases seen.	No. of cases in which a family taint was confessed.	No. of cases in which parent or parents were lepers at time of birth of offspring.	REMARKS.
Agra	54	3	1	This case is very doubtful.
Aligarh	10	1	1	No statement made as to condition of parent at time of birth.
Almora	108	17	4	
Bangalore . . .	55	4	2	In 1 no statement.
Belgaum . .	23	1	1	
Benares	32	1	1	
Bombay	259	34	8	
Burdwan . . .	21	...	...	
Calcutta	89	5	3	In 2 no statement.
Calicut	26	...	...	
Cawnpore . . .	10	..	...	
Conjeeveram . . .	18	5	3	In 1 no statement.
Darjeeling . . .	3	...	...	
Dehra Dun . . .	88	15	6	
Delhi	11	...	...	
Dharmsala . . .	12	3	3	
Fyzabad . . .	17	1	1	
Gwalior	13	1	1	No statement.
Gya	42	6	6	In 6 no statement. 1 very doubtful.
Hyderabad . . .	21	...	...	
Jubbulpore . . .	15	3	2	In both no statement.
Jummoo	2	1	1	No statement.
Kapurthalla . . .	10	3	3	In all no statement.

Table I—*continued.*

Family Taint in the Direct Line.

NAME OF ASYLUM OR LOCALITY.	No. of cases seen.	No. of cases in which a family taint was confessed.	No. of cases in which parent or parents were lepers at time of birth of offspring.	REMARKS.
Lahore	2	...	...	
Lucknow	13	...	...	
Madras	184	28	22	In 18 no statement and 3 cases doubtful.
Madura	113	16	7	In 4 no statement.
Mandalay	177	5	3	In all no statement.
Meerut	9	...	...	
Moulmein	25	2	1	
Nagpur	174	7	6	In all no statement.
Naini and Allahabad	66	1	1	In 1 no statement.
Naini Tal	2	...	...	
Patiala	9	1	1	No statement.
Peshawar	2	...	...	
Poona	45	5	4	In one case father, grandfather, and brother lepers.
Prome	30	1	...	
Purulia	99	18	9	In 5 no statement.
Rangoon	62	5	3	In 3 no statement.
Rawalpindi	47	11	5	In 3 no statement.
Sialkot	40	4	2	
Subathu	46	8	1	In 1 no statement.
Tanjore	16	4	3	In 3 no statement.
Tarn Taran	146	28	9	In 1 no statement.
Thayetmyo	62	8	4	In 2 no statement.
Trichinopoly	16	3	1	No statement.
Umballa	32	5	1	
Yerrowda Prison	15	...	...	
TOTAL	2,371	264 = 11·1 per cent.	130 = 5·48 per cent.	No statement in 74 cases.

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...direct line could be traced in on...

...of in 5·48 per cent. Now, in 74 cas... must be raised a dise...either no statement was made as to the condi...idered con-parent or parents at the time of the child's birth or some ...f his ex-...as expressed as to diagnosis, and allowing for this, it wo... patient, perhaps be nearer the mark to say that a true family taint in the ...and-direct line could be established in about 4 per cent. of the cases ...hy seen.

Family taint in the direct line is, therefore, established in only a few cases, and, consequently, as far as these figures go, one is forced to look for other and more potent causes for the origin of the disease. If the results of Höegh and Bidenkap,[12] who traced the disease in the direct line in 25 per cent., and further those of Van Sommeren,[13] who amongst 31 cases found only two hereditary ones, prompted Virchow to the remark that heredity cannot explain the spread of the disease, how much less do the above figures justify the belief in the high importance of an hereditary influence?

Table I has been constructed from the cases actually seen and examined by the Commissioners. As, however, many patients confessed to having leprous children or parents, either deceased or absent, it is necessary to construct another table including all these cases. This is done in Table II which is arranged on the same plan as the previous one.

(12) Höegh and Bidenkap: Norsk Magazin for Lagevidenskaben, 1860, Vol. XIV.

(13) Van Sommeren: A brief Historical Sketch of the Madras Leper Hospital.

TABLE II.

Family Taint in the Direct Line.

Name of Asylum or Locality.	No. of cases seen.	No. of cases in which a family taint was confessed.	No. of cases in which parent or parents were lepers at time of birth of offspring.	Remarks.
Agra	55	4	1	This case is very doubtful.
Aligarh	10	1	1	In 1 no statement.
Almora	113	21	5	
Bangalore	56	5	2	In 1 no statement.
Belgaum	23	1	1	
Benares	32	1	1	
Bombay	260	35	8	
Burdwan	21	...	...	
Calcutta	89	5	3	In 2 no statement.
Calicut	26	...	...	
Cawnpore	10	...	...	
Conjeeveram	19	6	4	In 1 no statement.
Darjeeling	3	...	...	
Dehra Dun	89	16	6	
Delhi	11	...	...	
Dharmsala	12	3	3	
Fyzabad	17	1	1	
Gwalior	13	1	1	In 1 no statement.
Gya	43	7	7	In 1 doubtful diagnosis, 6 no statement.
Hyderabad	21	...	...	
Jubbulpore	16	4	3	In 3 no statement.
Jummoo	2	1	1	In 1 no statement.
Kapurthalla	10	3	3	In 3 no statement.
Lahore	2	...	...	
Lucknow	13	...	...	

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Table II—*continued.*

Family Taint in the Direct Line

Name of Asylum or Locality.	No. of cases seen.	No of cases in which a family taint was confessed.	No. of cases in which parent or parents were lepers at time of birth of offspring.	Remarks.
Madras	194	35	24	In 3 doubtful diagnosis. 18 no statement.
Madura	115	23	10	In 5 no statement.
Mandalay	179	5	3	In 3 no statement.
Meerut	9	...	...	
Moulmein	25	2	1	
Nagpur	176	9	7	In 7 no statement.
Naini and Allahabad	66	1	1	In 1 no statement.
Naini Tal	2	...	...	
Patiala	9	1	1	In 1 no statement.
Peshawar	2	...	...	
Poona	45	5	4	
Prome	31	2	...	
Purulia	105	25	10	In 5 doubtful diagnosis. In 4 no statement.
Rangoon	62	5	3	In 3 no statement.
Rawalpindi	51	15	7	In 3 no statement.
Sialkot	40	4	2	
Subathu	48	10	1	In 1 no statement.
Tanjore	16	4	3	In 3 no statement.
Tarn Taran	154	36	12	In 2 no statement.
Thayetmyo	63	9	4	In 2 no statement.
Trichinopoly	17	4	1	In 1 no statement.
Umballa	32	5	1	
Yerrowda Prison	16	1	...	
Total	2,423	316 =13·0 per cent.	146 =6·0 per cent.	In 83 insufficient statement.

ording to this enlarged table
direct line existed in 6 per ce.
the corresponding ratio of the former ta
hand, the number of cases noted as doubtful
the detailed statement is greater, and as the additional
could not be seen, the suspicion of a wrong diagnosis also is
excluded. Making allowance for all these factors it may be said that this second table fairly represents a percentage of 4·5 of a possible heredity, so that the conclusions derived from a comparison of these two tables are more or less in harmony, and as far as such figures go, establish only to a slight degree the existence of a family taint, and 4 per cent. might, therefore, fairly be accepted as representing the ratio of a specific family taint in the direct line.

These figures tend to confirm Hansen's disbelief in an inherited specific predisposition. In constitutional diseases such as leprosy the greatest caution must be exercised in accepting any theory of inheritance. As the importance of inheritance as an ætiological factor vanishes, so that of external conditions must rise into prominence. And parent and child being subjected usually to exactly similar surrounding conditions and circumstances, it is well within the bounds of probability that the disease has started *de novo* in both. One might otherwise with equal reason classify rickets amongst the hereditary diseases. Now that the pathology of fœtal rickets has been elucidated, it is well known that rickets is a disease of bad nutrition and is never congenital or hereditary; yet it would not be difficult in London to find a family taint in 4 per cent. of rickety patients. Unless the influence of external causes can be excluded, and an inheritance proved in a majority of cases and on sound evidence, the hereditary transmission of, or specific predisposition to,

a disease cannot be said to be established. A protest must be raised when such cases as quoted by M. de Valencé are considered convincing proofs of an hereditary transmission.[14] For one of his examples of inherited leprosy rests on the "confession of a patient, that he vaguely remembered having heard that his maternal grandmother had had leprosy."[15] His parents apparently were healthy and he himself was certainly untainted when his three children were born. Two of the latter became leprous and died before the patient himself was affected. The details in the history of inheritance of the other case are too insufficient. Of four sisters and two brothers of apparently healthy descent, the two latter became diseased. One of them died without issue, while the other had a leprous son. "The sisters had children affected with leprosy, and it is possible at the present day to trace easily the descendants of these four sisters and enumerate the members of the family who died from the disease."[16] Thus as the case stands, a transmission by inheritance can at best only be established from one of the brothers to his son. Yet this evidence, in a leading article of the *Lancet*,[17] has been described as convincing on the question of hereditary transmission. It is on this account only, that notice had to be taken of M. de Valencé's paper.

The great importance of external conditions in the ætiology of leprosy must be admitted, and the figures therefore tend to reduce the importance of inheritance so greatly, that practically it may be altogether disregarded.

Again, if heredity have an appreciable influence on the spread or

(14) *Lancet*, 1890, Vol. I, page 1077.
(15) *Lancet*, 1890, Vol. I, page 1065.
(16) *Lancet*, 1890, Vol. I, page 1077.
(17) *Lancet*, 1890, Vol. I, page 1065.

origin of the disease, it should be possible to trace leprosy through several generations in a sufficiently large number of cases. In each instance, therefore, when a family taint in the direct line was acknowledged, careful enquiries were made as to the condition of the grand-parents. Only eighteen cases could be obtained where the disease could be traced from the grandfather or grandmother downwards. Now in five cases the statements were insufficient or the diagnosis of leprosy doubtful (Nos. 3, 4, 5, 8, 9), and in further six cases the children were in each instance born of healthy parents, so that the conditions are such as to exclude an inherited specific predisposition (Nos. 1, 11, 12, 13, 14, 15). These cases will now be given in an analysed form.

TABLE IIa.

Analysis of Cases where a Family Taint in the Direct Line could be traced through more than One Generation.

(*a*) *Bombay:* (1) Mother = Leper (HER FAMILY HISTORY WAS QUITE CLEAR).

Two sons = both healthy. Daughter = Leper (BECAME A LEPER BEFORE HER MOTHER).

Daughter = Leper. Son = Leper.

Both these children were born when their mother was healthy, and they became affected about the same time as their grandmother, three to five years after their mother. The whole family was seen and examined.

(*b*) *Dehra Dun:* (2) Maternal grandfather = Leper.
Mother = Leper.
Daughter = Leper (This patient was seen and examined).
Two children who died as healthy infants.

In each instance the parent was a leper before the birth of the child.

(*c*) *Gya:* (3) Father = Leper (HIS FAMILY HISTORY WAS QUITE CLEAR).
Only son = Leper
Only child = Leper } (These two were seen and examined).

In each instance the parent was a leper before the birth of the child.

Table IIa—*continued.*

Analysis of Cases where a Family Taint in the Direct Line could be traced through more than One Generation.

(*d*) *Kapurthalla:* (4) Grandmother = Leper (NO STATEMENT MADE, WHETHER PATERNAL OR MATERNAL).
Father = Leper.
Son = Leper (This patient was seen and examined).
No statements were made whether the parent in each instance was a leper before or after the birth of the offspring, nor as to the number of children each parent had.

(*e*) *Madras:* (5) Grandfather = Leper (NO STATEMENT MADE, WHETHER PATERNAL OR MATERNAL).
Mother = Leper.
Son = Leper (This patient was seen and questioned).
Three children (one of them said to be a leper, but diagnosis extremely doubtful).

(6) Father and Mother = Lepers.
Two daughters = Lepers (These two and the child were seen and examined).
One of these has a child = Leper (BUT DIAGNOSIS IS EXTREMELY DOUBTFUL).

(7) Maternal grandfather = Leper.
Mother = Leper (This patient and her leprous child were seen).
Two children and one of them = Leper (the other is healthy, married and has several healthy grown-up children).

(8) Maternal grandfather = Leper.
Mother = Leper.
Daughter = Leper (This patient was seen and examined).

(*f*) *Madura:* (9) Father = Leper (HIS FAMILY HISTORY QUITE CLEAR).
Only child = Leper.
Two children and one of them = Leper (BUT DIAGNOSIS WAS EXTREMELY DOUBTFUL).

(*g*) *Poona:* (10) Grandfather = Leper.
Father and Mother = Lepers.
Two sons = Lepers (These two were seen and questioned).

(*h*) *Purulia:* (11) Grandparents on one side = Lepers.
Parents = Lepers.
Two out of five children = Lepers (These two were seen: all adults)

(12) Mother = Leper (HER OWN FAMILY HISTORY CLEAR).
Son = Leper. Daughter = Leper (BOTH SON AND DAUGHTER BORN LONG BEFORE MOTHER BECAME A LEPER).
Two sons = Lepers (BOTH BORN BEFORE MANIFESTATION OF DISEASE IN THEIR MOTHER).
The whole family was seen and questioned.

Table IIa—*continued.*

Analysis of Cases where a Family Taint in the Direct Line could be traced through more than One Generation.

(13) Father=Leper (3) (HIS FAMILY HISTORY WAS CLEAR AND HE BECAME A LEPER AFTER HIS SONS AND *NEPHEW WERE AFFECTED*).

Son I=Leper. (4) Son II=Leper. (5) Son III=Leper. (6) Son IV=Leper. (2) Four daughters=all healthy.

One child=healthy. Fourteen children and one of them=Leper.

Six children between the two and *one* of these=Leper (1) (WAS AFFECTED BEFORE ANY OTHER MEMBER OF THE FAMILY).

One child=Leper (BORN BEFORE DISEASE APPEARED IN FATHER).

One daughter=Leper. Two sons=healthy.

One child=healthy. Seven children=healthy.

Remarks.—In each case the offspring was born long before the disease appeared in the parent. Six members of this family were examined individually and separately, and an absolute uniformity of statements obtained. The figures in brackets indicate the order in which the disease appeared in the various members of this family. The father acquired leprosy one year or so before his death and was an old man at the time of his death.

(14) Mother=Leper (HER OWN FAMILY HISTORY WAS CLEAR).

Two sons=healthy. One daughter=Leper (ACQUIRED THE DISEASE LONG BEFORE HER MOTHER).

Two sons=Lepers (BORN AFTER THE MANIFESTATION OF THE DISEASE IN THEIR MOTHER).

The whole family was seen and questioned.

(*i*) *Tarn Taran*: (15) Father and Mother=Lepers (THEIR OWN FAMILY HISTORY WAS CLEAR AND THEY ACQUIRED THE DISEASE AFTER ALL THEIR CHILDREN WERE BORN).

Three sons=Lepers. One daughter=Leper (SHE MARRIED WHILE HEALTHY AND SEPARATED FROM HUSBAND AS SOON AS THE DISEASE APPEARED IN HER).

One child=Leper (DEAD AT THE AGE OF 12 YEARS AND WAS BORN SEVERAL YEARS BEFORE THE MOTHER BECAME AFFECTED).

Four members of this family were seen and questioned.

(16) Father=Leper (HIS OWN FAMILY HISTORY IS CLEAR).

Son I=Leper. Son II=Leper (BOTH BORN LONG BEFORE APPEARANCE OF LEPROSY IN THE FATHER).

One son=Leper (ALSO BORN LONG BEFORE APPEARANCE OF DISEASE IN HIS FATHER).

The whole family was seen and examined.

Table IIa—*concluded.*

Analysis of Cases where a Family Taint in the Direct Line could be traced through more than One Generation.

(17) Father=Leper (HIS OWN FAMILY HISTORY WAS CLEAR).

Son I=healthy. Son II=Leper (BOTH BORN AFTER THEIR FATHER BECAME A LEPER).

One daughter=Leper.

(*k*) *Thayetmyo*: (18) Mother=Leper (HER OWN FAMILY HISTORY WAS CLEAR).

Seven children and *one* of them=Leper.

Two children and *one* of them=Leper (DIAGNOSIS EXTREMELY DOUBTFUL).

Here no statements were made as to date of appearance of leprosy in the parents.

Even allowing that in a certain number of cases the patients knew little of their grandparents, if heredity exercised any appreciable influence on the spread of leprosy, it should have been possible to obtain a far greater number of cases where the disease could be traced through two generations at least. As, however, all the doubtful cases have been included, the figures may be assumed to represent the truth as nearly as possible. Accepting even all the eighteen cases the ratio would still be less than 1 to 100. Now in a disease like leprosy it may be urged that in most leprous families the grandchildren would have knowledge of the condition of their immediate ancestors. Again this analysis shows the necessity of a careful enquiry in each case into the time at which the parent or parents became leprous, for it would evidently be absurd to consider Case 13 an instance of hereditary transmission, as here the grandchild actually acquired the disease before the common ancestor. In Cases 1 and 14 the child also became affected before the mother. In such instances it obviously cannot be a specific predisposition based on the existence of leprosy in the parent which has been transmitted to

the offspring. If a predisposition has been transmitted to the child, it must have been the physiological or anatomical conditions which have rendered the ancestor a favourable subject for the *de novo* acquisition of the disease, but it is certainly not a legitimate inheritance of a leprous predisposition. For whatever the child inherited it would have inherited, whether the parent subsequently became a leper or not. And excluding contagion it may be quite possible that in all the members of the family the disease started *de novo*, the necessary predisposition having been acquired. To allow such cases as proofs of an inherited specific predisposition would obviously be a "post hoc ergo propter hoc" argument.

III.—From the fact that leprosy occurs often in several members of a family, authors have argued the existence of a specific hereditary predisposition. That such an argument cannot hold good may be seen from the fact that instances occur where two or three brothers or sisters simultaneously suffer, although they are not aware that they had any leprous ancestors. Leloir lays special stress on this point, and doubtless if it can be shown with sufficient frequency that several children become affected, while the parents remain healthy, this fact, coupled with the small percentage of direct family taint, found in the present enquiry, would still further diminish the importance of heredity as a cause of the continuance of the disease. It would tend to show that individuals habitually exposed to the same conditions are liable to be affected in the same manner. And as often years elapse between the appearance of the disease in two children of the same family, it reduces the importance of heredity still further. The fact of the parent being a leper before the birth of the child does not alter this statement, for it has been found in many cases that the son of a leprous parent contracted the disease long

before his younger brother, destined subsequently to become a leper, was born.

Enquiring into this matter, it is found that in 101 instances or in about 4·5 per cent. of the cases examined, two or more children became lepers, while their parents were perfectly healthy, and in most of these cases no other relative was affected. The correspondence of this ratio with the above is remarkable. In this case heredity can of course have played no part, as it is impossible to look to atavism for support, for the reasons assigned above. The conclusion is strongly suggested that though the disease in a small proportion of cases appears to run in families, this cannot be ascribed to a specific inherited predisposition.

A table is given showing the distribution of these cases amongst the various asylums and places visited.

TABLE III.

Instances where two or more Children were Lepers but their Parents healthy.

NAME OF ASYLUM OR LOCALITY.	No. of instances.
Agra	1
Aligarh	1
Almora	13
Bombay	7
Calcutta	2
Conjeeveram	3
Darjeeling	1
Dharmsala	1

Table IV—*continued.*

Cases where Father and Mother were Lepers at the Time of Marriage.

NAME OF ASYLUM OR LOCALITY.	No. of couples.	No. of persons these couples represent.	No. of children.	No. of children who became lepers.
Meerut	6	12	...	...
Nagpur	1	2	...	...
Patiala	1	2	1	...
Rangoon	1	2	...	...
Rawalpindi	14	26	10	1 (??)
Sialkot	14	26	3	2
Subathu	2	4	5	...
Tarn Taran	22	42	18	...
Umballa	1	2	...	...
TOTAL	98	188	65	3 or 5=4·6 per cent. or 7·6 per cent.

V.—The only decisive way of disentangling the influence of heredity and external conditions would be to imitate the experiment of the emigrant Norwegian lepers, and to remove the offspring of lepers not only from their parents but also into an area where leprosy is not endemic. Placed under such conditions, with good food and hygienic surroundings, heredity would have full and absolute play. There are two asylums in India where children born of leprous parents are kept apart from them and tended with great care; this is done at Almora and Purulia. The latter unfortunately has not been sufficiently long in existence, hardly more than two years. The Almora Orphanage, however, is much older and thus capable of affording very valuable information. The children born of either one

or two leper parents are removed from their surroundings and carefully attended to in the Orphanage. Endemic influences cannot of course be excluded. Drs. Lewis and Cunningham[18] say in their report: "There are at present (1877) in the Orphanage of Almora 12 children of lepers now or formerly in the Asylum. The total number of such children who have been admitted into the Orphanage is 14, but of these 1 died, and another, a girl of 22, has now left the Orphanage, is married, and has children healthy to all appearance. Of the 12 remaining 7 were born in the Asylum of two leprous parents; 5, the offspring of one leprous and one healthy parent, were born in the villages to which their parents belonged. Their ages ranged from 19 to 5 years; their health and general condition is excellent, and as yet they show no signs of leprosy."

The Commissioners were able to ascertain the corresponding facts with regard to 23 inmates of the Orphanage. Of these three were born before the manifestation of the disease in the parent, their ages being 31, 28, and 19, respectively, and five were under 10 years, their ages ranging from 7 years to 6 months (7, 7, 4, 3, and ½ years, respectively). All these eight individuals were up to date in good health. This leaves 15 cases, and of these, as will be seen from Table V, only 1 has contracted the disease, all the others having remained perfectly well, or having died free from leprosy in adult age. The number of children is certainly not a large one, and deductions should naturally be made with care, but the figures, especially if compared with the other ratios obtained, tend still further to diminish the importance of heredity. For omitting the eight cases, for the reasons mentioned, an assumed hereditary transmission was traceable in 6·6 per cent.

(18) Loc. cit., page 67.

TABLE V.

Almora Asylum Orphanage.

No.	Sex.	Age.	Condition of parents at birth.	Period spent with parents.	Period spent in Asylum previous to admission into Orphanage.	Period spent in Orphanage.	Present condition.
1	Female.	28 years	Father and mother lepers	2 years	2 years	16 years	Healthy at 20 years of age.
2	„ .	28 „	Ditto .	2 „	2 „	15 „	Healthy up to date.
3	„ .	27 „	Ditto .	1 year .	2 „	24 „	Ditto.
4	„ .	25 „	Father healthy; mother leprous . .	8 years	6 „	11 „	Healthy; died of diarrhœa.
5	„ .	24 „	Father and mother lepers	2 months	3 „	20 „	Healthy up to date.
6	„ .	22 „	Father healthy; mother leprous . .	1 year .	½ year	20 „	Ditto.
7	„ .	21 „	Ditto .	7 months	2 years	19 „	Healthy; died of phthisis.
8	*Male* .	*21* „	*Father and mother lepers*	*2 years*	*4* „	*11* „	*Leper for last 4 years.*
9	Female.	20 „	Ditto .	2 „	2 „	15 „	Healthy; died of epilepsy.
10	„ .	18 „	Ditto .	1 year .	1 year	16 „	Healthy up to date.
11	„ .	17 „	Father healthy; mother leprous . .	5 years	3½ years	8½ „	Ditto.
12	Male .	15 „	Father and mother lepers	6 „	...	9 „	Ditto.
13	Female.	14 „	Ditto .	1 year .	2 years	11 „	Ditto.
14	„ .	13 „	Father leprous; mother healthy	10 years	2 „	1 year	Ditto.
15	Male .	12 „	Father and mother lepers	2 „	...	10 years	Ditto.

VI.—In the fourth section the condition of children born of two leprous parents was enquired into. Here the condition of all the children born after the manifestation of the disease in one or other parent will be considered. It must be remarked that in many cases the children could not be personally examined by the Commissioners, and in these cases the parent's statements had to be accepted. Doubtless this is a source of error and due allowance must be made. It was found that amongst 500 children of all ages born of either two leprous parents, or from one leper parent and one healthy parent, 21, or 4·2 per cent., became lepers. In eight of these the existence of the disease was extremely doubtful, while in some others no statements were made as to the condition of the parent or parents at the time of the child's birth. Even if 19 be accepted as the actual number of the offspring attacked, the ratio yielded very nearly approaches the truth. But as some children were still young it may be objected that they had not yet reached the age at which leprosy generally shows itself, and it is desirable to re-arrange the offspring in two classes—(*a*) all those of ten years and over, and (*b*) all those of sixteen years and over. Taking ten as the minimum age, it was found that there were 150 individuals born after the appearance of the disease in the parent or parents, and of these 10 had become lepers, or 6·6 per cent. Again, taking sixteen as the minimum age, there were 82 individuals born of leprous parents, of whom six subsequently contracted the disease, or 7·31 per cent. Making all allowances for errors in diagnosis and inaccurate statements, it will be apparent that the figures correspond closely with those of former tables.

TABLE VI.

Condition of Children born after Manifestation of Disease in Parent or Parents.

NAME OF ASYLUM OR LOCALITY.	No. of children.	No. of children affected.	REMARKS.
Agra	15	...	
Aligarh	5	...	All young.
Almora	29	1	
Bangalore	20	1	Very doubtful diagnosis.
Belgaum	2	...	10½ years old.
Benares	5	...	8½ years old.
Bombay	10	2	
Burdwan	5	...	
Calcutta	12	...	
Calicut	20	...	
Cawnpore	5	...	All under 10 years.
Conjeeveram	12	2	10½ years old.
Darjeeling	2	...	Under 6 years.
Dehra Dun	20	1	Very doubtful diagnosis.
Delhi	6	...	All under 6 years.
Dharmsala	1	...	
Fyzabad	4	...	
Gwalior	3	...	Under 12 years.
Gya	5	1	9½ years old.
Hyderabad	16	...	From 11½ years old.
Jubbulpore	9	...	
Jummoo	1	...	6 years old.
Kapurthalla	3	1	Aged 25; very doubtful diagnosis.
Lucknow	1	...	

Table VI—*continued.*

Condition of Children born after Manifestation of Disease in Parent or Parents.

NAME OF ASYLUM OR LOCALITY.	No. of children.	No. of children affected.	REMARKS.
Madras	43	3	2 very doubtful.
Madura	56	2	
Mandalay	35	...	All adults.
Meerut	3	...	
Moulmein	9	1	Very doubtful.
Nagpur	30	...	
Naini and Allahabad	17	...	
Naini Tal	1	...	
Patiala	1	...	
Prome	3	...	
Purulia	4	1	Very doubtful.
Rangoon	9	...	
Rawalpindi	12	2	No statement made.
Sialkot	4	2	All adults; no statement made.
Subathu	11	...	
Tanjore	1	...	Under 5 years old.
Tarn Taran	34	...	
Thayetmyo	5	1	Very doubtful.
Trichinopoly	7	...	All under 10 years old.
Umballa	1	...	18 years old.
Yerrowda Prison	3	...	Under 7 years old.
TOTAL	500	21 = 4·2 per cent.	

TABLE VII.

Condition of Children born after the Manifestation of the Disease in the Parent or Parents, taking 10 years as the Minimum Age.

NAME OF ASYLUM.	No. of children.	No. of children affected.	REMARKS.
Agra	3	...	
Aligarh	1	...	
Almora	23	1	
Bangalore	4	...	
Belgaum	1	...	
Benares	3	...	
Bombay	2	1	
Calcutta	1	...	
Calicut	10	...	
Conjeeveram	1	1	
Dehra Dun	6	...	
Hyderabad	2	...	
Jubbulpore	2	...	Very doubtful diagnosis.
Kapurthalla	3	1	12 years old.
Lucknow	1	...	
Madras	4	...	
Madura	17	1	
Mandalay	10	...	
Meerut	3	...	
Nagpur	12	...	
Naini and Allahabad	7	...	
Patiala	1	...	
Prome	1	...	
Purulia	3	1	Very doubtful diagnosis.

Table VII—*continued.*

Condition of Children born after the Manifestation of the Disease in the Parent or Parents, taking 10 years as the Minimum Age.

NAME OF ASYLUM.	No. of children.	No. of children affected.	REMARKS.
Rangoon	1	...	
Rawalpindi	4	2	
Sialkot	4	2	
Subathu	4	...	
Tarn Taran	13	...	
Thayetmyo	2	...	
Umballa	1	...	
TOTAL	150	10 = 6·6 per cent	

TABLE VIII.

Condition of Children born after the Manifestation of the Disease in the Parent or Parents, taking 16 years as the Minimum Age.

NAME OF ASYLUM.	No. of children.	No. of children affected.	REMARKS.
Agra	2	...	Ages 19 and 16.
Almora	13	1	
Benares	1	...	

Table VIII—*continued.*

Condition of Children born after the Manifestation of the disease in the Parent or Parents, taking 16 years as the Minimum Age.

NAME OF ASYLUM.	No. of children.	No. of children affected.	REMARKS.
Bombay	2	1	
Calicut	2	...	
Dehra Dun	5	...	
Hyderabad	1	...	
Kapurthalla	3	1	Very doubtful diagnosis.
Madras	3	...	
Madura	6	...	
Mandalay	8	...	
Meerut	3	...	
Nagpur	7	...	
Naini and Allahabad	7	...	
Prome	1	...	
Purulia	3	1	Very doubtful diagnosis.
Rawalpindi	1	...	
Sialkot	4	2	
Subathu	2	...	
Tarn Taran	6	...	
Thayetmyo	2	...	19 and 16 years old.
Umballa	1	...	18 years old.
TOTAL	82	6 = 7·31 per cent.	

VII.—Lastly, the influence of an inherited specific predisposition may be studied by the selection of cases in which a family taint in the direct line can be traced, and by an enquiry into the condition of the brothers and sisters. Now, as was shown above, in many instances where there is evidence of a family taint, the patients were born at a time when their fathers and mothers were absolutely healthy though they afterwards became lepers. These cases might be termed instances of "false heredity." Again others were actually born of a leper parent or parents. If new tables be constructed with a view to showing how many of the brothers and sisters of lepers with a "false" hereditary history have become lepers in comparison with the number of brothers and sisters of lepers with a "true" hereditary history who have become lepers, additional light will be thrown upon the influence of heredity. For, in the former case, the predisposition cannot have been legitimately inherited, and, if hereditary, must be related to general non-specific conditions of the parent. In the case of patients actually born of leprous parents great care has been taken to ascertain that their brothers and sisters were also born after the manifestation of the disease in the parent or parents. In some cases no statements were made, and it is quite possible that some of the brothers and sisters were born at a time when their immediate ancestors were healthy. In other cases, with regard to the patient himself, whose brothers and sisters form the subject of the present enquiry, no statement was made as to whether he or she had a true or false history of heredity. These cases have been entered as though they were instances of true heredity, and thus errors in statistics are more or less counterbalanced, and the statistics placed in a more favourable light from the heredity point of view.

It has been found that 62 patients with a true hereditary history had 156 brothers and sisters born also, as far as could be ascertained, after the manifestation of the disease in their fathers and mothers; and of these 21 became lepers, or 13·4 per cent.

On the other hand, 61 patients, with a history of false heredity, had 150 brothers and sisters, and of these 31, or 20·6 per cent., were lepers. This is certainly a most surprising result, and, if coupled with the number of instances of several children becoming lepers while their parents remained healthy, removes the importance attributed to a specific hereditary predisposition. The figures are certainly somewhat small, but yet allow of decisive conclusions being drawn from them. Now, even if the cases with insufficient statements be excluded from Table IX, and added to Table X, the ratio would still militate against the influence of an actual heredity. For, in spite of making such allowances, the ratio could not be reduced below 15 per cent. or 20 per cent. This appears to be a strong argument against inheritance, for if this agency had much influence the ratios should not only be reversed, but the percentage of lepers in Table IX ought to be much greater than that of Table X. The only inference to be drawn from these tables, and from the fact of children being often affected though their parents remain healthy, is that leprosy appears to have a tendency to attack several members of a family, but that a legitimate specific hereditary transmission cannot be regarded as the cause. In another place it will be shown whether contagion can explain this fact. It should never be forgotten that a disease due to extraneous agents and conditions, such as leprosy seems to be, may at any time affect people living in similar surroundings and possessing similar constitutions. And it is precisely amongst the members of a family that we get such conditions and relations. However this will be dealt with more fully in the chapter on Contagion.

TABLE IX.

Condition of the Brothers and Sisters of Leprous Patients with a "True" History of Heredity.

NAME OF ASYLUM OR LOCALITY.*	No. of patients actually born of leper parents.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Agra	1	4	...	Doubtful.
Aligarh	1	2	...	No statement.
Almora	1	1	...	
Bangalore	3	7	...	
Belgaum	1	2	...	
Benares	1	2	...	
Calcutta	2	4	...	
Dehra Dun	4	9	1	
Dharmsala	2	5	1	
Gwalior	1	5	...	No statement.
Gya	5	12	...	
Jubbulpore	1	1	1	No statement.
Jummoo	1	3	...	Ditto.
Madras	10	28	8	In some no statement.
Madura	2	6	1	
Nagpur	4	7	1	No statement.
Naini and Allahabad	1	2	...	
Patiala	1	1	...	No statement.
Poona	1	1	1	
Purulia	3	7	2	
Rangoon	1	1	...	
Rawalpindi	4	11	1	No statement.

* Bombay has been omitted on account of insufficient details, as in no case the number of brothers and sisters was stated.

Table IX—*continued.*

Condition of the Brothers and Sisters of Leprous Patients with a "True" History of Heredity.

NAME OF ASYLUM OR LOCALITY.*	No. of patients actually born of leper parents.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Sialkot	1	4	...	
Tanjore	1	4	...	
Tarn Taran	4	10	3	
Trichinopoly	1	6	...	No statement.
Thayetmyo	3	8	1	
Umballa	1	3	...	
TOTAL	62	156	21= 13·4 per cent.	

TABLE X.

Condition of Brothers and Sisters of Leprous Patients with a "False" History of Heredity.

NAME OF ASYLUM, ETC.	No. of patients with such history.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Agra	2	3	1	Bombay has been omitted for the reason given in foot-note, page 259.
Almora	10	29	5	
Bangalore	2	4	...	

* Bombay has been omitted on account of insufficient details, as in no case the number of brothers and sisters was stated.

Table X—*continued.*

Condition of the Brothers and Sisters of Leprous Patients with a "False" History of Heredity.

NAME OF ASYLUM, ETC.	No. of patients with such history.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Conjeeveram	1	4	1	
Dehra Dun	4	16	...	
Fyzabad	1	2	...	
Jubbulpore	2	8	...	
Madras	5	13	3	
Madura	3	8	3	
Mandalay	2	5	...	
Purulia	9	22	8	
Rangoon	1	1	...	
Rawalpindi	3	4	1	
Sialkot	1	1	...	
Subathu	2	2	2	
Tarn Taran	7	13	6	
Thayetmyo	1	1	...	
Trichinopoly	2	8	1	
Umballa	3	6	...	
TOTAL	61	150	31= 20·6 per cent.	

A glance at Tables IX and X might at first sight lead to the belief that the children born of lepers or of parents who became lepers at any time after the birth of these children suffer more fre-

quently from leprosy than do the children born of people remaining free from the disease. To enquire into the truth of this statement a Table, X*a*, has been added to show the condition of the brothers and sisters of leprous patients born of parents healthy before and after the birth of such patients. It has been found that 850 patients had 2,853 brothers and sisters, of whom 97, or 3·4 per cent., subsequently contracted leprosy. On the other hand, from Tables IX and X, it will be seen that 123 patients with a history of "true" or "false" heredity had 306 brothers and sisters, of whom 52, or 17·0 per cent., subsequently became affected.

Does this prove a specific hereditary influence or predisposition? Now it is evident that from Table IX no conclusion can be drawn as to the fate of the offspring of leprous parents as a whole, for here only those families have been selected where one parent and one child at least are lepers, and where a specific hereditary influence may actually be assumed to exist, the child in each instance being born of a leprous parent. All those cases, however, have been omitted where a leprous parent had absolutely healthy children. Table IX does not therefore assert anything about *all* the children born of leprous parents. How many of these become diseased has been considered in previous paragraphs. Selecting, however, those families where, through the fact that one child is affected like its parent, a specific hereditary predisposition is granted to exist—and of such only a small number was found—Table IX shows that contrary to expectation of 218 children with such an inherited predisposition only 83, or 38 per cent., became lepers. That this ratio should be higher than in Table X*a* simply amounts to stating that in these families the assumed hereditary taint actually exists, but it says nothing as to the number of children born of leprous

parents who subsequently become lepers. Table IX, therefore, clearly cannot be contrasted with Table X*a* with a view of finding out the greater predisposition of the offspring of leprous parents as a whole.

A similar objection must be raised when Table X is employed to prove the same assumption. For here only those families are chosen where one child at least became a leper, while those children which permanently remained healthy have been excluded. To make the conclusion as to the offspring in general a legitimate one, *all* the children born of such parents who, though healthy at the time of birth of these children, subsequently became lepers, should be considered. A comparison of Tables X and X*a* can evidently only prove this, that in those families where a parent becomes a leper after the birth of his children, and where also at least one of these children subsequently becomes a leper, the children as a whole are more liable to suffer from the disease, just as a comparison of Tables IX and X*a* can only show that in those families where at least one parent is a leper before the birth of his children, and where also at least one of these children subsequently becomes a leper, the children as a whole are somewhat more liable to suffer from the disease than the children in those families where the parents are healthy, and remain so, while at least one child is a leper.

But clearly all this does not prove anything in favour of a specific hereditary predisposition causally related to the leprosy in the parent. In fact, it argues strongly for an acquired predisposition both by the parents and the children, and this in the case of the latter may have been facilitated by some congenital abnormality not related to leprosy, but inherited from the former.

On the other hand, it seems astonishing from a consideration of Table X*a*, that selecting those families where the parents were healthy, but one child at least a leper, of 850+2,853 or 3,703 children 850+97 or 947 individuals, or 25 per cent., should have become lepers, though the parents, as far as could be ascertained, were free from any family taint in the direct line. The same percentages for Tables IX and X would be 37 and 43·6, respectively. It might now be well to compare these figures with results obtained from a consideration of such families where the parents remained healthy, and were confessedly free from any family taint in the direct line, but at least two of the children were lepers. It will be seen from Table X*b* that in such families 96 patients had 292 brothers and sisters, of whom 111 were lepers: or that selecting these families, of 388 children 207, or 53·3 per cent., were diseased. This is very instructive, as though here an hereditary specific predisposition is excluded as rigorously as possible, yet the disease shows a predilection for certain families.

It is only when examined in this light, that deductions regarding the offspring as a whole can be made with a view to elicit the influence of a specific hereditary predisposition. Now as individuals of alien blood, such as husbands and wives of lepers who live with the family under similar conditions, suffer to the extent of from 5 to 6 per cent., the above arguments are strengthened.[19] Evidently all that can be argued from these considerations is, that leprosy in a certain number of instances has a tendency to affect several members of a family, but that this is not due to a specific hereditary predisposition.

(19) *Cf.* Chapter on Contagion.

TABLE Xa.

Condition of Brothers and Sisters of Leprous Patients whose Parents remained Healthy.

Asylum or Locality.	No. of patients with such history.	No. of their brothers and sisters.	No. of these affected.	Remarks.
Aligarh	6	12	1	It must be remarked, that in several instances when the brothers and sisters of a leper were healthy, their number had not been enquired into, or at times no mention at all made of them. On the other hand, whenever a leper had a leprous brother or sister, full information has always been given. Taking due consideration of this fact, it is evident that the percentage at the end of this table is too high. Agra, Bombay, Calicut, Hyderabad, Subathu and Tarn Taran have been omitted on account of insufficient details. Omissions, however, though few, exist also in some of the other stations. Making due allowance for these, it may be said that 2 per cent. indicates the true ratio.
Almora	74	256	16	
Bangalore	21	71	...	
Belgaum	2	9	...	
Benares	13	32	...	
Calcutta	67	245	2	
Cawnpore	5	11	...	
Conjeeveram	12	54	3	
Darjeeling	1	4	1	
Dehra Dun	67	228	6	
Delhi	7	23	...	
Dharmsala	7	17	2	
Gwalior	9	35	...	
Gya	30	98	3	
Jubbulpore	5	15	1	
Kapurthalla	1	1	...	
Lahore	1	4	...	
Lucknow	2	4	...	
Madras	59	214	15	
Madura	47	209	9	
Mandalay	136	495	7	
Moulmein	16	53	...	
Nagpur	53	131	...	
Naini and Allahabad	8	31	1	
Naini Tal	1	2	...	
Patiala	8	27	1	

Table Xa—*continued.*

Condition of Brothers and Sisters of Leprous Patients whose Parents remained Healthy.

ASYLUM OR LOCALITY.	No. of patients with such history.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Prome	24	65	2	
Purulia	18	41	9	
Rangoon	41	122	1	
Rawalpindi	19	54	6	
Sialkot	22	75	4	
Tanjore	9	31	...	
Thayetmyo	46	156	4	
Trichinopoly	5	20	...	
Umballa	8	18	3	
TOTAL	850	2,853	97= 3·4 per cent.	

TABLE Xb.

Condition of Brothers and Sisters of Leprous Patients belonging to Families where the Parents remained healthy, but at least Two Children were Lepers.

ASYLUM OR LOCALITY.	No. of patients belonging to such families.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Agra	1	1	1	
Aligarh	1	1	1	
Almora	13	49	16	

Table Xb—*continued.*

Condition of Brothers and Sisters of Leprous Patients belonging to Families where the Parents remained healthy, but at least Two Children were Lepers.

ASYLUM OR LOCALITY.	No. of patients belonging to such families.	No. of their brothers and sisters.	No. of these affected.	REMARKS.
Calcutta	2	8	2	
Conjeeveram	3	13	3	
Darjeeling	1	4	1	
Dehra Dun	6	28	6	
Dharmsala	1	4	2	
Gya	2	8	3	
Hyderabad	1	1	1	
Jubbulpore	1	1	1	
Madras	12	25	15	
Madura	9	34	9	
Mandalay	6	26	7	
Naini and Allahabad	1	1	1	
Patiala	1	6	1	
Prome	2	8	2	
Purulia	7	17	9	
Rangoon	1	1	1	
Rawalpindi	5	15	6	
Sialkot	3	5	4	
Subathu	3	3	3	
Tarn Taran	8	22	9	
Thayetmyo	3	8	4	
Umballa	3	3	3	
TOTAL	96	292	111= 37·9 per cent.	

VIII.—Before giving a summary of this chapter, a few remarks must be made upon a point which has an important bearing on the question of heredity: what will be the risk of increase to the leper population, assuming heredity to be a more or less influential agent in the spread of the disease? This matter was thoroughly gone into by Drs. Lewis and Cunningham,[20] who came to the conclusion that "taking all the information attainable there appears to be no great risk of increase to the leper population of Kumaun as far as the disease is dependent on heredity for its multiplication."

Following their line of argument, it will be seen, by referring to Table XI, that 2,915 children were born before or after the manifestation of leprosy in the parent or parents. Almora and Meerut have been left out of consideration, because the total number of children could not be obtained in all instances where both the parents were lepers.

Strictly speaking, only the children actually born of lepers should be considered. However, as here the greatest possible increase to the leper population is under discussion, and this matter is simply a side issue to the question of heredity and of practical rather than scientific value, all objections will be waived, and all the children included. Thus the case will be stated as strongly as possible. Besides, the fact exists that whether the parent was a leper before or after the birth of his children, a certain number of the latter suffer at one time or another. Whether this is due to a specific hereditary influence has been previously discussed, and for the present purpose it will be best simply to accept the fact, and enquire what would be the probable increase to the leper population.

In Table XI all those children who died in infancy have

(20) Loc. cit., page 63.

been excluded for obvious reasons. Now 2,915 children have been the issue of 1,564 marriages. These marriages have been grouped together in Tables XII and XIII. Of these 71 were contracted by individuals both suffering from the disease, so that these would represent 142 lepers. This is not quite correct, as some patients married twice. But while facilitating calculations, it will in no way affect the ratio, and moreover will compensate for omissions among the other leper marriages. These were contracted between a leper and a healthy person and amount to 1,493. Thus 1,493 lepers must be added to the above 142, which gives a total of 1,635. In the case of the 1,493 marriages between a healthy and an affected person the fact that in a few instances the healthy individual subsequently became diseased has been neglected, and thus matters will be equalised.

The 1,635 lepers would be replaced by 2,915 children. A few of the children have already died, but as this fact was not consistently enquired into, it will be best to consider them all to be alive. The 1,635 lepers therefore have contributed a permanent addition of 1,280, for 1,635 of the children must be deducted as merely replacing their leprous ancestors. Now it is certain that all the children would not become lepers. At present only 78 of the children are affected, but this small number may be due to the fact that many of them are still young, and it may be reasonably expected that some of them will show manifestations of the disease at a more advanced age. Tables VII and VIII fully justify this suspicion. However, a certain proportion of them are adults and married and have healthy children.

Taking everything into consideration, it is unlikely that more than 5 per cent. of the 2,915 children will become lepers. This

would indicate that of the total number of children born up to the present, 145 only are likely to become lepers through the agency of heredity. Hence a leprous population of 1,635 individuals may be expected to transmit the disease to 145. This latter number, as far as the statistical tables show, is too high. It would appear probable from Tables VI, VIII, and XI that about 80 of the children would become lepers through the assumed influence of heredity.

Thus there is no danger of a spread of the pest by hereditary transmission and predisposition. In fact the figures show conclusively that not only could there be no increase to the leper population by means of this agency, but that all other causes being removed, the disease must die out. It is, on the strength of these data, not astonishing to find that the disease should have been practically extirpated amongst a community like the above-mentioned Norwegians, who forsook their own country and wandered into fresh areas and surroundings, apparently out of reach of the factors capable of originating the disease. To this point, however, reference will be made when considering the question of contagion.

TABLE XI.

Children born Before and After Manifestation of Disease in Parent or Parents.

Asylum or Locality.	Children born before appearance of leprosy in parent.	Children born after appearance of disease in parent.	Total number of children.
Agra	52	15	67
Aligarh	30	5	35
Bangalore	52	20	72

Table XI—*continued.*

Children born Before and After Manifestation of Disease in Parent or Parents.

Asylum or Locality.	Children born before appearance of leprosy in parent.	Children born after appearance of disease in parent.	Total number of children.
Belgaum	7	2	9
Benares	59	5	64
Bombay	201 (11)	10	211
Burdwan	28	5	33
Calcutta	66	12	78
Calicut	28	20	48
Cawnpore	10	5	15
Conjeeveram	30	12	42
Darjeeling	4	2	6
Dehra Dun	154 (4)	20	174
Delhi	27	6	33
Dharmsala	11	1	12
Fyzabad	16	4	20
Gwalior	8	3	11
Gya	73	5	78
Hyderabad	8	16	24
Jubbulpore	17 (1)	9	26
Jummoo	2	1	3
Kapurthalla	9	3	12
Lucknow	12	1	13
Madras	208 (6)	43	251
Madura	137 (6)	56	193
Mandalay	127 (1)	35	162

NOTE.—The figures in brackets indicate the number of children born before the appearance of the disease in the parent or parents who became lepers: 58 out of a total of 2,447, or 2·3 per cent.

Table XI—*concluded.*

Children born Before and After Manifestation of Disease in Parent or Parents.

Asylum or Locality.	Children born before appearance of leprosy in parent.	Children born after appearance of disease in parent.	Total number of Children.
Moulmein	15	9	24
Nagpur	265 (2)	30	295
Naini and Allahabad	57	17	74
Naini Tal	...	1	1
Patiala	6	1	7
Poona	47 (1)	...	47
Prome	27 (2)	3	30
Purulia	172 (9)	4	176
Rangoon	62	9	71
Rawalpindi	40 (4)	12	52
Sialkot	53	4	57
Subathu	37 (1)	11	48
Tanjore	14 (1)	1	15
Tarn Taran	151 (2)	34	185
Thayetmyo	50 (5)	5	55
Trichinopoly	1 (1)	7	8
Umballa	57	1	58
Yerrowda Prison	17 (1)	3	20
Total	2,447 (58) = 2·3 per cent.	468	2,915

Note.—The figures in brackets indicate the number of children born before the appearance of the disease in the parent or parents who become lepers: 58 out of a total of 2,447, or 2·3 per cent.

TABLE XII.

Marriages.

Asylum or Locality.	No. of sterile marriages.	Marriages with issues		No. of couples the children of which were born before the appearance of the disease in the parent.	Total number of marriages.
		Between two lepers.	Between a healthy person and a leper.		
Agra	8	...	10	20	38
Aligarh	...	..	3	5	8
Bangalore	9	...	12	22	43
Belgaum	6	...	1	2	9
Benares	4	...	4	19	27
Bombay	72	...	10	100	182
Burdwan	7	...	2	9	18
Calcutta	30	...	7	27	64
Calicut	2	...	12	7	21
Cawnpore	2	...	3	5	10
Conjeeveram	1	...	6	6	13
Darjeeling	...	...	2	...	2
Dehra Dun	15	2	8	46	71
Delhi	2	...	...	7	9
Dharmsala	2	...	1	6	9
Fyzabad	5	...	3	5	13
Gwalior	4	...	1	4	9
Gya	11	...	3	27	41
Hyderabad	5	...	6	2	13
Jubbulpore	1	...	5	5	11
Jummoo	...	...	1	1	2
Kapurthalla	1	1	...	4	6
Lucknow	6	...	...	3	11
Madras	13	1	21	52	87
Madura	18	...	24	32	74

Table XII—*continued.*

Marriages.

Asylum or Locality.	No. of sterile marriages.	Marriages with issues		No. of couples the children of which were born before the appearance of the disease in the parent.	Total number of marriages.
		Between two lepers.	Between a healthy person and a leper.		
Mandalay . . .	26	...	15	38	79
Moulmein . . .	6	...	5	5	16
Nagpur	30	...	17	90	137
Naini and Allahabad .	14	...	9	30	53
Naini Tal . . .	...	...	1	...	1
Patiala	3	1	...	3	7
Poona	14	...	...	22	36
Prome	9	...	3	8	20
Purulia	12	...	3	63	78
Rangoon . . .	16	...	6	19	41
Rawalpindi . . .	7	7	2	16	32
Sialkot	14	1	1	21	37
Subathu	26	1	3	13	43
Tanjore	4	...	1	5	10
Tarn Taran . . .	33	12	9	52	106
Thayetmyo . . .	7	...	3	20	30
Trichinopoly . . .	5	...	2	1	8
Umballa . . .	8	...	1	20	29
Yerrowda Prison . .	3	...	1	6	10
Total .	461*	26	227	850	1,564

IX.—Again, the diminished procreative power of lepers and the comparatively high mortality among their offspring operate power-

* Of these 45 were severally contracted by two persons, both lepers.

fully against the possible risk of increase in the leper population. The latter point is well established, and Drs. Lewis and Cunningham have drawn attention to it so prominently in their report that it would be useless to enter into any further discussion upon the subject. It may therefore be stated that a certain number of children born of leprous parents would be probably short-lived.

There is also no doubt that the reproductive power of lepers is diminished. It is fully established through clinical and pathological observation, that leprosy causes extensive morbid changes in the sexual glands and disturbs the sexual functions. That this applies to men has never been denied. But that it is equally true of women has been proved by Dr. Arning,[21] who also demonstrated the existence of a leprous ovaritis. It has been shown from Table IV that the issue of 98 marriages between two lepers amounts to only 65. Again Table XIII shows that of 85 couples no less than 55, or 64·7 per cent., were sterile. Hence, assuming all the 65 children to live and grow up, according to these figures 168 lepers (*cf.* Table XIII) would be replaced by 65 persons, that is, there would be an actual decrease of 62 per cent. in such a population. Assuming that 5 per cent. of the 65 children became lepers, it seems that 168 lepers would be replaced by 3 or 4 lepers, or if the diffusion of the disease depended on heredity only, there would be an actual decrease of almost 99 per cent. in the leprous population. No doubt these figures are small, yet they allow of the following conclusions:—(*a*) There is no evidence that marriages among lepers in any way *per se* increase the risk of diffusing the disease, as far

(21) Journal of the Leprosy Investigation Committee: No. 2, Feb. 1891, page 127.

as heredity is concerned. (*b*) Even allowing the latter the fullest play and setting aside all other causes of leprosy, the disease would gradually die out in such a population as has just been considered. Intermarriages, therefore, amongst lepers cannot be regarded as harmful to the community, or as calculated to spread the disease, so far as this depends on hereditary transmission or predisposition.

But how does the case stand when the marriages between a healthy person and a leper are considered? It is more difficult to acquire absolutely accurate information from the data obtainable, and the following statements must be regarded as suggestive only. In almost every instance husbands and wives were healthy at the time of marriage, and one or other contracted the disease some years later. It would evidently not be fair to compare the numbers of children born before, with those born after, the appearance of the disease in either husband or wife, and then draw deductions as to the influence of leprosy on the reproductive power. For, firstly, in a large number of cases one person forsakes the other as soon as the disease becomes marked, and, secondly, by the time the disease manifests itself the age of the patient is often so advanced that the loss of reproductive power may be attributed to organic and functional causes.

Two tables have been prepared (Tables XIV and XIV*a*) to show how far leprosy may be said to influence the reproductive power of the persons affected. Only such persons have been chosen who cohabited for at least five years after the appearance of the disease, and all patients over forty years of age have been rigorously excluded. In this manner it may be hoped to obtain a few suggestive facts.

In Table XIV such marriages where the husband only was a

leper have been grouped together, and it is found that of 296 couples 176, or 59·4 per cent., had no issue after the appearance of the disease in the husband, while the progeny of the 120 non-sterile couples amounted to only 249 individuals. That is, under the assumed conditions, the average number of children to each couple amounts to less than one.

Again from Table XIV*a* it is seen that of 88 couples where the wives only were affected, no children were born in 62 instances after the manifestation of the disease, the total number of children born to the diseased mothers amounting to 49. Thus 70·4 per cent. of the women became sterile with the appearance of the disease, and the average number of children to each is considerably less than one.

Though these statistics evidently cannot represent the absolute truth, they at least justify the belief in a loss of procreative power causally related to leprosy, and tend to diminish the fear of a diffusion of the disease through marriage with lepers. This is also fully borne out by the history of the leper settlement at Kalawao (Hawaii).[32] The latter was established in 1866, and 2,864 persons were consigned there as lepers from that time to 1884. At the end of this period of eighteen years, 26 children survived who had been born at the settlement of leper parents, and only two of these 26 were lepers.

From all these considerations then it may fairly be concluded that marriages among lepers, and with lepers, do not increase the risk of a diffusion of leprosy by means of the offspring, and this to a great extent is due to the relative sterility of leper whether males or females.

(32) Report of Dr. Fitch: Appendix to Report on Leprosy in Hawaii, 1886, page

TABLE XIII.

Table showing the Fertility of Leprous Couples (both Husbands and Wives being Lepers).

NAMES OF ASYLUM, ETC.	No. of leper couples.	No. of such without any issue.	REMARKS.
Almora	9	4	A leper woman married twice.
Belgaum	1	1	
Bombay	3	3	
Dehra Dun	4	2	In one case a child was born who died as an infant. No children since.
Kapurthalla	2	1	A leper woman married twice.
Lucknow	2	2	
Madras	1	...	
Mandalay	1	1	
Meerut	6	6	
Nagpur	1	1	
Patiala	1	...	
Rangoon	1	1	
Rawalpindi	14	7	
Sialkot	14	13	
Subathu	2	1	
Tarn Taran	22	11	
Umballa	1	1	
TOTAL	85 = 168 persons.	55 = 64·7 per cent.	

TABLE XIV.

To show the Relative Sterility of Couples where the Husband is a Leper and the Wife Healthy.

ASYLUM OR LOCALITY	Total number of such couples.	No. of those which are sterile.	No. of those which have children.	No. of children.
Agra	12	2	10	15
Aligarh	3	...	3	5
Almora	4	3	1	3
Bangalore	14	6	8	14
Benares	3	...	3	4
Bombay	7	7	...	...
Burdwan	4	3	1	2
Calcutta	11	8	3	6
Calicut	10	2	8	16
Cawnpore	5	4	1	2
Conjeeveram	9	4	5	10
Darjeeling	1	...	1	1
Dehra Dun	5	1	4	7
Delhi	2	2	...	...
Fyzabad	5	2	3	4
Gwalior	5	4	1	3
Gya	9	7	2	2
Hyderabad	9	5	4	11
Jubbulpore	5	1	4	8
Lucknow	2	2	...	...
Madras	27	17	10	24
Madura	32	16	16	44
Mandalay	19	12	7	12
Moulmein	3	...	3	7
Nagpur	25	21	4	11

Table XIV—*continued.*

To show the Relative Sterility of Couples where the Husband is a Leper and the Wife Healthy.

Asylum or Locality.	Total number of such couples.	No. of those which are sterile.	No. of those which have children.	No. of children.
Naini and Allahabad	15	10	5	10
Prome	9	6	3	6
Purulia	12	12	...	...
Rangoon	6	6	...	...
Sialkot	1	...	1	1
Subathu	3	1	2	5
Tanjore	3	2	1	1
Tarn Taran	6	2	4	8
Thayetmyo	5	4	1	1
Trichinopoly	5	4	1	6
Total	296	176 =59·4 per cent.	120	249

TABLE XIVa.

Table to show the Relative Sterility of Couples where the Wife is a Leper and the Husband Healthy.

Asylum or Locality.	Total number of such couples.	No. of those which are sterile.	No. of those which have children.	No. of children.
Almora	2	1	1	1
Bangalore	9	6	3	5
Bombay	6	5	1	1
Calcutta	9	6	3	4
Cawnpore	1	...	1	1

Table XIVa—*continued.*

Table to show the Relative Sterility of Couples where the Wife is a Leper and the Husband Healthy.

Asylum or Locality.	Total number of such couples.	No. of those which are sterile.	No. of those which have children.	No. of children.
Conjeeveram	1	1	...	...
Darjeeling	1	...	1	1
Dehra Dun	3	3	...	...
Delhi	1	...	1	6
Gya	1	1	...	...
Hyderabad	1	...	1	3
Madras	7	4	3	4
Madura	8	6	2	3
Mandalay	8	4	4	10
Moulmein	1	1	...	...
Nagpur	13	11	2	6
Purulia	6	6	...	...
Rangoon	1	1	...	...
Rawalpindi	1	...	1	1
Tanjore	2	2	...	...
Tarn Taran	5	3	2	3
Umballa	1	1	...	...
Total	88	62= 70·4 per cent.	26	49

Summary.

The Commissioners have thus come to the conclusion that there is no evidence that leprosy in India is transmitted through heredity from parent to child, their reasons being—

(1) no authentic congenital case has ever been put on record, nor was one seen in this country;

(2) true family histories of leprosy could be obtained in only 5 or 6 per cent. of the cases;

(3) many instances occur of children being affected while their parents remain perfectly healthy;

(4) the percentage of children, the result of leper marriages, who become lepers is too small to warrant the belief in the hereditary transmission of the disease;

(5) the facts obtained from the Orphanage at the Almora Asylum disprove the existence of a specific hereditary predisposition;

(6) only 5 or 6 per cent. of the children born after the manifestation of the disease in the parents become subsequently affected;

(7) the histories of the brothers and sisters of leper patients with a true or false hereditary taint seem to show that little importance can be attached to inheritance as an agent in the perpetuation of the disease.

For the same reasons it may be assumed that the specific hereditary predisposition to leprosy is but slight and practically does not exist.

Lastly, it has been shown that taking all the information obtainable and even allowing the fullest influence to heredity, there appears to be no risk of an increase to the leper population of India, so far as the disease is dependent on heredity for its multiplication, and that marriages with, or intermarriages between, lepers cannot be regarded in the light of a danger to the community.

CHAPTER V.

Contagiousness of Leprosy.

ALL modern authorities are agreed that leprosy is an infective disease, that is, one caused by a microbe, the bacillus lepræ, which obtains access to the body from without, and subsequently multiplies within the organism, calling forth the characteristic lesions. The next point to be decided is whether or no leprosy is *contagious*.

It is only too frequently taken for granted that a disease due to a pathogenic germ is necessarily also contagious. That this need not be the case will be shown later on. Here it may suffice to recall to mind two instances of infective diseases which are by no means contagious, *viz.*, malaria and pneumonia.

With regard to the contagiousness of leprosy, the views of authors are highly conflicting, and a brief allusion will be made to the statements of some of them. These matters have, during the last few years, been so much before the world that this reference need only be very brief. Flügge[1] says: "the diffusion of leprosy by contagion is *exceedingly rare* and evidently can only take place *under special and predisposing favourable conditions*" (the italics are not Flügge's). Now it may be mentioned at once, as this will guide subsequent arguments, that the more weight is attached to special favourable conditions, in fact what in clinical language is called "the suitable nidus," the further contagion disappears into the back-ground.

(1) C. Flügge: Die Mikroorganismen. Leipzig, 1886, page 221.

Indeed, for all practical purposes—and these are what the legislator or sanitary reformer has to consider—it vanishes altogether.

Hansen,[2] on the other hand, is a contagionist, and expresses himself thus: "I do not know the way in which leprosy is communicated or transferred from a leper to a sound person, but I am most inclined to believe that it is done by a sort of inoculation, that there is wanted a very intimate intercourse and some defect in the integument of the infected." Now, in the chapter on Heredity mention has been made of Hansen's researches in America. And it seems strange that he should consider the fate of the emigrated Norwegian lepers an absolute proof against an hereditary transmission of the disease, while he is unwilling to allow that the same argument applies *à fortiori* against contagion. How is the fact to be explained that in a community of one hundred and sixty lepers the disease should die out amongst their descendants, without altogether disbelieving in contagion, or attaching to it a nominal importance? In fact Hansen's facts seem to disprove the theory of contagiousness much more forcibly than that of hereditary predisposition.

Leloir,[3] who is also a contagionist, defends his views in the following words: "Mais comme l'a bien dit le docteur Vidal le 13 octobre 1885 à l'Académie de médecine, il faut remarquer que beaucoup de maladies dont la transmission par voie d'inoculation n'est plus niée aujourd'hui présentent souvent des inoculations difficiles, irrégulières telles la varicelle, la diphthérie, telle la tuberculose, etc. On pourrait ajouter à la liste des affections le lupus, l'érysipéle lui-même." This statement challenges some comment. Firstly, there

(2) A. Hansen: Journal of the Leprosy Investigation Committee, No. 2, and Lancet, June 28, 1880, page 1431.

(3) H. Leloir: Traitè Pratique et Théoretique de la Lépre. Paris, 1886, page 301.

can be no doubt as to the ease with which diphtheria and erysipelas can be inoculated, and it is a well-established clinical fact that these two diseases spread *par excellence* through contagion. Very few would venture to place them in the same category with leprosy, and attempt to establish any analogy between them and the latter. Secondly, if the contagiousness of leprosy is no greater than that of lupus or tuberculosis, the whole matter will be reduced to a question of abstract scientific interest, and is thereby at once removed from the sphere of the legislator, in so far as such strong measures as segregation and social interference are concerned. No one would ever think of enforcing such measures for lupus or tuberculosis.

Lastly, reference must be made to Virchow,[4] who, in 1859, was asked by the Norwegian Government to investigate the disease in their country, and examined hundreds of cases. He sums up his views on contagion as follows: "Therefore all we can say is, that all cases of supposed contagion or inoculation brought forward may be explained in various ways, and moreover are so rare that as far as the general ætiology of the disease is concerned they may in any case be disregarded." Practically, leprosy, according to him, does not spread by contagion, though it may scientifically be correct to classify it amongst the contagious diseases. This point will be further considered in another place.

Before giving a detailed account of the views held by the Commissioners, it will be necessary to make a few general remarks on some points which should guide the enquirer in a similar research, and also to criticise the forms of argument used by many authors. The importance of the subject will excuse this digression.

Firstly, when the question of contagion is under consideration, it

(4) R. Virchow: Krankhafte Geschwülste, Vol. II, page 507.

is of the utmost importance to separate the abstract and scientific meaning of the term contagion from the practical one. It is not sufficient to determine by experiment, or on the evidence of one or two authentic clinical cases, that a disease is infective and contagious, but a careful enquiry must be made into the part played by contagion under ordinary surroundings and practical conditions.

Secondly, the connotation of the term infection must not be misconstrued or unduly extended. Bacteriological and laboratory researches have certainly contributed much to the knowledge of the ætiology of many diseases, but the actual question of contagion cannot be settled in this way. Clinical and epidemiological evidence is of the greatest importance.

To elucidate these points, let it be supposed that a disease is known to be an infective one. When it is acknowledged that a disease is infective, all that is asserted is, that "the disease may be transmitted from the affected to the healthy individual," but this premises nothing as to the natural mode of diffusion of the disease in question, and whether, or to what extent, under ordinary practical conditions, such transmission does take place. "The natural mode of transmission of an infective disease depends on the faculty pathogenic germs possess of leaving the body of the diseased in the full possession of their power of setting up an infection. If they be thrown off from any surface of a diseased organism in sufficient quantity, and in a sufficiently resistant state, a transmission of the disease from the affected to the healthy person *may* take place, and the diffusion of the disease in such cases, under certain conditions, *may* be effected through contagion.* If, however, the pathogenic

* If, however, the germs or their spores already are, or become, widely distributed throughout space, the risk of contagion may be disregarded as compared with the *de novo* acquisition of the disease.

germs reproduced in the body of the diseased person never leave the latter, or only do so without possessing the power of causing subsequent infection, the infective disease caused by such germs is non-contagious. The germs in such cases must have a place somewhere, whence they can always *de novo* attack the healthy organism."[5]

Amongst the infective contagious diseases remarkable differences exist as to the degree of contagiousness, depending partly on the unequal liability of the healthy body to be invaded by germs, and partly on the resistance and quantity of germs leaving the diseased body. Again, the paths by which the microbes can enter may be such as only in rare cases permit of their establishing themselves within the tissues, and finally, the human body may possess certain protective means which offer great obstacles to the attacking germ.

A disease may thus scientifically be grouped amongst the infective and contagious diseases, and yet practically and clinically hardly deserve this name. It is to be regretted that the modern advances of bacteriology and animal inoculations have tended to make not only the public, but also scientific writers, disregard the evidence derived from clinical and epidemiological experience. Without, for a moment, under-estimating the importance of bacteriological and animal experiments, it is necessary to guard against taking a one-sided view of the matter, a tendency to which, in these times, has often been only too manifest. Experimenting, as the bacteriologist generally does, on highly susceptible animals, he easily runs the risk of arguing beyond his premises, and of drawing conclusions from his experiments which he applies without sufficient reserve to the natural mode of infection. It is quite impossible to deduce the ætiology of an infective disease from bacteriology alone; clinical and epide-

(5) C. Flügge loc. cit. and also Flügge: Grundriss der Hygiene, page 466.

miological observations carefully made, must always be taken into account. A susceptible animal may easily be infected by experiment, and yet the case may be quite different with man.

The best example to elucidate this point will be found in tuberculosis. The researches of Koch have shown that this disease is not only infective but also contagious. It is, however, equally certain that in the ordinary human surroundings the conditions necessary for the multiplication and proliferation of the tubercle bacilli *never* exist. The diffusion of the disease depends thus, firstly, on its transmission from one individual to another, and, secondly, on the fact that the bacilli may remain dormant, in the full possession of their virulent properties, for a considerable time, without, however, as was just mentioned, showing any signs of proliferation. They are in fact true parasites. Animals, such as guinea-pigs, are infected with the greatest ease. Again, the tubercle bacillus, as Cornet[6] has shown, is widely distributed throughout space without any loss of vigour. *À priori* one would be inclined to assume that tuberculosis must be a highly contagious disease. But experience teaches beyond doubt that of healthy individuals living in the immediate neighbourhood of phthisical patients, by no means all become affected; "in fact perhaps only slightly more than would become affected if the opportunity for infection were less."[7] The *disposition* of an individual is of the highest importance in bringing about a tubercular infection. In fact, as Flügge says, "the disposition actually governs the mode of diffusion of tuberculosis." And that in tuberculosis the danger of infection is so small is chiefly explained by the fact that the existence of the bacillus, and its introduction into the human

(6) G. Cornet: Ueber Tuberculose. Leipzig; and footnote on page 287.
(7) C. Flügge: loc. cit., page 219.

body, are not sufficient to bring about the infection without the necessary individual disposition.

The causes and nature of such disposition may be unknown, but that it exists no one denies, and its bearing on contagion is evident. The experiments of Monti, Leo, Charrin, Roger, and E. Klein[8] have thrown much light on the nature of disposition and susceptibility. These will be more fully alluded to in Chapter VI. The greater the prominence given to this disposition, the less the absolute influence of contagion becomes. "We notice the best example of an individual disposition in tuberculosis, where the greater or smaller collection of resistent germs around the human body has, as we know from experience, a subordinate importance in the diffusion of the disease. It has become an almost general practice in tuberculosis to counteract nearly exclusively this individual disposition, and not, as with other infective diseases, aim at a careful removal of the pathogenic germs."[9]

Keeping these points in mind the nature of the contagiousness of leprosy will be discussed from a clinical point of view, and the results gained from bacteriological work will be given in the Laboratory Report. The evidence collected by the Commissioners during their stay in India can, in the first instance, only apply to this empire. They have come to the following conclusion, that *though they consider leprosy an infective disease, caused by a specific bacillus, and moreover also a contagious disease, they are of opinion that there is not sufficient evidence that leprosy is maintained or diffused by contagion; indeed under the ordinary human sur-*

(8) Nineteenth Annual Report of the Local Government Board, 1889-90, Supplement, page 217.
(9) C. Flügge: loc. cit., page 614.

roundings the amount of contagion which exists is so small that it may be disregarded, and no legislation is called for on the lines either of segregation, or of interdiction of marriages with lepers.

It would be useless in an enquiry of this nature to take the word of the native inhabitants. That the native ordinarily does not fear the disease may be easily gathered from the fact that in bazaars leprous vendors are not rarely found selling food or sweets. Again, however unclean the natives may consider a leper, as long as he is in the possession of money, he is almost always welcome. The readiness also with which the native police and their officials handled lepers brought up for inspection before the Commissioners was very noticeable. There are of course differences in this respect in the various parts of India, and the Commissioners can only speak with confidence of the places visited. This, however, has already been discussed in another chapter. It has all along struck the Commission that though a native on being questioned will, as a rule, state that leprosy is a contagious disease, yet his own acts do not support his statements.

Neither would it be permissible to be too much guided by the opinions of medical observers in India. The leper, not really being such an outcast or deplorable individual as European opinion considers him to be, is seldom seen in the dispensaries, preferring to haunt bazaars or other crowded places to beg. Most Civil Surgeons have not much opportunity, therefore, of studying the ætiology of this disease, with the exception of such of them as happen to have large asylums under their jurisdiction. Moreover, the knowledge that no treatment of an outdoor nature is of any service

in these cases prevents Civil Surgeons from encouraging their attendance.

The Commission have only been guided by facts obtained by themselves. From these they deduce the following arguments against a contagion which they deem worthy of a practical consideration.

I. All the cases brought forward and demonstrated to the Commissioners as instances of contagion have broken down with one possible exception. These, as a rule, concerned hospital or asylum servants. A list of these cases is appended at the end of this chapter (Table I). Now it seems very significant that the cases which were supposed to prove the contagion theory, and on which such theory was really based, almost entirely collapsed.

II. In not a single case could contagion, or the possibility of contagion, be actually demonstrated in a manner free from objection. The native leper, in an overwhelming majority of instances, offers no suggestion as to the contraction of his disease; denies that he has ever had any contact or intercourse with lepers, and simply accuses fate. Occasionally they mentioned that for some time they had eaten with lepers, but their stories were in most instances too extravagant for belief. For instance, a patient who had been a sailor, says that while in Demerara, a leper used to visit his ship, and all the crew used to smoke the same hookah with this leper. Two years later this man contracted leprosy. Similar stories are not rare, but they certainly cannot be regarded seriously. In other cases a man will relate that many years ago he came into contact with a leper. Such statements have led some authors to assume a long incubation period for the disease. Now it may be questioned whether it is still possible to talk of contagion after an incubation

period, which, in many instances, must have extended over ten or more years. Whatever view may be taken of this matter, the assumption of an irrationally and disproportionately long latent period is certainly a weak point in any theory of contagion. And it becomes still weaker when it is considered that, in the case of leprosy the people who presumably have been exposed to the risk of contagion during this "long incubation period," have lived in an endemic area.

Special attention was paid by the Commission to the effect of people eating with lepers. Table V, at the end of this chapter, shows that seventy-nine lepers ate and drank for some time out of the same vessels with two hundred and five healthy people, and of the latter 7 per cent. afterwards became lepers (Table V). Now these cases all occurred in families. But it has been shown in Chapter III that leprosy has a tendency to appear in families, though the cause of this is not a specific hereditary transmission, nor an hereditary predisposition, but most likely explicable by the fact that the members of a family are more likely to be subjected to the same abiding external causes. But in any case the figures obtained from Table V do not permit a great influence to be conceded to contagion, even if the instances of possible contagion were quite free from all objection.

III. In a family contagion has of course the widest play. However, it has been found that the disease does not spread sufficiently amongst the members of one family to warrant the conclusion that it is contagious to any extent. Table II shows that an assumed contagion could be traced in only 5 per cent.

IV. Again, when the intimate intercourse between man and wife is considered, it is astonishing how seldom the wife "catches" the

disease from her husband, or *vice versâ*. Yet it is well known that bacilli may easily be found in the spermatic fluid,[10] and here then there is an opportunity offered for oft-repeated inoculation through the mucous membrane of the genital tract. Yet a case is hardly ever found where *bonâ fide* the disease may be assumed to have spread from husband to wife (Tables III and III*a*).

V. Next, vaccination has been mentioned as affording a risk of diffusing leprosy. This statement is not borne out by facts ascertained in India. Firstly, the number of persons actually vaccinated in India is very small in comparison with the general population. Taking the total number of people in Bengal, North-Western Provinces and Oudh, Punjab, Central Provinces, Berar, Lower Burma, Assam, Madras, Bombay, and Coorg, it appears that in the year 1889 of 7,985,543 children available for vaccination only 2,178,464, or 27·2 per cent. were vaccinated.[11] Secondly, a considerable number of these were vaccinated from the calf. Thirdly, where arm-to-arm vaccination is practised, lymph is taken from the vaccinifer at an age when leprosy rarely occurs. Fourthly, the experimental evidence of the Norwegian doctors, quoted by Leloir, seems to indicate that it is highly questionable whether such inoculation, as vaccination implies, would be able to produce the disease in another person, even granted that the vaccinifer were a leper, and that the lymph contained bacilli. Lastly, as will be seen more fully from the Laboratory Report, three of the Commissioners vaccinated a number of lepers over healthy and affected areas, and subsequently examined the lymph for

(10) Nature, Origin, Transmissibility, the Methods of Propagation and Transmission of Leprosy, by M. E. Besnier. Translated by Surgeon-Major O. Baker, Calcutta, 1889, pages 8 and 10.

(11) Twenty-sixth Annual Report of the Sanitary Commissioner with the Government of India, 1889. Appendix to Section VII, page 164.

the characteristic bacilli. In no case were undoubted leprosy bacilli found, either in the lymph or crusts; of ninety-three specimens only six were slightly suspicious (Table VI at the end of this chapter). It has been ascertained by experiment (Laboratory Reports) that if a blister be raised over apparently healthy skin, its serous fluid will be quite free from the bacilli, while in blisters raised over leprous nodules, bacilli may in some cases be found. And surely no one would ever think of vaccinating an individual over a tubercle or diseased part, or if he did so, would employ such lymph to vaccinate another individual. The practical danger, therefore, of leprosy being diffused in this manner, even supposing the disease to be highly contagious, is minimal. The instances quoted in literature of leprosy being "vaccinated" on to a healthy child are too equivocal and ambiguous to carry much weight.[12]

Dr. Arning,[13] who obtained more positive results and found bacilli in the vaccine lymph, states distinctly that he obtained bacilli only in cases of extensive cutaneous leprosy. The latter fact removes at once all fear, because all vaccinators would have some hesitation in vaccinating from such a person. Surgeon-Major Oswald Baker's results,[14] on the other hand, fully agree with those of the Commission, although, in his vaccination experiments, he selected sites solely with reference to the presence of characteristic patches of leprosy. From what has been said it is evident that practically there is no risk of a diffusion of the disease in vaccination from arm to arm.

VI. Attempts were made to enquire into the possibility of a

(12) W. T. Gairdner, Brit. Med. Journ., 1887; and Daubler, Monatschr. f. prakt. Dermat. 1889.

(13) Journal of the Leprosy Investigation Committee, No. 2, Feb. 1891, page 131.

(14) Nature, Origin, Transmissibility, etc., of Leprosy, by M. E. Besnier. Translated by Surgeon-Major O. Baker, page 4.

leprous community becoming or acting as a centre whence the pest might be diffused amongst the population. A research of this nature is obviously connected with great difficulties in a vast country like India.

The census returns present strong arguments against such a spread of leprosy, since the ratio figures remain more or less unchanged. An absolute value, however, cannot be attached to these figures, as here the diagnostic powers of laymen and the inaccurate statements of natives are liable to mislead an enquirer. Nevertheless census returns do possess a high relative value, especially if, as in the present instance, opportunities are offered of comparing the statistics of three decennial periods. On these points reference must be made to Chapter III of this report.

Tarn Taran allows valuable deductions to be drawn as to the risk of the spread of disease around a leprous community. A quarter of the town, in absolute continuity with the rest, is kept apart for, and inhabited solely by, lepers. This condition of things, however, has not caused a diffusion of the disease among the healthy population, the number of lepers remaining more or less stationary, or, at least, never increasing beyond the original ratio. Nor has any diffusion, even to the immediate neighbourhood of the leper quarter, ever been known to occur. Lepers wander to Tarn Taran to bathe in its holy tank, which is supposed to possess special healing powers, and thus the number of lepers is always maintained. But the disease is not especially prevalent amongst the people of the district, and though this leper quarter has existed for many years, the disease has not increased amongst the original inhabitants. For the figures in support of this statement, reference should be made to Chapter III, where this point is specially discussed.

Unfortunately on account of insufficient data this line of argument cannot be followed out. But it may be remarked, and taken for what it is worth, that it is an almost universal opinion of scientific and intelligent men in India that a spread of the disease has never been known to take place from a leper centre to its neighbourhood.[15]

VII. It is believed in some parts of India, especially in the hill tracts, that those who go barefoot are liable to be directly inoculated with leprosy through wounds or ulcers of the feet. In one station in Burma, visited by the Commissioners, they were informed that leprosy had spread among the members of a school after lepers had been in the habit of walking about the compound. As, however, an examination of some of the supposed lepers in this school showed that several of them were not suffering from this disease, this statement was considered open to doubt.

At Almora an opportunity occurred of examining the paths and banks in the asylum compound, on which the lepers were in the habit of walking and sitting. One hundred cover-glasses were prepared from earth taken from these sites, and only ten leprosy bacilli were found in the whole number of specimens. But at present no criterion of a living leprosy bacillus exists, and in any case the small number found in an extended series of observations, on the most favourable material, would suggest that the danger of individual inoculation is possible, but that the risk of the diffusion of the disease in this manner is very slight. On the other hand, it is noteworthy that an examination of dust from huts inhabited by lepers suffering from the tubercular and anæsthetic types of the disease, and afflicted with ulcers on their feet and hands, confirmed the

(15) *Cf.* Chapter II: Belgaum and Bombay.

observation of Kaurin,[16] who has not succeeded in finding bacilli in the earth, dust or air in the rooms of lepers (Table VII).

VIII. This chapter may be concluded by stating that the Commissioners during their enquiries met with many cases where people, either voluntarily or for various reasons, lived in asylums with lepers, often eating and drinking with them, or smoking with them, perhaps attending to their wants and ailments, and yet remained perfectly untainted, often after many years of such association. The value of such instances may be questionable as evidence against contagion, being entirely on the negative side, still they are highly instructive in throwing light on the practical dangers of contagion, and until an actual positive case, free from all objection, is published, increase in worth. Such an absolutely positive case so far is not known, as a perusal of the Journal of the Leprosy Investigation Committee will show. In the absence of such information, it is permissible to deny the practical possibility of the spread of leprosy by contagion. A case can only be positive, if it answers all the conditions postulated by Virchow: "It must be an observation made with the full assurance and every guarantee of certainty." A study of Tables IV and IV*a* will show that of one hundred and four persons living with lepers in asylums, under the most favourable conditions for contracting a communicable disease, only one, or possibly two, became lepers (1 or 2 per cent.).

Summary.—In coming to the conclusion that, as far as practical conditions and surroundings are concerned, the danger of leprosy being diffused and spread by contagion is exceedingly small, arguments have been used which do not possess the character of novelty, India, however, offers no other, and since extended and numerou

(16) Kaurin: Journal of the Leprosy Investigation Committee, No. 2, Feb. 1891, page 68.

experiments obviously cannot be made on man, and knowledge gained from animal inoculations cannot be entirely relevant, deductions can only be drawn from clinical evidence. This clinical evidence, for India at any rate, is strongly against a measurable contagiousness of leprosy. It is in fact with leprosy exactly as with tuberculosis, and all that has been said about the latter affection may be applied equally to the disease which forms the subject of the present enquiry.

In leprosy, therefore, the disposition of the individual plays an important part, just as it does in phthisis. With the assumption of such disposition the practical importance of contagion is reduced to a minimum, and it becomes the duty of the reformer to find means to prevent and counteract this disposition. With the discovery of the bacillus lepræ by Hansen the infective nature of the disease has been finally established, and subsequent experiments have also shown that in the scientific sense of the word the disease must be classed amongst the contagious diseases as defined above. But these facts must not obscure all other evidence, and it must not be forgotten that contagion is but a relative term, and that where reforms and similar measures are under consideration, the actual and concrete influence of contagion is the factor to be considered. This, however, has been shown to be as small as, or even rather less than, in the case of tuberculosis.

APPENDIX TO CHAPTER V.

Several tables, with some necessary comments, will now be given to illustrate the arguments used in the previous pages.

TABLE I.

Cases brought forward in Proof of the Contagiousness of Leprosy.

(*1*) *Agra.*—One of the dressers whose duty it was to rub gurjun oil into the skin of lepers was said to have developed leprosy in consequence. The hospital assistant, however, without being asked, volunteered the statement that this dresser was a leper before he commenced his duties.

(*2*) *Ajodhya.*—A woman was supposed to have caught the disease from her husband. On visiting her in her own home it became evident that she was not even a leper, but had suffered from paronychia of the thumb of one hand, which had led to a slight deformity of the nail and ungual phalanx.

(*3*) *Bombay.*—(*a*) A patient in the Trombay Asylum emigrated to Mauritius, and returned to India in 1884 with swelling of the eyebrows and black patches on the cheeks. When this asylum was opened in 1885 he began work as a ward boy, and continued in that capacity until a year ago, when signs of leprosy became so marked that he became a patient in the asylum.

(*b*) A student in Bombay, while making an autopsy on a leper, pricked his hand. A short time afterwards he suffered from fever, and the hand became inflamed and swollen. Pain and thickening in the course of the ulnar nerve followed, and later there was wasting of the muscles supplied by this nerve together with anæsthesia over the area supplied by it. Similar nerve symptoms then occurred on the opposite side, and the patient suffered from pain in the region of the heart. He was under treatment for some time, and the symptoms all disappeared. Shooting pains in the course of the ulnar nerves recurred during some months, but eventually disappeared again and he remained quite well. At no time did any discoloured anæsthetic patches occur on the body, and the anæsthesia and wasting were confined to the distribution of the ulnar nerve.

This history clearly points to a neuritis set up by septic infection, which subsided under treatment. Yet the case when it first occurred was quoted as an example of the inoculability of leprosy.

(4) *Calicut.*—The patient had been a sweeper at the asylum for twenty-three years. In this capacity he also had to dress the worst ulcers. He used to mix freely with the lepers and eat what they left. According to his own assertions he was quite well twenty-three years ago and developed leprosy only two years ago. He belonged to Calicut and was of a very low caste. His father was not a leper, and this was confirmed by the cook of the asylum, who had resided there for thirty years. The father had been a sweeper for six years, his son succeeding him on his death. The patient's mother, and his six brothers and sisters, were all healthy. An old inmate of the asylum, on the other hand, asserted that the sweeper had suffered from leprosy for at least six years, but was apparently healthy twenty years ago.

This case is the only one quoted in support of contagion which has not utterly broken down, and whatever value it possesses in the way of positive evidence must be conceded.

(5) *Madras.*—The cook of the asylum had been employed for fifteen years, and was suffering from incipient leprosy. He had a discoloured and anæsthetic patch over the right knee, and a similar patch on the right side of his face. He asserted that these changes were of six months' standing, and denied all family history of leprosy.

On making enquiries of an old patient (an intelligent Eurasian and a pupil-teacher), it was found that the patch in the face existed before the cook came to the asylum; moreover, at that time he attended as an out-patient and was treated with gurjun oil. Without the knowledge of the patient his brother was interrogated, and he assured the Commissioners that the patch on the face had existed over fifteen years, and that their mother, another brother, and one of the sister's sons also were lepers. This witness had always considered his brother to be a leper.

It is quite clear that this case is valueless as an instance of contagion.

(6) *Tanjore.*—A Hindu, aged 45, who had suffered from leprosy for ten years, stated that his wife had lately become a leper. He had constantly lived with her. The Commission visited the latter at her house and found on examination that she complained of a feeling in the soles of her feet as though she were walking on wool. She had no anæsthesia or patches, and was certainly not a leper.

(7) *Trichinopoly.*—A patient in the asylum stated that he suffered from a skin eruption eleven years ago. He wished to be admitted into the asylum as a patient, but admission was refused as the disease was too slight. He was employed as a ward cooly, and used to dress the lepers' ulcers. He contradicted himself as to dates, but was certain that the disease appeared after he joined the asylum.

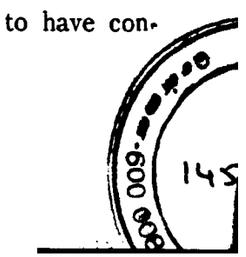
On referring to the records of the asylum it was found that the patient "was admitted as a leper on the 25th of March, 1880, and discharged on the 17th May, 1880," when he was said to be cured. Further it was found in the letter book of the asylum that the patient commenced his duties as ward cooly in July, 1882, during the absence on leave of the former ward cooly. This man, who happened to be in Trichinopoly, was questioned separately and without his having had any communication with the patient. He stated that when he returned from leave he threw up the appointment, and the patient was confirmed in it. Three years later the leprosy became more marked, and he was placed on the asylum register as a patient.

In this case the statement of the patient, that he contracted leprosy after he became an attendant at the asylum, was clearly disproved by the evidence of the asylum records and that of his predecessor in office.

The above eight examples illustrate very clearly the class of cases usually adduced to prove the contagiousness of leprosy. The cases at Madras, Agra, Trombay, Calicut, and Trichinopoly all relate to persons employed, at one time or other, in the asylums. At Ajodhya and Tanjore the evidence offered is that of husband and wife, while at Bombay the case quoted is that of a student who accidentally infected himself during an autopsy on a leper. For the details of the last case the Commissioners are indebted to Surgeon-Major Hatch of Bombay. The case was published when it first occurred, and was widely quoted as an example of the inoculability of leprosy.[17] The first symptoms, indeed, were very suspicious, but the Commission are of opinion that the further progress of the case, as related by Dr. Hatch, entirely negatived the original diagnosis of leprosy. Besides, it is well known that infective processes are not unfrequently followed by peripheral and central nervous changes.

In the two cases in which the wife was supposed to have con-

(17) *Lancet*, May 17, 1890, page 1064.



tracted leprosy from the husband, an examination showed that the woman was not a leper at all, while in four out of five cases in which asylum attendants were supposed to have been infected by the inmates, the former were shown to have been lepers before they entered on their duties.

Thus the Calicut case alone remains. But the statement of the patient himself and that of the old inmate of the asylum are considerably at variance as to dates. And it must always be remembered that in a country like India, where leprosy is endemic, it is quite possible that the patient may have acquired the disease from some source other than the asylum: in other words, that the case may have been merely a coincidence.

TABLE II.

Table showing in how many Instances the Disease may be said to "have spread" from one Member of a Family to Another.

NAME OF ASYLUM, ETC.	No. of lepers.	No. of people living with them.	No. of such people affected.	REMARKS.
Agra	28	55	4	One case is doubtful.
Aligarh	5	19	...	
Almora	39	86	8	In one case the disease appeared sixteen years after the death of the leper.
Bangalore	4	15	2	One case doubtful.
Benares	10	34	...	
Bombay	60	105	8	
Burdwan	12	37	...	
Calcutta	40	67	2	
Calicut	13	43	...	

Table II—*continued.*

Table showing in how many Instances the Disease may be said to "have spread" from one Member of a Family to Another.

NAME OF ASYLUM, ETC.	No. of lepers.	No. of people living with them.	No. of such people affected.	REMARKS.
Cawnpore	8	20	...	
Conjeeveram	13	40	2	
Darjeeling	2	8	...	
Dehra Dun	44	116	11	Of these two became lepers many years after the death of the leper they came in contact with.
Delhi	5	18	...	
Fyzabad	7	20	...	
Gwalior	2	9	...	
Gya	16	46	1	
Hyderabad	7	12	...	
Jubbulpore	8	22	...	
Jummoo	1	3	...	
Kapurthalla	2	2	1	This case is very doubtful.
Lucknow	5	6	...	
Madras	60	156	17	Five cases doubtful.
Madura	5	21	...	
Mandalay	79	205	4	In one case the child acquired the disease at the same time as the father.
Moulmein	9	14	2	These cases are both doubtful.
Nagpur	67	166	3	
Naini and Allahabad	6	14	...	
Patiala	1	2	1	
Prome	20	62	4	One case doubtful.
Purulia	25	53	11	Ditto.
Rangoon	20	33	1	
Rawalpindi	9	19	...	
Sialkot	1	3	...	

Table II—*continued.*

Table showing in how many Instances the Disease may be said to "have spread" from one Member of a Family to Another.

Name of Asylum, etc.	No. of lepers.	No. of people living with them.	No. of such people affected.	Remarks.
Subathu	5	14	...	
Tanjore	7	16	2	These two cases are doubtful.
Tarn Taran	37	53	4	
Thayetmyo	24	51	7	Three cases are doubtful.
Trichinopoly	6	6	...	
Umballa	7	20	...	
Total	719	1,691	95	Diagnosis doubtful in seventeen cases.

In the construction of this table only those cases have been chosen where it was stated that the members of the respective families lived together. In many instances no special notice had been taken of this fact, and all these have been disregarded unless a case of leprosy occurred amongst the children. Now, no doubt, as in many of the cases left out on account of insufficient details there has been no separation or segregation of the leper, this table will over-rate the chances of a possible contagion rather than under-estimate it. It will be seen that about 5 per cent. of the persons living in daily intercourse with lepers under the same roof at one time or other developed the disease. A number of children have been left out of consideration because they died as infants or while very young. This has been done in order to give the argument in favour of contagion every possible advantage, for surely, where

contagion as such is discussed, age need hardly be considered. If a disease be contagious it should be so at any age. But if it is necessary to assume a long incubation period, as many writers do, which sometimes extends through infancy and childhood, until what is called the probable age of attack is reached, this is surely an unjustifiable extension of the term contagion, or a practical denial of the influence of contagion by a reduction to an absurdity. Again, in some cases entered in the table, the diagnosis of leprosy was extremely doubtful, and in one case the child contracted the disease at the same time as the father. Thus by allowing the validity of these cases a further advantage is allowed to the hypothesis of contagion. And yet in only 5 per cent. of the cases could a possible influence be assumed.

But the ratio obtained from this table of course cannot be taken to represent the actually existing chances of infection. For from among the total community coming into contact with lepers, only a few have been selected. Moreover all other causes, such as endemicity, or a *de novo* origin of the disease, have been altogether disregarded. However, assuming, for argument's sake, that all the above cases are genuine instances of contagion, and that the spread of leprosy depends on this alone, a little reflection will show that, selecting such a community as is implied in the tables, the disease would die out after a few generations. Has this not been entirely confirmed by the history of the Norwegian emigrants?

In the chapter on hereditary transmission and predisposition it was seen that the disease has a predilection for certain families. It was further shown that heredity cannot justly be considered the cause thereof. It may be asked, whether this fact be not explicable on the theory of the contagiousness of leprosy. From a casual

inspection of Tables IX, X, X*a*, and X*b* of Chapter IV, it might appear as though this were really so. Further consideration will prove, that even assuming the spread of leprosy within a family to be due to contagion, yet the danger of a general increase of the disease throughout the country on this account is exceedingly slight. Firstly, amongst all the families of which one member at least was a leper, in only from 5 to 8 per cent. at the most can the disease be said to have spread within the family. Secondly, this diffusion within the family is very slight. For as Table II of this chapter shows, of 1,691 persons who came into close contact with lepers, only 95, or about 5 per cent., became affected.

Comparing Table I of Chapter IV with the present table, there seems at first sight to be a great discrepancy. From the former it might appear that in 264 out of 2,371 families, or in 11·1 per cent., the disease had attacked more than one member, that is, spread more or less within the family; while according to the latter only 95 out of 1,691 individuals living with a leper in a family became affected. The difference in the figures is due to the fact that in the enquiry into an hereditary taint, the evidence afforded by the patient's immediate ancestors naturally was fully considered, as well as that afforded by his own children, while for the construction of Table II of this chapter, only the latter and the history of husbands or wives were taken into account. If the tables had been constructed on equal lines, no doubt many more instances of an assumed contagion might have been added. But, on the other hand, a far greater number of cases would have been found where such a possible infection could not be claimed. For there are many instances where a leper confessed to having lived with his parents and brothers and sisters without infecting them. Again, in a discussion of heredity it makes no difference whether the leprous ancestor was separated from his family or not. In Table I of Chapter IV, therefore, all the cases have been considered where the children had been removed from their parents, or developed the disease many years after their death. Finally, of the 264 lepers with an hereditary history, several were members of the same families, so that if the table is to be used as an argument for a spread by contagion within a family, evidently certain deductions will have to be made.

Making every allowance for these differences in the construction of the tables, it is evident that the discrepancy is only apparent.

Table I of Chapter IV, therefore, as it stands cannot be applied to prove or disprove contagion. It can only show, that taking all the families in which one individual at least is a leper, in considerably less than 11·1 per cent. another member of the same family becomes affected. To apply it to the subject under discussion, all instances in which contagion can be positively excluded must be omitted and each family taken as a whole. As the cases mentioned in Tables IX and X of the same chapter are comprised in Table I, no further comment is necessary regarding them. The deductions from Table X*a* are strong arguments in favour of the views expressed in this chapter, while the cases selected for the construction of Table X*b* are too special to permit any general conclusions being drawn from them.

The discrepancies are, therefore, only on the surface. To study the possible influence of contagion within a family, all the members of such family must be considered—wives and husbands as well as children—and it must be demanded that the individuals had actually exposed themselves to the risk of infection by intercourse with

lepers, that is,—separation and segregation must be absolutely excluded. All these conditions have been fulfilled in Table II of this chapter.

TABLE III.

To show in how many Cases a Supposed Contagion can be traced from Husband to Wife, or vice versâ.

Name of Asylum, etc.	No. of couples of which one individual was affected with leprosy for at least five years.	No. of instances in which disease spread from one to the other.	Remarks.
Agra	15	1 (1)	(1) Husband was a leper at time of marriage and died three years later. Fourteen years after his death the widow was attacked. Wife a leper sixteen years after death of husband.
Aligarh	4	...	
Almora	9	1	
Bangalore	10	2	
Benares	7	...	
Bombay	26	3(2)	(2) In one case the husband was a leper; twelve years after his death the widow was attacked by leprosy.
Burdwan	9	...	
Calcutta	24	...	
Calicut	11	...	
Cawnpore	4	...	
Conjeeveram	7	...	
Darjeeling	1	...	
Dehra Dun	9	1(3)	(3) Husband was a leper for four years and then died; two years later his widow was affected.
Delhi	5	...	
Fyzabad	6	...	
Gwalior	5	...	
Gya	8	1	
Hyderabad	6	...	
Jubbulpore	5	...	
Lucknow	2	...	

Table III—*continued.*

To show in how many Cases a Supposed Contagion can be traced from Husband to Wife, or vice versâ.

Name of Asylum, etc.	No. of couples of which one individual was affected with leprosy for at least five years.	No. of instances in which disease spread from one to the other.	Remarks.
Madras	25	1	
Madura	41	3(4)	(4) In two cases the diagnosis was very doubtful.
Mandalay	34	...	
Moulmein	2	...	
Nagpur	26	3(5)	(5) In one case husband and wife affected at the same time.
Naini and Allahabad	8	...	
Prome	11	...	
Purulia	13	4	
Rangoon	9	1	
Rawalpindi	1	...	
Sialkot	1	...	
Subathu	4	...	
Tanjore	3	...	
Tarn Taran	17	4(6)	(6) In one case wife acquired leprosy three years after husband's death, who had had leprosy for six years.
Thayetmyo	7	...	
Trichinopoly	5	...	
Umballa	1	...	
TOTAL	381	25	

It will be seen from this table that there were 381 couples of which one member became a leper and continued to live with the other for a certain period. Or, expressing it in other words, 381 healthy persons lived intimately with an equal number of individuals,

exposing themselves to all possible risk of contagion, and of this number only 25 subsequently became affected, or 6·5 per cent. Six of these cases may fairly be subtracted, so that of 381 people living with an equal number of lepers only 19, or 4·9 per cent., became diseased, accepting the doubtful cases, two in number, as trustworthy.

Only those cases have been chosen where couples had been married for at least five years, the average time being from seven to ten years. There can be no doubt that an enquiry into the condition of people married to lepers is the best mode of ascertaining how far contagion influences the diffusion of the disease. As Drs. Lewis and Cunningham said in their report[18]: "The means which most naturally suggests itself for enquiring into the theory that leprosy is a contagious disease is an examination of the history of all the married lepers, for were the result of this to show that the wives or husbands, as the case might be, of lepers suffer frequently from the disease this would be some evidence in favour of contagion." From the above table it will be seen, that even allowing a long incubation period, the ratio of people affected is too small to warrant a belief in contagion as a factor of much importance. If it be remembered that the married people lived in endemic areas, the possibility of a *de novo* development of the disease must undoubtedly exist.

In Table II the evidence afforded by whole families is given, and it is at once seen that these two tables correspond in every respect. However, do instances of a possible contagion exist amongst people who have been married to lepers for less than five years? There were only five such cases, two at Bombay, two at Madras, where each individual after having lived with a leper husband for

(18) Op. cit, page 56.

three years developed the disease, and one at Tanjore. Here two people had been married for sixteen years, and then the husband became a leper and was left by his wife, who, within six months, was said likewise to have been attacked by the disease. The woman could not be examined, and the diagnosis was highly questionable. But even allowing the latter to be correct, it would seem probable that this is not an instance of contagion, but that in these two people the disease was the effect of the same causes.

A table will now be added, giving the number of people who have been married to lepers, and who have lived with them for less than five years, for periods varying from six months to four years, three years being the average, since the manifestation of the disease, and stating in how many instances leprosy occurred.

TABLE IIIa.

To show in how many Instances Persons married to Lepers for a Period of less than Five Years subsequently became Lepers.

NAME OF ASYLUM, ETC.	No. of couples of which one individual was affected with leprosy for less than five years.	No. of instances of possible contagion.	REMARKS.
Agra	9	...	
Aligarh	1	...	
Almora	9	...	
Bangalore . . .	4	...	
Benares	2	...	
Bombay	27	2 (1)	(1) In one case a woman married a leper. He died two years later and four years afterwards the widow became a leper.
Burdwan	3	...	
Calcutta	5	...	

Table IIIa—*continued.*

To show in how many Instances Persons married to Lepers for a Period of less than Five Years subsequently became Lepers.

Name of Asylum, etc.	No. of couples of which one individual was affected with leprosy for less than five years.	No. of instances of possible contagion.	Remarks.
Calicut	4	...	
Cawnpore	1	...	
Conjeeveram	2	...	
Dehra Dun	12	..	
Delhi	1	...	
Fyzabad	1	...	
Gwalior	1	...	
Gya	5	...	
Hyderabad	2	...	
Jubbulpore	1	...	
Lucknow	1	...	
Madras	24	2	
Madura	16	...	
Mandalay	22	...	
Moulmein	7	...	
Nagpur	22	...	
Purulia	5	...	
Rangoon	15	...	
Rawalpindi	1	...	
Tanjore	1	1(?)	(?) Husband and wife acquired leprosy within six months of each other.
Tarn Taran	7	...	
Thayetmyo	9	...	
Trichinopoly	1	...	
Umballa	1	...	
Total	222	5	

It follows from this table that there were 222 couples of which one individual became a leper and continued to live with the other for a period of less than five years, and of the latter only five became tainted, or 2·2 per cent. The Tanjore case can perhaps hardly be included, so that the ratio would be reduced to ·9 per cent. It may be assumed that a certain number will at a future date become affected. But in any case the two Tables III and III*a* taken together are strong arguments against an amount of contagion worthy of serious practical consideration, as they plainly show that husbands or wives of lepers do not frequently suffer from the disease. The fact that in Table III a greater percentage of husbands or wives became affected may be explained in other ways than by mere prolonged contact with lepers. For it must not be forgotten that in almost every instance, at the time of marriage both individuals were healthy, and that subsequently one of them became affected, that is, all these people must presumably have lived under conditions favourable to the appearance of the disease. The further facts that they all have lived in endemic districts, and that leprosy is a highly chronic disease, make it less surprising to find a higher ratio of lepers in Table III.

TABLE IV.

Enumeration of Cases where Persons resided with Lepers for Longer or Shorter Periods, coming in constant and close Contact with them, without being attacked by the Disease.

These may be classified as—

(1) Hospital or asylum officials.

(2) Persons detained in asylums for some time on account of mistaken diagnosis, or voluntary inmates.

TABLE IVa.

Evidence afforded by Asylum Officials.

NAME OF ASYLUM.	Capacity of Official.	Term of Office.	Condition.	REMARKS.
Agra =5 Officials.	(1) Hospital Assistant	14 years	Healthy	Does not live at asylum but visits it twice or thrice a day.
	(2) Cook	30 ,,	,, died.	Did not live at asylum, but cooked there all day.
	(3) Water-carrier	15 ,,	Healthy	
	(4) Sweeper	15 ,,	,,	Lives at asylum with wife and family.
	(5) Six Dressers	1 year	,,	These men had to rub lepers with gurjun oil, and did so for one year. They left two years ago, and are quite healthy.
Almora =3 Officials.	(1) Resident Superintendent	20 years	Healthy	Aged forty-two years, is married and has had eight children, varying in age from eighteen to one and a half year. Whole family live in asylum compound, and are healthy.
	(2) Asylum Assistant	25 ,,	,,	
	(3) Asylum Assistant	17 ,,	,,	
Calcutta =10 Officials.	(1) Washerman	20 years	Healthy	Have to wash clothes of the inmates of the asylum.
	(2) ,,	14 ,,	,,	
	(3) Porter	7 ,,	,,	All the servants at the asylum have to change their clothes once a day, and the dressers twice. All have to take a daily bath.
	(4) Dresser	15 ,,	,,	
	(5) ,,	9 ,,	,,	
	(6) ,,	1¾ ,,	,,	
	(7) Sweeper	7 ,,	,,	
	(8) ,,	4 ,,	,,	
	(9) ,,	4 ,,	,,	
	(10) Water-carrier	17 ,,	,,	
Calicut =3 Officials.	(1) Cook	30 years	Healthy	
	(2) Ward-cooly	12 ,,	,,	
	(3) Sweeper	23 ,,	Leper for two years	His cousin is also a leper.

Table IVa—*continued.*

Evidence afforded by Asylum Officials.

NAME OF ASYLUM.	Capacity of Official.	Term of Office.	Condition.	REMARKS.
Dehra Dun =5 Officials.	(1) Keeper	12 years	Healthy	All live on the premises of the asylum.
	(2) Sweeper	,,	,,	
	(3) ,,	,,	,,	
	(4) ,,	,,	,,	
	(5) Gardener	6 years	,,	
Dharmsala =4 Officials.	(1) Compounder	13 years	Healthy	Lives on the premises in a detached bungalow with wife and family. All healthy. Cf. note about gardeners under Tarn Taran.
	(2) Gardener	1 year	,,	
	(3) Keeper	3 years	,,	
	(4) Sweeper	1 year	,,	
Madras =9 Officials.	(1) Dresser	13 years	Healthy	
	(2) ,,	12 ,,	,,	
	(3) ,,	10 ,,	,,	
	(4) ,,	9 ,,	,,	
	(5) ,,	3 ,,	,,	
	(6) ,,	3 ,,	,,	
	(7) Tailor	12 ,,	,,	Has to mend the clothes of the lepers (over 200 in number).
	(8) Washerman	8 ,,	,,	Has to wash the clothes of the lepers.
	(9) Cook	15 ,,	Leper	Was treated for leprosy before he joined the asylum (cf. Table I).
Naini =1 Official.	(1) Attendant	Many years.	Healthy	Lives at the colony.
Rawalpindi =3 Officials.	(1) Dresser	25 years	Healthy	
	(2) Messenger	13½ ,,	Doubtful	Fingers and face look a little suspicious, but there is no anæsthesia.
	(3) Cook	25 ,,	Healthy	His mother was a leper in the asylum. He was brought up there, and afterwards engaged as cook.

Table IVa—*concluded.*

Evidence afforded by Asylum Officials.

Name of Asylum.	Capacity of Official.	Term of Office.	Condition.	Remarks.
Sialkot =5 Officials.	(1) Compounder	22 years	Healthy	The former water-carrier left after being at asylum fourteen years. He is still alive and healthy.
	(2) Water-carrier	12 ,,	,,	
	(3) Sweeper	2 ,,	,,	
	(4) Washerman	2 ,,	,,	
	(5) Gardener	2 ,,	,,	
Subathu =5 Officials.	(1) Messenger	12 years	Healthy	All these officials live on the premises.
	(2) Carpenter	15 ,,	,,	
	(3) Compounder	8 ,,	,,	
	(4) ,,	3 ,,	,,	
	(5) Missionary	4 ,,	,,	
Tarn Taran =11 Officials.	(1) Keeper	3 years	Healthy.	Lives at asylum with his wife and family.
	(2) Gardener	13 ,,	,,	They, like all the servants, live on the premises. They use the excreta of the lepers for gardening purposes. Their families also reside here.
	(3) ,,	6 ,,	,,	
	(4) Water-carrier	4 ,,	,,	
	(5) ,,	1 ,,	,,	
	(6) ,,	½ ,,	,,	
	(7) Sweeper	7 ,,	,,	They clean out the tanks where the lepers bathe, and while doing so are in the habit of having a bath themselves.
	(8) ,,	6 ,,	,,	
	(9) ,,	5 ,,	,,	
	(10) ,,	1 ,,	,,	
	(11) Compounder	2 ,,	,,	

From this table it will be seen that of sixty-nine asylum officials only three were affected with leprosy. Of these one had the disease before he entered the service (Madras), and a second case is doubtful (Rawalpindi), so that actually only one out of sixty-nine had become tainted, or 1·5 per cent. Including the doubtful case a possible contagion can be traced in only 3 per cent., a fact which, even on the assumption that some of the attendants may subsequently develop leprosy, nevertheless gives little support to the view that leprosy is spread by contagion.

TABLE IVb.

Enumeration of Instances where Persons other than Officials resided in the Asylums with Lepers.

(1) *Almora.*—In the asylum there were eleven people, blind, lame, and imbecile. Of these, two inmates, aged forty and forty-five years, respectively, had been there for twenty-eight years. Two, aged sixty and forty, had lived there for eight years, and another, aged forty, for seventeen years. The other six had resided at the asylum severally for eleven, five, three, three, two, and one year. All these were examined by the Commissioners and found to be free from leprosy.

(2) *Calcutta.*—Here there were two cases.

(*a*) A Native Christian, seventy years old, who has been in the asylum twenty-five years, lives in a ward with lepers. He eats and drinks with them and often consumes what they have left, or what they give him. He drinks out of the same vessels and smokes the same hookah. He was found to be suffering from multiple molluscum fibrosum, but otherwise quite healthy. He will not leave the asylum, as he finds it comfortable.

(*b*) A woman, who suffers from hysteria and eczema, has been in the asylum for twelve to thirteen years in close contact with four leper inmates. She is quite free from leprosy.

(3) *Lucknow.*—(*a*) A man and his mistress were admitted on July 7th, 1876. He was a leper, she was healthy and lived in the poor-house with him and the other inmates until June 9th, 1881, when he died. Their child is now twelve years old. He and his mother remained in the poor-house with the lepers, so that she has been there fifteen years and—

(*b*) her boy, twelve years. Both are quite healthy.

(*c*) A woman was admitted July 5th, 1878, under the supposition that she suffered from leprosy. She married a leper in the poor-house, but had no issue. A son of a former marriage, sixteen years of age, came to Lucknow and has remained there with her, so that she and—

(*d*) her son have lived amongst lepers for thirteen years without becoming tainted by the disease.

(4) *Madras.*—A boy, an Eurasian, seventeen years of age, had been in the leper asylum for two years. He was admitted on account of a wrong diagnosis and was very reluctant to leave. He was quite healthy.

(5) *Subathu.*—(*a*) A man, who up to the present time is free from the disease, has lived at the asylum for seven years, sharing a room with a leper, and eating and drinking with him.

(*b*) A woman, whose husband was a leper and died at Subathu, has lived there for eight to nine years. She is quite healthy and has remained at the asylum to help the female lepers.

(*c*) Another woman was admitted from motives of charity, and has lived among the lepers for ten years, being at present free from the disease.

(*d*) A third woman has voluntarily lived among the lepers for three to four years, and has remained healthy.

(6) *Tarn Taran.*—(*a*) A woman married a leper at the asylum and lived there for ten years. They had two children. She and —

(*b* and *c*) her two children are healthy.

(*d*) A drover's wife, after her husband's death, had frequent intercourse with the lepers of the asylum for the last twelve years, giving birth to three children. At present all these are over eight years of age. She and —

(*e*, *f* and *g*) her children, have resided there and are absolutely healthy.

(*h* and *j*) Two children were seen, ten and twelve years of age, respectively, who were born at the asylum of leper parents, and being orphans, have been maintained there. They showed no signs of the disease.

(*k*) A woman, twenty-five years of age, was examined. Both her parents were leper inmates of the asylum when she was born. Her father died when she was two years old, and her mother when she was twelve years of age. She was then taken care of by a female leper in the asylum and has married a leper there. She had three children now, all over four years of age. She and—

(*l*, *m* and *n*) her children are perfectly healthy.

These cases will now be given in a tabular form.

Table IVb—*continued.*

Name of Asylum.	Condition on Admission.	Present Age.	Term of residence at the Asylum.	Present Condition.
Almora = 11 Cases	All these eleven people were either blind, lame, or imbecile.	40 years	28 years	Healthy.
		45 ,,	28 ,,	,,
		40 ,,	17 ,,	,,
		60 ,,	11 ,,	,,
		60 ,,	8 ,,	,,
		40 ,,	8 ,,	,,
		30 ,,	5 ,,	,,
		55 ,,	3 ,,	,,
		20 ,,	3 ,,	,,
		20 ,,	2 ,,	,,
		20 ,,	1 ,,	,,
Calcutta = 2 Cases	(1) Molluscum fibrosum.	70 years	25 years	Healthy.
	(2) Hysteria	About 50 yrs.	12-13 ,,	,,
Lucknow = 4 Cases	(1) Healthy woman	About 30 yrs.	15 years	Healthy.
	(2) Son of (1)	12 years	12 ,,	,,
	(3) Healthy woman	About 30 yrs.	13 ,,	,,
	(4) Son of (3)	16 years	13 ,,	,,
Madras = 1 Case	Eurasian boy, free from leprosy.	17 years	2 years	Healthy.

Table IVb—*concluded.*

NAME OF ASYLUM.	Condition on Admission.	Present Age.	Term of residence at the Asylum.	Present Condition.
Subathu =4 Cases .	(1) Healthy man .	Adult. .	4 years	Healthy.
	(2) „ woman .	„ .	8-9 „	„
	(3) „ „ .	„ .	10 „	„
	(4) „ „ .	„ .	3-4 „	„
Tarn Taran =13 Cases .	(1) Healthy woman .	Adult .	10 years .	Healthy.
	(2) (3) Her two children.	Under 10	Since birth .	„
		„	„ .	„
	(4) Healthy woman .	About 40 yrs.	12 years .	„
	(5) (6) (7) Her three children (4).	All over 8 years.	Since birth .	„
			„ .	„
			„ .	„
	(8) Healthy child .	12 years .	12 years .	„
	(9) „ „	10 „ .	10 „ .	„
	(10) Healthy woman	25 „ .	25 „ .	„
	(11) (12) (13) Her three children.	All over 4 years.	Since birth .	„
			„ .	„
		...	„ .	„

In this table are thirty-five cases, all well authenticated and personally examined by the Commission, where people were exposed to the dangers of contagion, and yet escaped the disease. Adding these thirty-five cases to the previous sixty-nine cases of Table IV*a*, it will be seen that of one hundred and four persons, living under conditions highly favourable for contracting a communicable disease

only one, or possibly two, have become lepers; that is, 1 or 2 per cent.

TABLE V.

Evidence in Favour of Contagion afforded by Persons Eating and Drinking with Lepers.

Asylum or Centre.	No. of Lepers.	No. of persons eating and drinking with them.	How many of these are affected.	REMARKS.
Agra . . .	5	11	3	In one case two brothers used to eat and drink together: one of them became a leper, and fifteen years later, the other. In another case a leper father and his two daughters used to eat together. He died, and some years later the two daughters became diseased at the same time.
Aligarh . .	1	1	1	Child used to eat with a leper father, and eight years after his death also became tainted.
Benares . .	4	4	1	Child used to eat with leper mother, and five years after her death became also a leper.
Calcutta . .	19	43	3	In one instance two brothers used to eat together, and within a year both were lepers. In another, a child had eaten with its leper father, and was twelve years old when the latter died. Sixteen years after his death the child became a leper.
Delhi . . .	2	5		
Gwalior . . .	1	1		
Gya . . .	7	7	...	The wives were accustomed to eat with their leper husbands, but it was distinctly stated that they washed the plates after the husbands had used them, and before they employed these for their own meal.

Table V—*continued.*

Evidence in Favour of Contagion afforded by Persons Eating and Drinking with Lepers.

Asylum or Centre.	No. of Lepers.	No. of persons eating and drinking with them.	How many of these are affected.	Remarks.
Mandalay .	24	84	5	In one case a child used to eat from the same vessels as her mother, and thirteen years after the latter's death she also became affected.
Prome . . .	13	40	3	In one instance a family of nine used to eat from the same dishes. One of them became a leper and died a year later. They continued after this to eat from the same vessels, and two years later another member of the family was attacked. The others remained healthy.
Thayetmyo . .	10	16	5	In one case the disease appeared in two people at the same time, and, therefore, one cannot be said to have infected the other. In another case the diagnosis of leprosy in the supposed infected person was extremely doubtful, and in a third instance two persons eating together developed the disease within a year of each other.
Total .	86	212	21	

For the construction of this table, only the information obtained from asylums and districts, in which the Commissioners paid special attention to this point, has been considered. In all the cases, with the exception of those in Gya, families used to eat at the same time, and out of the same vessels. Since at Gya the wives previously washed the dishes from which their leper husbands had eaten, these seven cases must be excluded in calculating the possible influence of contagion. Hence the figures show that of two hundred and five persons who exposed themselves by eating and drinking with lepers to the danger of infection, only twenty-one became affected. Moreover, in the one instance where two individuals who had constantly eaten together from the same vessels became diseased at the time, there can be no question of contagion. Again, cases of persons who had not come into contact with lepers for a long period cannot be quoted in favour of contagion. Five such instances are to be found in Table V, one from Agra, one from Aligarh, one from Benares, one from Calcutta, and one from Mandalay. The assumption of so long an incubation period as five years or more means practically reducing the influence of contagion to an absurdity. Six cases, therefore, must be subtracted from the twenty-one instances of possible contagion through eating and drinking with lepers. Besides, in one case (Thayetmyo), the diagnosis was extremely doubtful, and in two cases (Calcutta and Thayetmyo), the disease may be said to have commenced in the two persons at the same time, as they were affected within a year of each other. It is also very questionable whether one of the cases recorded from Prome can be cited with all fairness as an example of possible contagion from these causes. Allowing, however, that these cases are free from objection, the figures show that of two hundred and five persons who exposed themselves to the risk of contagion by eating and drinking with lepers, fifteen might be claimed as exemplifying an assumed influence of contagion, or 7·3 per cent. These figures cannot be said to favour the hypothesis of an infection through eating and drinking with lepers, especially when it is remembered that 7·3 per cent. is probably considerably above the actual ratio, which perhaps is more nearly represented by 5 per cent., even assuming that the spread of the disease among these people depends on contagion only.

TABLE VI.

Observations on Lepers vaccinated at the Almora Asylum.

Eighty-six persons were vaccinated, and of these 40 successfully.
Of the 40 successful cases, 34 were anæsthetic lepers.
5 were mixed cases.
1 was tubercular.

Observations on Lepers vaccinated at the Almora Asylum.

In 31 patients the vesicle was normal.
2 „ „ „ „ purulent.
2 „ „ „ „ purulent and mixed with blood.
1 „ „ „ „ normal on one arm and purulent on the other.
1 „ „ „ „ normal but mixed with blood on puncturing it.
1 „ „ „ „ immature and mixed with blood on puncturing it.
1 „ „ „ „ immature.
1 „ „ „ „ immature and the crust removed for examination.

Ninety-three cover-glasses were prepared and stained for bacilli, and specimens were also taken later from the crusts of two vesicles which had been normal.

Condition of site of vaccination—In 14 patients the skin was normal.
„ 12 „ sensation was diminished.
„ 13 „ there was an anæsthetic patch.
„ 1 patient the skin was tuberculated.

Results:—In no case were leprosy bacilli undoubtedly found.

In two specimens prepared from a vesicle over a tuberculated ear there were one or two badly stained bacilli.

In four specimens from an anæsthetic patch there were some rods which stained with fuchsin and looked suspicious, but none of these were certain.

TABLE VII.

I.—Observations on Earth at the Almora Leper Asylum.

SOURCE OF EARTH.	No. of cover-glasses examined.	Number of Leprosy Bacilli found.
1. Path in front of office . .	25	3 on 1 cover-glass. 2 „ 1 „ „ 1 „ 1 „ „ 1 „ 1 „ „
2. Path down hill to leper quarters .	25	1 „ 1 „ „ 1 „ 1 „ „
3. Path under bank where lepers sit	25	1 „ 1 „ „
4. Bank where lepers sit . .	25	No bacilli. [One suspicious.]
TOTAL .	100	10 bacilli in 100 observations.

II.—Examination of Dust from Leper Huts.

SOURCE OF DUST.	No. of cover-glasses examined.	RESULT.
1. Room inhabited by tubercular leper with ulcers on both feet	100	Negative.
2. Ditto ditto . .	100	„
3. Ditto with ulcer on right foot .	100	„
4. Three rooms inhabited by anæsthetic lepers with extensive ulcerations.	50	„
5.	50	
6.	50	

CHAPTER VI.

Sanitation, Diet, and Diseases in relation to Leprosy.

Sanitation.

IT has been seen that heredity and contagion are altogether insufficient to explain the spread of leprosy, and other ætiological factors must be sought for. In diseases like leprosy and tuberculosis, it is always difficult to find the exciting cause. For, with the recognition that a specific bacillus enters the body, the matter is but little advanced. The enquirer must always ask, why a widely diffused microbe, such as that of tuberculosis or leprosy, should cause a particular disease in some people and not in others. What is it that establishes the necessary specific predisposition?

This question is as obscure for leprosy as it is for tuberculosis. In this chapter the more important causes, supposed to bring about such specific predisposition, will be discussed.

When a disease, as is the case with leprosy, is so generally distributed over a vast country, attention must be directed to the general life and hygienic surroundings of the people. Does defective sanitation cause a specific predisposition to leprosy?

Since all classes of the community and all races appear to be subject to leprosy, remarks upon the sanitary environment of the inhabitants of India must be necessarily general in character. In the cities and larger centres of population considerable progress has of late years been made in sanitary improvement, though very much yet remains to be done. In the smaller towns and villages, however, little has been accomplished, or, indeed, is practicable in the present

state of native opinion. Fortunately the nature of the employment of the great mass of the population necessitates an outdoor life, and the free air-flushing of the village site, and the rapid desiccation of the objectionable matters thrown upon it, go far to reduce the consequences of habitual neglect of sanitary principles.

These matters will now be dealt with in detail. As regards conservancy, it is found that the cities and larger towns are provided with suitable latrines, which are in charge of an adequate conservancy staff; and which are largely used by the people. The excreta from these latrines, and also the general refuse and rubbish of the town, are regularly removed to a distance, and there buried, burnt, or otherwise suitably disposed of. Although differences exist in various localities as regards the efficiency of these arrangements, they may be described as upon the whole fairly good. But no large centre of population is free from many nuisances. Ruined huts and waste pieces of land are frequently used for purposes of nature, cesspools exist in many courtyards and in immediate proximity to wells, excavations full of fetid water are frequently observed, and other sources of danger to the public health are only too common.

In the smaller towns and villages little or no attempt at organising conservancy arrangements is made. The villager deposits the refuse and sweepings of the dwelling in the immediate vicinity of the house or hut, in some cases from indifference, in others to avoid theft of such matters before their employment for agricultural operations. For purposes of nature he generally resorts to a field in the neighbourhood, or to the banks of a stream or pond. Refuse water is allowed usually to flow from his hut into the adjacent road. It should be noted, however, that despite the frequently

objectionable nature of its surroundings, the interior of the average village dwelling is usually fairly clean.

Most cities and towns have a more or less satisfactory system of drainage as regards the main thoroughfares, but the climatic conditions of the country, and the fact that so many months of the year are almost rainless, render the flushing of these water-ways a matter of extreme difficulty, and often an impossibility. Where the configuration of the land permits, attempts are often made to utilize streams and other sources of water for the purpose, but a glance at a physical map of India will show how in the extensive flat plains of the country no such procedure is possible. Again, although much has been, and is being, done to ensure efficient drainage of inhabited sites, such efforts have up to the present time been more especially directed to the carrying off of water from the thoroughfares and streets. Small drains, it is true, connect the dwellings with the larger channels, but inasmuch as the inhabitants bathe at the public wells and tanks, and practically only employ such water in their houses as is required for drinking and cooking, little or no flow in these connecting drains is usually observable. Few towns as yet in India possess a water-supply laid on in pipes to the houses.

In the villages and smaller centres of population water finds its way more or less completely from the site through small cuttings in the soil, or through a channel created naturally by the heavy rainfall during the monsoon.

As regards water-supply, some of the larger towns and cities, as Calcutta, Bombay, Madras, Agra, and certain others, are provided with a good supply laid on in pipes. But the great majority of places have no such arrangements, and here the water is taken

from wells, rivers, springs, tanks, or lakes. From both wells and rivers the water is usually more or less impure, though superior to that from lakes and tanks. Spring water when obtainable is, as might be expected, usually good. Attempts to sink Artesian wells have succeeded in a few localities, but have more generally failed. Local authorities in the larger centres have of late years succeeded in improving the supply by such measures as keeping in repair the wells that hold good water and closing those of which the contents are impure, by remodelling tanks and preventing their pollution, but in the villages little at present can be done in this direction.

Habitations necessarily vary in size with the wealth and position of the owner or tenant. They are more generally built of bricks and mud in the plains, of stone in the hill tracts, and of bamboo and wood in Burma and certain parts of India. In towns and cities they are usually placed in close proximity to one another, and thereby efficient ventilation and air-flushing of the site are interfered with. Overcrowding of these dwellings is the rule, and in the hill tracts is often excessive. The residents, however, live largely in the open air, and doors and windows are either seldom closed, or are so carelessly constructed that air freely enters the house or hut.

In connection with habitations, it should be mentioned that not only in India but also in other parts of the world the mosquito has been much feared. As Dr. Arning says:[1] "Ashmead seems to fear much the bite of the mosquito, and I quite agree with him that the idea of transmission of leprosy through the sting of an insect is a very plausible one." Dr. Arning himself "frequently examined mosquitoes bacterioscopically, which were found inside the mosquito nets of beds containing cases of severe cutaneous leprosy. He

(1) Journal of the Leprosy Investigation Committee, No 2, February 1891, page 132.

caught the insects when they were quite full of the blood sucked from the patients. He never discovered any trace of leprosy bacilli, either in or upon them." Some members of the Commission examined flies and mosquitoes, but also with negative result, as will be seen from the Laboratory Report. Other considerations, however, make it appear extremely unlikely that a propagation of the disease should be due to these insects. It is hardly possible that the toxic principle of the mosquito should contain bacilli, even though they be present in the blood with which the insect is gorged. Again, cases of transmission from patients to hospital or asylum officials should be very common if this theory be true. Yet in Calcutta, where mosquitoes and flies abound, no instance of a transmission of the disease from a leper to a healthy individual has ever occurred at the asylum. All over the plains these insects are common, and nevertheless, with the exception of a single case in Calicut, no instance of infection could be found, even amongst the people who voluntarily resided with the lepers in the asylums. In fact it may be said that the whole chapter on contagion lends no support to the insect theory.

Personal cleanliness in India is very much a matter of climate. Where this is warm or mild all classes bathe frequently, Hindus more especially. In such localities linen and cotton clothes are worn and are regularly washed. But in colder latitudes far less attention is paid to regular ablutions, and where the climate demands the use of woollen clothing, such garments are seldom washed and become extremely foul. In the hill tracts the people may be said to rarely bathe, and are, as a rule, extremely dirty in their persons and habits. Most people possess shoes, but they are as often as not discarded, the owner walking for preference barefoot.

Scabies is very common all over India, and it has been asked whether or no this may contribute to the spread of the disease. This assumption may be refuted by the arguments used against the theory of propagation by insects, and it will not be necessary to enter into this question any further. It may be mentioned, however, that itch pustules were examined for the bacillus in several instances, but always with negative result.

It is quite impossible to assume that defective hygiene, whether general or personal, alone would originate leprosy. For, as will be seen from the above short review, the hygienic conditions vary considerably even in leprous areas, and the disease capriciously spares certain portions of a country where the condition of the population is identical with that of those attacked. In India the disease is found in the richest and poorest provinces. Defective hygiene, exposure to hardships and privations, filthy dwellings, and want of personal cleanliness cannot be said to predispose to leprosy more than to any other disease of like nature. They necessarily must aggravate and accelerate it, when it is once established; and for this reason, where they exist, require improvement.[2]

Diet.

The next question to be considered is the important one of diet. Since the earliest days in the history of leprosy the greatest influence in the ætiology of the disease has been attributed to defective or bad dietetic conditions. In turn almost every foodstuff has been accused. In ancient medical history the eating of certain kinds of fish, fresh or decayed, was considered of great importance

(2) *Cf.* R. Liveing: op. cit., pages 76 and 77.

and this opinion has persisted to the present day. Too much or too little animal or vegetable food has also been held responsible for the origin of the disease, or specific predisposition to the same. This influence of diet was naturally as keenly disputed by others.

That food should have a specific effect in the ætiology of a chronic disease is *à priori* quite within the bounds of possibility. No one who believes in the infective nature of leprosy would of course assume food to be a final exciting cause, for it is implied in the term "infective disease" that this must be a parasitic organism. It must be remembered, however, that when these various food-theories were propounded, the bacillus had not been discovered, and that, therefore, the views of many of the older authors would not be misrepresented, by stating that they claimed for diet only a direct effect in the establishment of a specific predisposition.

Certain forms of diet are capable of producing grave morbid conditions, as, *e.g.*, *Lathyrus sativus* and *Ergot*; others, on the other hand, cause more general changes in the body, and may thus possibly lead to those conditions which establish a specific predisposition.[3] In fact the experiments of H. Leo[4] lend much support to such a theory. "He administered phloridzin in small doses along with food to white mice for some days previous to inoculation, with the result that sugar became present in the tissues of the experimental animal. He found that the animals which are normally but little susceptible to glanders infection became highly susceptible to it, if previously dosed with phloridzin." In connection with this subject, it may also be mentioned, that Charrin and Roger[5] have

(3) R. Liveing: op. cit., page 83.

(4) H. Leo: Zeitschrift für Hygiene VII., 3; and Nineteenth Annual Report of the Local Government Board; Supplement, 1889, page 217.

(5) Charrin and Roger: La Semaine Médicale, 1890, 4; and Nineteenth Annual Report, etc., loc. cit.

shown "that ordinary normal rats, which, as is well known, are very little susceptible to anthrax, become susceptible to this disease in a marked degree if they are, when caged, made to work a treadmill so as to become thoroughly fatigued."

Food must necessarily modify the constitution of the tissues and may do so in such a way as to prepare them to respond at once to the introduction of microbes against which they would otherwise have proved refractory or insusceptible. Thus Monti[6] has shown that by a separate injection of a sufficient quantity of the chemical products of the Proteus vulgaris into rabbits, mice or other rodents, these animals could easily be made to succumb to an infection with cultures of the Diplococcus pneumoniæ, which through repeated subculture or age had lost their virulence, and which without the chemical products of the saprophyte were also entirely harmless. As Dr. E. Klein,[7] who confirmed and extended Monti's experiments, says, "insusceptibility of the tissues is, as is well known, considered by some authorities to be connected with, if not wholly dependent on, the chemical nature of the tissues; so that while the tissues are normal or in full vigour (if the phrase may be allowed for the purpose of illustrating my meaning), a particular microbe getting access to them fails to thrive—cannot, so to speak, overcome the resistance or inimical action offered by the tissues. This power of resistance of tissues can, however, be greatly reduced or even abolished by certain means, such as depression of their vitality either due to ptomaines and certain other chemical substances which have invaded them, or to nervous exhaustion, and the like."

(6) Nineteenth Annual Report, etc., loc. cit.

(7) E. Klein: Nineteenth Annual Report of the Local Government Board. Supplement 1889. page 217.

Where food is considered of ætiological importance in the production of an infective disease, there are, broadly speaking, two ways in which it may cause the latter. "First, by a direct introduction of the bacillus into the alimentary tract; secondly, by causing changes in the tissues capable of rousing into activity a bacillus already existing in them,"[8] or of offering a suitable soil to a bacillus subsequently introduced into them.

These points will be kept in view in the following discussion. At the present moment three substances have been specially singled out as having a causal relation to leprosy, *viz.*, fish, salt, and water. However, before discussing the effect of food in general, and of these three articles in particular, a few remarks on the diets of the Indian community must be made.

The inhabitants of India are almost entirely vegetarians, and the majority of people do not touch flesh from one year's end to another. Muhammadans, it is true, make flesh, other than that of the pig, a regular article of diet, but expense usually prevents its extensive consumption, and it is used generally in small quantities to supplement the main vegetable elements of their food. Hindus are vegetarians, but certain of the lower classes will readily eat meat, and Chamars will even consume the flesh of animals which have died of disease. Rajputs, especially in Central India, and wild forest tribes, eat what flesh may be killed in the chase, though some of pure Hindu blood refuse that of deer and pigs. Fowls and eggs are readily eaten in some parts of the country, but are regarded with abhorrence in others. Milk, curds, "ghee," or clarified butter are universally consumed.

(8) Journal of the Leprosy Investigation Committee, No. 1, August 1890, page 77.

As above stated, the great majority of inhabitants are vegetarians and live upon the crops raised in the country, the coarser grains being used by the poorer, and the finer by the richer, classes of the community. The subjoined table and remarks taken from the Report of the Famine Commission in 1880 will show the distribution and consumption of the various staples.

Province.	Percentage of food-growing area under		
	Wheat or Barley.	Millets.	Rice.
Punjab	54	41	5
North-Western Provinces	57	34	9
Bengal, Assam, and Burma	Not known (but principally rice).		
Central Provinces	27	39	34
Berar	17	82	1
Bombay	7	83	10
Madras	—	67	33
Mysore	—	84	16

"In the Punjab, North-West Provinces and Oudh, in Behar, in the northern part of the Central Provinces, and in Gujrat, the poorer classes live on the millets grown in the rains and on barley and gram; the richer classes eat principally wheat and rice. In Bengal proper and Orissa, and the eastern portion of Central India (and in Burma) rice is the principal food, the coarse early rice being mainly taken by the poor, the finer late rice by the rich. In the South, or Mahratta-speaking part of the Central Provinces, in Berar, in Bombay, the Deccan, and the northern part of Madras, the two large millets—jowar and bajra—form the principal food, the Brahmins usually living on imported rice and wheat. In Mysore the ordinary food is the small millet (ragi). In the southern part of Madras and

the western districts of Bombay rice is chiefly consumed, though there is a good deal of millet grown and eaten." All classes mix pulses with their food in order to obtain the necessary nitrogenous elements. Maize, though grown more or less everywhere, is not so largely consumed as might have been expected. Vegetables, such as spinach, pumpkins, carrots, potatoes, and useful wild herbs are largely used, and condiments, such as chillies, are taken with the meal to assist digestion. Fruit, such as that of the mhowa, mango, plantain, and cocoanut are eaten when obtainable, and oil and salt form part of every dietary.

Both sea and fresh-water fish are largely consumed wherever they can be caught. Dried fish is used all round the coast, especially in the Madras Presidency and Burma. In the latter country "nga-pi" or dried fish, more or less in a state of decomposition, is almost universally eaten, but in small quantities and more as a condiment than as a food.

It would be a mistake to suppose that the consumption of fish in India is in any degree confined to the coast or the vicinity of large rivers. Nearly every tank, pond, lake, or rivulet holds species which are caught and eaten. In many parts of India this forms a portion of the dietary of even the higher castes.

Food in India is usually eaten out of metal or earthen vessels, or platters made of dried leaves, the consumer sitting on the ground, or upon a mat, and using the fingers in place of knives and forks. Among Hindus it is customary for the males to eat before the females. Flesh is roasted, stewed, or boiled, pulses are usually boiled, and grain is either parched, or far more frequently ground, and the flour made into unleavened cakes known as "chupatties." Rice is boiled and either eaten alone or in the form of curries. As already

stated, cooked vegetables, salt, oil, and condiments form portions of almost every dietary. Sugar, sweetmeats and fruits are also largely consumed.

Of all articles of diet none has been held more responsible for the causation of leprosy than fish. This view has of late years gained considerable importance through the weight of the authority of Mr. Jonathan Hutchinson who stated the fish hypothesis with great force at the Tenth International Medical Congress at Berlin.[9] As Virchow and Dr. Liveing have pointed out, the theory is very old and has reappeared from time to time. Virchow's views on the subject have not infrequently been misrepresented, and it may, therefore, not be out of place here to quote his own words:[10] "The more general use of bad fish very (ungewöhnlich) frequently coincides with endemic leprosy. This statement, however, is subject to exceptions, but then, as a rule, another noxious dietetic article is accused, and comparative observations might be made as to whether or no the same deleterious substance exists in fish and these other articles of food." It will not be necessary to say more about the history of the fish hypothesis, as this has been done so concisely by Dr. Liveing in his Goulstonian lectures. One passage, however, of special interest with regard to leprosy in India is worthy of notice. Dr. Liveing says[11]: "The combination, however, of milk and fish seems to have been considered especially favourable to the disease. Bernhard Gordon says: 'Comedere lac et pisces in eadem mensa inducit Lepram.' And it is not a little remarkable that the same opinion obtains in the

(9) Journal of the Leprosy Investigation Committee, No. 1, August 1890, pages 77-87.
(10) R. Virchow: op. cit., page 507.
(11) R. Liveing: op. cit., page 33 and footnote, page 34.

present day in India." There is no doubt that in certain parts of the empire this opinion is very prevalent, especially in Kashmir and the hill districts. Lepers in India also frequently assert that after a fish meal their state is exacerbated.

Coming now to the discussion of the fish hypothesis and its application to Indian leprosy, it will be best to take it in its two chief parts. Mr. Jonathan Hutchinson says[12]: "it is possible that fish may cause the disease in one of several ways. First, it may be by the direct introduction of the bacillus into the stomach; secondly, it may be that some element in fish-food rouses into activity a bacillus already existing in the tissues."

Taking the first point, it should be possible to find the bacillus not infrequently in fish caught in endemic areas. *A priori*, judging from the similarity between leprosy and tuberculosis, it would not seem impossible that the Bacillus lepræ is capable of growing in a cold-blooded animal. The Commission paid special attention to this point, and some members examined a large number of fish, fresh and dried, or prepared as "nga-pi," but with absolutely negative result. Dr. Arning, who has studied this part of the question most carefully, has also never been able to find any leprosy bacilli in fish. The value of such investigations is necessarily that of negative evidence, but until positive cases have been shown, is worthy of all consideration.

The fish hypothesis premises that all lepers at one time or another have eaten fish in some form. Perhaps India is the most suitable country to investigate this matter, for here people of all castes and religions are thrown together. There is no reasonable doubt whatever that the majority of Brahmins never touch

(12) Loc. cit.

flesh or fish, and the same applies to many of the Banias or traders in certain districts, and almost without exception to the Jains. There are many Brahmins, however, settled on the shores of the Bay of Bengal, who habitually eat fish, and it is not at all rare to find Brahmins in hill districts and also in the plains who do not refuse fish. But in the plains they are much stricter in this respect. Jains, however, under no circumstances touch any animal food. They are, in fact, so particular, that it is a custom amongst many of them to fasten a piece of cloth to the upper lip to avoid inhaling small insects. They will not eat or drink in the dark, and always strain water through a cloth before drinking. Their priests have a broom with which they sweep the road before them to avoid the supposed guilt of killing insects by treading them under foot. This is a regular custom among the Bhabras in the Hoshiarpur District.[13] Now leprosy occurs amongst all these classes, and it seems indeed that the disease is impartially distributed among the fish-eating and non-fish-eating communities. It is only possible to talk of strong impressions, as accurate statistics and relative numbers cannot be drawn up, partly on account of the uncertainty of the present state of many castes and the changes which intercourse with Europeans has brought about. For instance, "among the lower ranks of Brahmins, great latitude is taken in regard to labour, food, etc., and their claim to the distinction of that caste is, in consequence, little recognised."[14] On the other hand, the conditions in which the better castes live are so different from those of the lower, which undoubtedly supply, not only absolutely, but also relatively, the greater number

(13) Gazetteer of the Hoshiarpur District, 1883-84, page 46.
(14) Lewis and Cunningham; op. cit., page 61, footnote.

of lepers, that comparisons would be unfair. The Banias or traders, of whatever caste or religion, for instance, are, generally speaking, a wealthy class, and the disease is acknowledged to be less common among prosperous people, though it does not spare them altogether. Now among the mixed class of Banias are many castes, the laws of which forbid the consumption of meat in any form. Many of the Banias in certain parts of India are attached to the Jain religion. In fact, it is a singular circumstance that many of them are devoted to this or some other modification of the Buddhist faith. When, therefore, a Bania or Brahmin leper denies ever having eaten fish, it is at least possible from what is known of the habits and customs of these communities. This is especially true of Agra and the North-West Provinces, so far as the Banias are concerned.[15] The Jains form one of the richest communities in India, yet the disease, though rarely, is found amongst them. Thus, at Hoshiarpur two of the Commissioners gathered, through the kindness of the Civil Surgeon Dr. Datta, reliable information concerning a leper from the Bhabra class, in whose case the cause of the disease could certainly not be ascribed to fish-eating.

It is not claimed that the fact of a man calling himself a Brahmin or a Bania is identical with saying that he has never eaten fish. It has already been said that many of the former consume animal food, and the latter include amongst their numbers many castes whose laws do not prohibit such a diet. This short exposition is meant to show that many of these people do never touch fish, and that, therefore, if a leper belonging to either class denies ever having done so, there is a fair probability of his statement being true.

([15]) M. A. Sherring: Hindu Tribes and Castes, 1872, pages 285-299.

Now Mr. Jonathan Hutchinson[16] objects and says: "In recording the denial on the part of leprosy patients that they have been fish consumers, caution must be exercised. Those who belong to castes which are forbidden to eat animal food will naturally be prone to deny that they have deviated from the rule. The temptation to eat fish as a condiment must, in the case of those restricted to an exclusively vegetable diet, be very great. It is, under those circumstances, precisely those who eat it most seldom (dwellers far inland, for instance) who are likely to receive it in its most dangerous state of decomposition. It must always be remembered that members of castes forbidden to take life often eagerly avail themselves of all opportunities as regards what has been found dead or killed by others. Thus the bare statement that leprosy prevails in classes who, from religious scruples, never eat animal food is usually of no real value. Careful and even sceptical inquiry must be made as to whether the individual lepers had really, in the case of preserved fish, invariably abstained."

There is of course much truth in these assertions of Mr. Hutchinson, and the above facts have only been mentioned to show that it is possible to find amongst lepers, individuals belonging to castes not allowed to touch animal food. Now Mr. Hutchinson is inclined to doubt the statement of lepers who profess to observe the rules which caste or religion enforce on them. Yet if a comparatively large number of lepers are found who state that they have never eaten fish, and these belong to castes or tribes of which it is known that their rules forbid the strict observer to touch meat or flesh in any form or shape, it seems improbable that all of them should have deceived the questioner or themselves.

([16]) Journal of the Leprosy Investigation Committee, No. 1, August 1890, page 79.

The Commission paid particular attention to this question, and found that one hundred and sixty-two individuals denied ever having touched fish (Table I). Many of these were allowed to partake of animal food, but denied ever having eaten fish, though in all cases leading questions were avoided as much as possible. To see in what percentage of cases an abstinence from this article of diet could be traced, every leper of a certain number of asylums was interrogated, with the result that out of 464 lepers 99, or 21·3 per cent., denied having ever partaken of fish.

TABLE I.

Number of Lepers who denied having ever eaten fish.

Asylum or Locality.	No. of persons who never ate fish.	Remarks.
Agra	7	3 Brahmins; 4 Banias.
Aligarh	3	1 Brahmin; 1 Bania.
Almora	30	9 Brahmins.
Bangalore	...	
Belgaum	...	
Benares	11	6 Brahmins.
Burdwan	...	
Calcutta	...	
Calicut	...	
Cawnpore	3	3 Brahmins.
Conjeeveram	...	
Darjeeling	...	
Dehra Dun	1	
Delhi	1	
Dharmsala	...	
Fyzabad	1	1 Brahmin.
Gwalior	3	1 Brahmin.
Gya	5	2 Brahmins.
Hyderabad	...	
Jubbulpore	1	1 Bania.
Jummoo	...	
Kapurthalla	4	

Table I—*continued.*

Number of Lepers who denied having ever eaten fish.

Asylum or Locality.	No. of persons who never ate fish.	Remarks
Lahore	...	
Lucknow	...	
Madras	1	
Madura	1	1 Brahmin.
Mandalay	2	2 Brahmins.
Meerut	...	
Moulmein	1	
Nagpur	3	3 Brahmins.
Naini and Allahabad	1	1 Brahmin.
Naini Tal	...	
Patiala	5	2 Brahmins; 1 Bania.
Peshawar	...	
Poona	3	1 Brahmin.
Prome	...	
Purulia	...	
Rangoon	...	
Rawalpindi	30	1 Brahmin.
Sialkot	15	
Subathu	5	5 Brahmins.
Tanjore	...	
Tarn Taran	8	
Thayetmyo	...	
Trichinopoly	...	
Umballa	14	1 Brahmin; 1 Bania.
Yerrowda Prison	3	1 Brahmin.
Total	162	44 Brahmins and 8 Banias.

The leper asylums of Almora and Dehra Dun are occupied by residents of the Himalayan hill tracts of Kumaun and Garhwal. The great elevations at which many of the villages in these tracts are situated, and their remoteness from rivers and streams, make fish a very rare article of food among the people. Some indeed

have never seen it. A careful enquiry was therefore made from the leper residents of the asylums quoted, and the result is given in the subjoined table:—

No. of Cases examined.	ATE FISH			
	Habitually.	Occasionally.	Very seldom.	Never.
200 . . .	39	57	58	46

It will be seen from this table that 23 per cent. of well-marked lepers had never tasted fish, while a very large number only used it now and then. This is quite in accordance with local opinion on the subject, not only in Kumaun and Garhwal, but also in Kashmir, where leprosy is common among the Gujars, a people in whose dietary this article of food seldom finds a place.

It is not within the limits of mathematical probability that the statement of all these people should have been intentionally or unintentionally incorrect, and the onus of proof, therefore, may fairly be considered to lie with them who discredit the statements of all patients alike.

There is thus, in the opinion of the Commission, no doubt that the consumption of fish is not the cause of leprosy. The fact that a fair number of cases of leprosy exists amongst people who have never touched such food argues sufficiently strongly against the exclusive fish hypothesis as above stated.

Salt also has been mentioned in connexion with leprosy, though by laymen rather than scientific writers. Mr. Conybeare during the early part of this year asked the Under-Secretary of State for India in the House of Commons "whether in the Presidencies of Madras and Bombay the price of salt has risen from 9 annas and 8 annas per maund in 1800 to R2-11 and R2-8 in 1890 respectively; whether he can state the facts, as to the increase or otherwise, in the price of salt for the other presidencies during the same period; whether in India the average consumption of salt per head for all purposes is only 10lb, while in the United Kingdom it is 72lb; whether it be a fact that leprosy has also increased during the same period; and whether the Government will direct the special attention of the Medical Commission on Leprosy in India to an investigation of the apparent connexion between the want of cheap salt and the spread of leprosy."

Accordingly special attention was paid to this subject, and, as far as possible, the most accurate information obtained. For the financial and statistical data regarding the price and consumption of salt the Commission are indebted to Mr. J. E. O'Conor, Assistant Secretary of the Government of India in the Department of Finance and Commerce, and they gladly here give expression to their obligation to him.

For the present argument, that is the relation between the want of cheap salt and the spread of leprosy, it will not be necessary to go further back than 1861. It is true that in the following provinces, *viz.*, Burma, Rajputana, Central India, Bombay, Sind and Baluchistan, Nizam's Territory, Madras, Mysore, and Coorg, in the decennial period of 1861—70 salt was cheapest, and has risen in price during the two succeeding decennial periods, and in some instances has done so to a considerable degree. On the other hand, in Assam, Bengal, North-Western Provinces, Oudh, Punjab, Central Provinces, and Berar, salt has steadily become cheaper.[17]

Thus, if there be any connexion between leprosy and salt, the best means are given for studying such connexion. For if the spread of the disease depend on want of salt it should have been

([17]) "Prices and Wages in India," compiled in the Statistical Branch of the Finance and Commerce Department of the Government of India: Eighth Issue; pages 86—93.

much more rapid throughout the first mentioned provinces. Before proceeding to the discussion of this point, a few words must be said as to the average consumption per head of salt in India.

The total quantity of salt passed into consumption during 1890 last year amounted to 2,801,800,000lb. There is, however, also a considerable quantity of salt made in Burma, of which no exact account is kept, but this is estimated at (at least) 41,143,000lb. There is, again, a considerable quantity manufactured from saltpetre, and some not inconsiderable quantities made in Native States. Adding all this to the quantity of which there is an account, and making allowance for quantities illicitly made and consumed, there cannot be less than 3,000 millions of pounds for the population of 286,697,000, or between 10 and 11lb per head: 11lb being perhaps the more accurate estimate.

Now, in comparing this with the 72lb per head in the United Kingdom, it must be remembered that in India this salt is used almost exclusively for personal consumption, very little being employed for cattle and economic or industrial purposes. Again, the majority of people being vegetarians salt is not in so great demand as in Great Britain and Ireland, nor, owing to the manner in which food is eaten, is there such a waste of salt in India.

Between 1870-71 and 1880-81 the consumption of salt increased by 19 per cent., and during the next ten years the increase was 21½ per cent. The increase of population in each decade is about 10 per cent., so that the ratio of increase in the consumption of salt was about twice as great as the ratio of increase in the population. And as the consumption is almost entirely human, it seems evident that if the people had enough salt twenty years ago, they have more than enough now.

In the interior of the Himalayas the price of salt is very high, not on account of the duty, for the salt consumed there is imported across the frontier duty-free, but as a result of the cost of transit. Excluding such exceptional tracts as Kumaun, Garhwal, and the Naga Hills, where the price of salt may be said to be about 7 seers to the rupee (about 2¼ annas per seer), the highest price of salt in India anywhere is 8 seers per rupee (or 2 annas per seer). The consumption per head being 11lb, the annual cost of salt per head is equal to 11 annas, less than one anna (exactly eleven pie) per month. The average price may, however, be taken to be about 10 seers to the rupee, and the annual cost per head at this rate is under 9 annas, or about 9 pie per month. It may be assumed that the highest cost per head is one anna monthly, which is less than one penny at the present rate of exchange. This is an extreme and exceptional price, for salt ranges at about 8 seers to the rupee in comparatively but few places.

Now while discussing the assumed connexion between salt and leprosy, it is necessary to enquire whether the price of this article prevents the native from procuring the amount of salt required to keep his body in proper health. It is difficult to say exactly how much salt a working man actually requires. It is certain "that the various saline matters are essential to health, that when they are not present in proper proportion nutrition is affected, as is shown by certain forms of scurvy: the peculiar dependence of proteid qualities on the presence of salt is proven, but beyond this very little is known."[18] Klein and Verson also have shown that a total abstinence from salt causes an appreciable loss of weight, that is, disturbances in the animal metabolism.

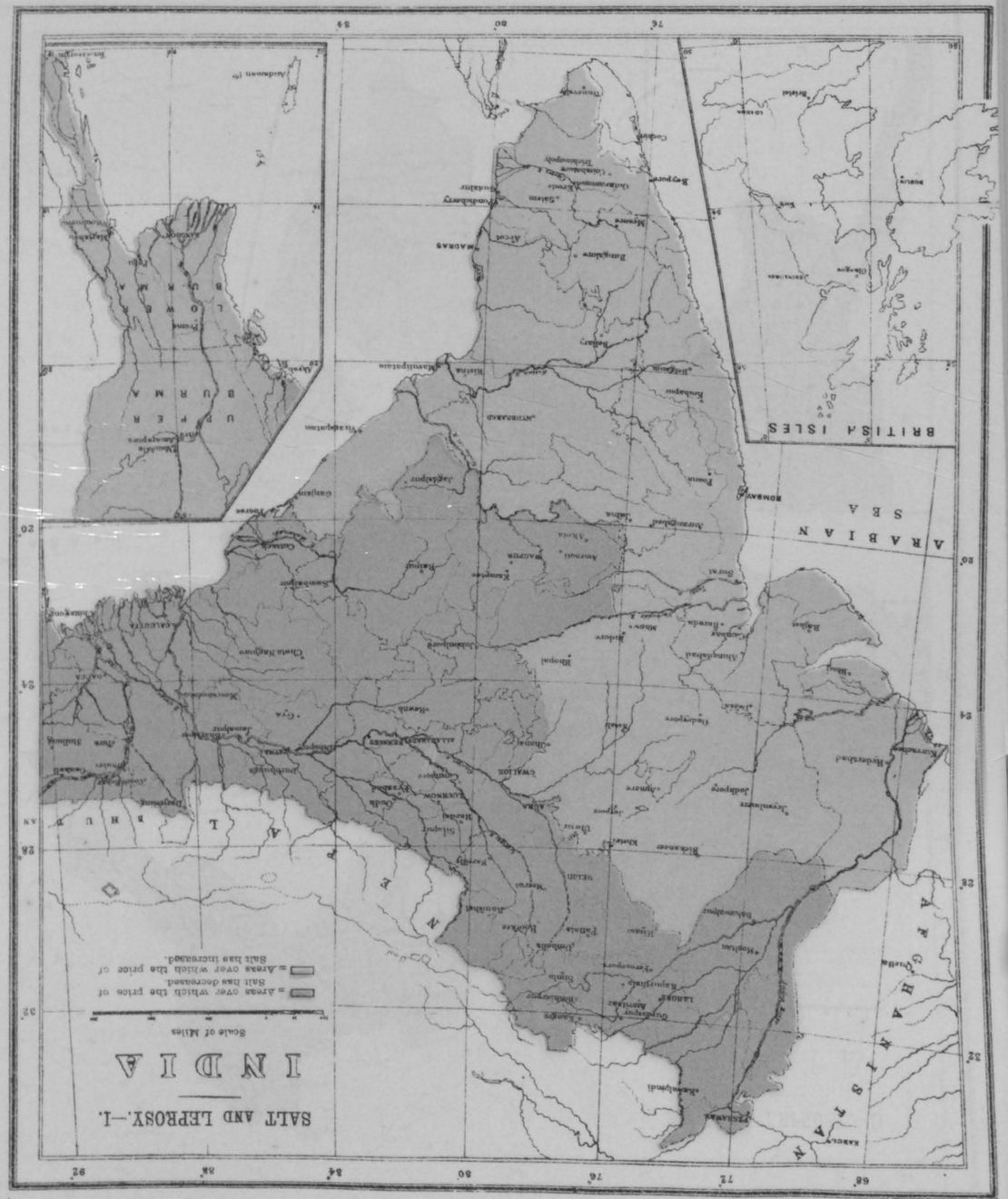
(18) M. Foster: A Text-book of Physiology: Third Edition, 1879, page 420.

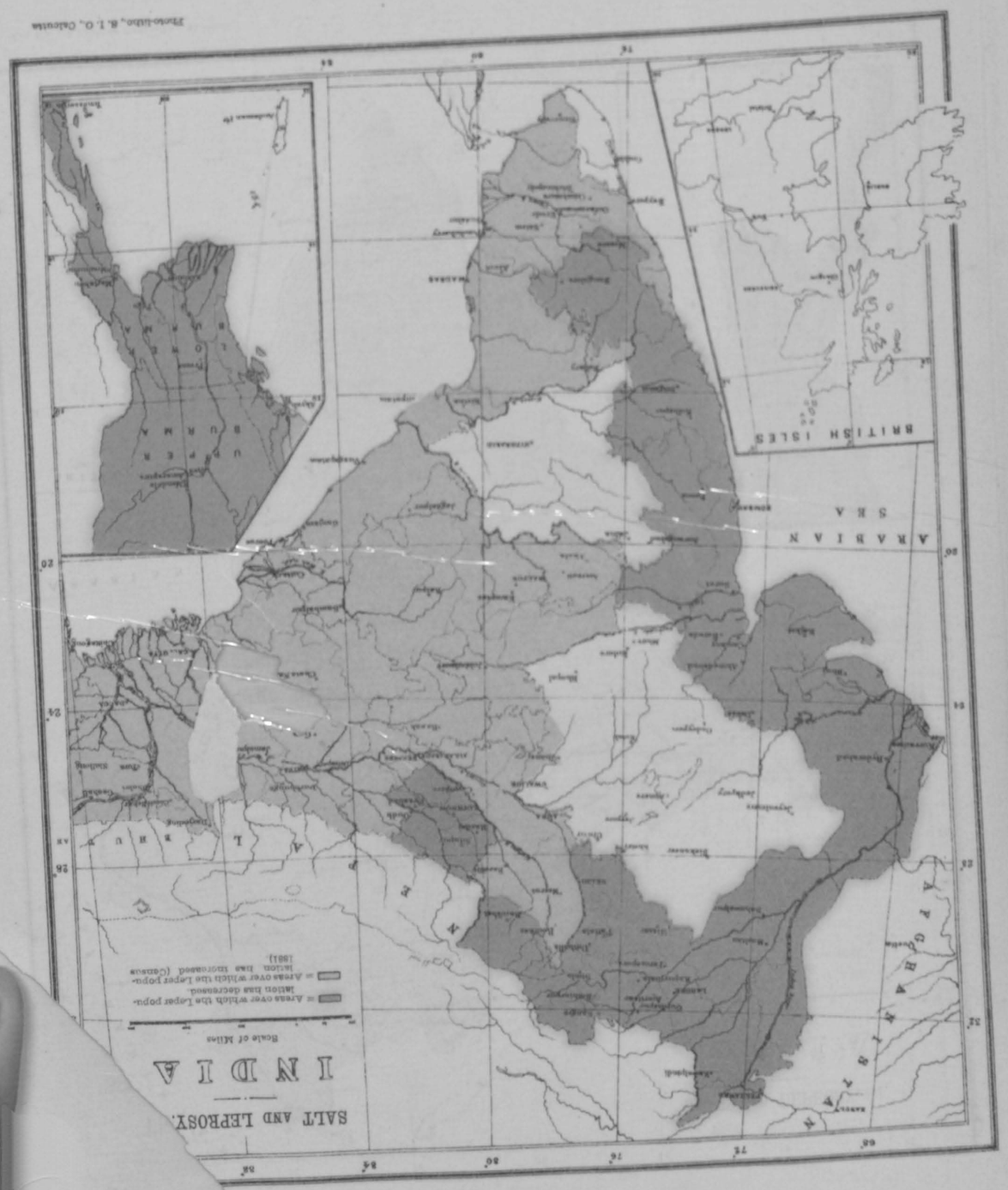
The best authorities on dietetics prescribe a daily allowance of 300 to 460 grains of salts, but this includes the sum total of all saline matters necessary for nutrition.[19] Hence 100 to 200 grains of sodium chloride might be considered a fair daily allowance. The prison allowance of salt for adult labouring prisoners in the North-Western Provinces and Oudh is 100 grains daily, being at the rate of 4¾lb per annum. In the Punjab the allowance is a quarter of an ounce, or 5·7lb per annum. In Bombay, the Central Provinces, Burma, and Assam the allowance is half an ounce, or about 11¼lb per annum, while in Bengal and Coorg it is 14¼lb per annum. In Madras, finally, the daily rate varies from half an ounce in the district prisons to an ounce in the Central Gaols, undoubtedly an unnecessarily large allowance. The scale varies with the diet of the prisoners, being highest in the rice-eating provinces, where also the consumption of salt by the population outside the prisons is largest. In the North-Western Provinces and Oudh, where the allowance is 4¾lb in the prisons, the consumption of salt per head throughout the province is about 8lb per annum, and it is much the same in the Punjab. And it is remarkable that though in the former provinces the price of salt has since 1871 decreased about 50 per cent., yet the annual consumption per head has remained almost stationary. The non-criminal population includes, it must be remembered, a large number of children, and allowance being made for these, the consumption per head of adult population in most districts is very much in excess of the prison allowance which experience has proved to be ample.

Now as it has been shown that 9 annas, even when the

(19) M. Foster: op cit., page 411.

Rep. No. 406, Leprosy Comm.—Sept. 91.—912. Photo litho., S. T. O., Calcutta





salt is most expensive, will buy the native all the salt he requires for his own personal use during the year, it cannot be said that the high price of this article debars him from obtaining his necessary supply of salt. So if there be any causal connexion between salt and leprosy, there is no reason why the native should go without his salt, as a few pence will procure his annual demand for it.

The best mode of showing that such a connexion between leprosy and the price of salt does not exist is a comparison of the leper returns for the three censuses of 1872, 1881, and 1891 side by side with the prices of salt during the decennial periods preceding the enumeration. This is done in Table II.

Under "A" all those provinces and divisions are given where, with the exception of the north-western division of the Punjab, the price of salt has steadily diminished, while under "B" those districts are found where the conditions are reversed. It will be seen that the figures do not establish any causal connexion between a high price of salt and leprosy, since from the statistics no relativity can be established. *The want of salt, therefore, cannot, in the opinion of the Commissioners, be held responsible for the origin or maintenance of the disease.*

To illustrate this statement more graphically two maps have been constructed. In the former the districts where the price of salt has risen have been coloured red, blue indicating a fall in the rates. Similarly, in the other map, red denotes an increase in the leper ratios since the first census, and blue a diminution. If there were any actual connexion between the price of salt and leprosy, the colours in the two maps ought to correspond. It will, however, be seen that they almost replace each other.

TABLE

Relation between Leprosy

A

Province.	Division.	Salt (from 1861—70) No. of seers per rupee.	Proportion of lepers per 10,000 (Census 1872).	Salt (1871—81) No. of seers per rupee.	Proportion of lepers per 10,000 (Census 1881).	Salt (1881—90) No. of seers per rupee.	Proportion of lepers per 10,000 (Census 1891).
Assam	Surma Valley	7·90	1·6	8·41	7·3	10·69	13·2
	Brahmaputra Valley	2·07		7·24	7·0	9·60	11·7
Bengal	Eastern	8·18	3·2	8·20	5·5	10·59	4·3
	Deltaic	8·76	6·1	8·84	7·1	11·28	5·2
	Central	8·39	16·2	8·54	19·0	11·13	16·0
	Northern	7·71	7·8	7·69	11·8	10·78	5·8
	Orissa	9·45	2·9	9·81	12·5	11·72	11·2
	Chota Nagpore	6·16	2·5	6·95	5·3	9·07	4·4
	Behar, South	8·31	5·9	8·10	7·7	10·90	5·6
	Behar, North	7·75	2·6	7·69	4·5	10·62	3·6
North-Western Provinces.	Eastern	6·8	2·4	7·73	3·1	10·04	2·6
	Central	6·88	3·4	9·32	3·5	11·43	2·2
	Western	7·36	2·5	9·67	2·4	11·80	2·1
	Sub-montane	7·08	2·9	8·49	4·3	11·04	3·6
Oudh	Southern	6·50	7·0	7·90	3·3	10·31	3·8
	Northern	6·78	6·9	7·89	3·5	10·65	3·9
Punjab	Southern	7·99	5·2	9·34	2·7	11·66	1·7
	Central	8·25	4·1	8·92	2·4	11·86	·9
	Sub-montane	9·50	8·8	9·88	4·6	13·57	2·5
	North-Western	25·86	6·4	23·18	3·6	22·86	2·0
	Western	10·67	2·2	15·04	1·3	21·49	·5
Central Provinces.	Western	7·09	4·1	8·82	8·5	10·27	Returns not received.
	Central	5·56	1·7	7·77	4·5	9·67	
	Eastern	6·71	3·6	7·58	7·7	9·04	
Berar	Berar	9·26	6·4	8·85	14·0	10·36	12·7

II.

and the Price of Salt in India.

B

Province.	Division.	Salt (from 1861—70) No. of seers per rupee.	Proportion of lepers per 10,000 (Census 1872).	Salt (1871—80) No. of seers per rupee.	Proportion of lepers per 10,000 (Census 1881).	Salt (1881—90) No. of seers per rupee.	Proportion of lepers per 10,000 (Census 1891).
Burma	Tenasserim	39·12	12·3	29·82	4·2	20·21	2·6
	Pegu (deltaic)	22·82	14·2	24·14	9·6	21·63	8·1
	Pegu (inland)	31·34	11·8	23·39	6·9	14·19	6·5
	Upper Burma (Mandalay)	...	...	...	...	19·03*	15·9
	Arakan	48·32	3·8	35·1	2·3	30·48	2·7
Rajputana	Eastern	...	...	23·48	...	12·46	·7
	Western	...	...	48·47	...	15·93	2·3
Central India.	Indore, Neemuch Cantonment, and Gwalior	...	...	14·88	...	11·32	...
Bombay	Konkan	16·85	8·6	14·20	7·6	12·04	7·5
	Deccan	15·95	10·8	12·71	6·7	11·65	6·8
	Khandesh	13·0	13·1	12·90	11·2	13·19	8·8
	Gujarat	17·94	5·4	16·51	2·8	13·72	1·9
	Kathiawar (Rajkot)	...	...	50·69	2·2	40·06	1·5
Sind and Baluchistan.	Kurrachee, Hyderabad, Shikarpur, Upper Sind Frontier, Quetta	36·45	1·3	31·33	1·1	12·83	·7
Nizam's Territories.	Secunderabad, Bolaram	12·51	...	9·88	...	9·86	...
Madras	Malabar Coast	18·37	6·6	14·23	6·3	13·62	5·6
	South, Central	15·86	2·5	14·26	2·8	14·09	·8
	Central	13·16	3·4	15·44	2·5	13·46	1·8
	East Coast, North	18·82	4·0	14·61	4·9	12·8	4·8
	East Coast, Central	19·07	3·7	15·93	3·6	13·9	2·7
	East Coast, South	19·02	5·8	15·82	5·9	14·24	4·2
	Southern	18·	2·9	15·69	4·1	15·5	2·1
Mysore	Mysore, Bangalore	13·4	4·9	10·82	2·6	9·96	3·4
Coorg	Coorg	13·57	4·8	9·47	2·4	9·9	1·3

* Average of period from 1887 to 1890.

Lastly, water has been considered to be the vehicle of the leprosy bacillus, and thus a means of infection. In some parts of India this is a popular idea, and according to Boinet[20] a similar opinion is held in certain parts of China. The theory seems, however, highly improbable. For whether it be assumed that leprosy bacilli contained in the water enter the body by the mouth or through the broken cuticle, on such hypothesis the disease should be much more generally diffused throughout the empire than it actually is. The tanks in India, filled with more or less stagnant water, are frequented by all classes of people, and the leper is by no means everywhere prevented from bathing with the rest, or filling his lota side by side with a healthy person. He also attends "Melas," and bathes in the company of other pilgrims. Any one who has witnessed the life around the tanks will easily conceive that a disease, if spread by means of water, should be diffused to an alarming extent and at a great rate, especially if such disease be endemic and always present.

It seems, moreover, very improbable that the leprosy bacillus is capable of multiplying outside the human body. It is, however, quite possible that, like the tubercle bacillus, it remains dormant for a long time after leaving its host. But the above considerations point strongly against the supposition that it exists in ordinary water in this condition. Besides the bacillus has never been found in water, and the observations of the Commissioners were absolutely negative in this respect. They examined water from the tank at Tarn Taran, which is supposed to be beneficial to lepers, and is, therefore, always thronged by them. Yet, as will be seen from the Laboratory Report, although a large number of microscopical

(20) E. Boinet: La Lèpre à Hanoi (Tonkin). Revue de Médecine, X, No. 8.

specimens were prepared, in not a single instance could the leprosy bacillus be detected. *Water, therefore, can hardly be held responsible for the propagation of leprosy.*

Enquiry has been frequently made from intelligent native gentlemen, as well as from patients, as to certain habits and influences which might be concerned in the origin and propagation of leprosy. No instance of the transmission of the disease from an animal to man was met with. Leprosy must be considered exclusively a human disease, and it does not attack domestic or other animals as is the case with tubercular disease.[21] The effects of premature marriages and the consumption of opium, alcohol, and other stimulants were also enquired into, but with entirely negative results. And indeed, though undoubtedly certain classes indulge to excess in opium, tobacco, the various forms of Indian hemp, alcoholic preparations from sugar, "mhowa," and the toddy palm, etc., the races of India must be regarded upon the whole as decidedly temperate.

Leprosy and Antecedent Diseases.

The question of the effect of syphilis was also considered. It seems that when syphilis first appeared it was thought by many to be a new form of leprosy, a modified leprosy. The reason for this belief was the fact that syphilis appeared in Europe about the same time when leprosy began to die out. The leading physicians of that time, however, strongly contested such views. The people were so convinced of the erroneousness of this theory that the lepers refused to admit syphilitic patients into their hospitals, and the latter had to build special hospitals for themselves.[22] "And as leprosy

(21) *Cf.* Journal of the Leprosy Investigation Committee, No. 2, February 1891, page 130.
(22) R. Virchow: op. cit., pages 500 and 501.

became more and more an unknown disease, partly speculation, partly the observation of certain endemic syphilides, led some physician back to the ancient supposition."[23]

At the present time some authorities have propounded the converse theory that leprosy is an effect of syphilis, a modified syphilis. There can be no doubt that syphilis pathologically is nearer related to leprosy than any other affection.[24] This, however, is no justification for the more modern theory. In fact, as far as local changes are concerned, an almost greater resemblance exists between lupus and leprosy.[25] Yet no one has ever ventured to identify these two affections with one another.

Sir W. J. Moore's name is closely associated with what might be called the syphilis-hypothesis. He considers leprosy a phase of inherited syphilis.[26] In the opinion of the Commissioners this view cannot be supported for the following reasons: (*a*) the history of this hypothesis, as briefly alluded to above, is entirely against it; (*b*) there is at most only a resemblance or analogy traceable between the two diseases; (*c*) certain clinical facts absolutely disprove the theory, unless the present views on syphilis be entirely modified or in part given up. The fact that people contract syphilis after having become affected with leprosy, is quite irreconcilable with the theory under consideration that leprosy is a phase of syphilis: and it matters little whether it be congenital or acquired. Of 154 individuals who confessed to having suffered from syphilis, 12 had been thus affected after the appear-

(23) R. Virchow: op. cit., pages 500 and 501.
(24) *Ibidem.*
(25) *Ibidem.*
(26) Journal of the Leprosy Investigation Committee, No. 1, August 1890, page 27, and Lancet, May 17, 1890.

ance of leprosy. At a few asylums every leper was consistently interrogated with a view to obtaining a more or less reliable ratio of syphilis amongst lepers. Out of 458 lepers examined 88, or 19·2 per cent., were found at one time or another to have suffered from this disease. This percentage may appear high, but too much importance must not be attached to the figures as they stand. In the first place the ignorance of the informants rendered it impossible to be sure that the disease they described was truly syphilis, and undoubtedly a certain number of cases must be deducted. Yet many of these lepers had the unequivocal marks of tertiary syphilis on their bodies. Again, no figures are obtainable showing the extent to which syphilis and venereal diseases prevail amongst the general population, though this is known to be very large. Lastly, it may be objected that in those instances where syphilis had been contracted subsequent to the development of leprosy, the ignorance of the informant rendered it difficult to decide with certainty as to which was the prior event. These concessions must naturally detract a great deal from the value of Table III. However, three authentic cases were seen by the Commissioners, where there could not be the slightest doubt that syphilis was contracted subsequent to the disease. These were all seen at the asylum in Madras. Two were young men who had suffered from leprosy for the last four and five years respectively: each of them had a typical roseolar secondary eruption, the characteristic appearances of the tongue, tonsils, and fauces, and moreover one of them presented the remains of a true Hunterian sore, while the other had lost the fraenum praeputii and showed an unmistakable scar. The third case concerned a low caste Hindu, twenty years of age, who had suffered from tubercular leprosy for the last twelve months

and at the time of examination actually had a primary sore on the glans penis. These three instances alone are sufficient to destroy the syphilis-hypothesis.

TABLE III.

Relation of Syphilis to Leprosy.

Asylum or Locality.	Syphilis Cases. Before Leprosy.	After Leprosy.	Total.	Remarks.
Agra	6	...	6	
Aligarh	1	...	1	
Almora	7[1]	1	8	(1) Three cases contracted syphilis at about the same time they became lepers.
Bangalore	1	1	2	
Belgaum	2	...	2	
Benares	3	...	3	
Burdwan	5	...	5	
Calcutta	10[2]	2	12	(2) One case contracted syphilis at the same time he became a leper.
Calicut	1	...	1	
Dehra Dun	5	2	7	
Delhi	1	...	1	
Dharmsala	4	...	4	
Fyzabad	3	...	3	
Gya	11	...	11	
Jubbulpore	2	...	2	
Kapurthalla	1	...	1	
Lucknow	2	...	2	
Madras	2	...	2	
Mandalay	21	1	22	
Moulmein	4[3]	...	4	(3) One case contracted syphilis about the time he became a leper.
Nagpur	22	2	24	
Naini and Allahabad	1	...	1	
Naini Tal	1	...	1	
Peshawar	2	...	2	

Table III—*continued.*

Relation of Syphilis to Leprosy.

Asylum or Locality.	Syphilis Cases. Before Leprosy.	After Leprosy.	Total.	Remarks.
Prome	6[4]	...	6	(4) One case contracted syphilis about the time leprosy began.
Purulia	4	...	4	
Rawalpindi	1	...	1	
Sialkot	1	...	1	
Tanjore	1	...	1	
Tarn Taran	2	...	2	
Thayetmyo	6[5]	3	9	(5) One case contracted syphilis about the time he became a leper.
Trichinopoly	1	...	1	
Umballa	2	...	2	
Total	142	12	154	

Again, even allowing that a certain number of the 154 cases specified in Table III were instances of gonorrhœa, does not the fact that the remainder contracted syphilis at all—and many of them had undoubtedly done so—militate against Sir W. J. Moore's theory? For as these men became lepers they must have been born in the particular phase of congenital syphilis. How, then, is it to be explained that many of these men presented the typical signs of tertiary syphilis concurrently with those of leprosy?

Lastly, it has never been shown that syphilis passes into leprosy, or *vice versâ*. Nor does syphilis ever modify the progress of the local changes in leprosy; and the converse also holds good.[27]

(27) R. Virchow: op. cit., page 501.

In conclusion, all that has been said on heredity may be used as arguments against this hypothesis.

It is, therefore, impossible to regard syphilis as having any real or specific influence on the production of the disease, or as acting otherwise than by lowering the general tone of the system, and thereby rendering it more liable to attack by, or hastening the progress of, leprosy.

Other diseases have been considered the possible cause of leprosy, as malaria or scrofula. It is, however, so plain that there is no scientific evidence for such assumptions that mere mention of these hypotheses will suffice.

Summary.—The Commission have come to the conclusion, after full consideration of the circumstances of life of the average native of India, that neither the form of diet, nor the sanitary environment of the individual, have any specific action in the causation of leprosy. But, as will be seen from what has been written, sanitation in this country is far from satisfactory, and leaves very much to be desired in the way of improvement. And it is probable that bad hygienic surroundings, deficient or improper food, poverty, exposure, and such diseases as syphilis, are all factors of great importance in reducing the vital powers of the organism and rendering it more susceptible to attack. All such indirect and non-specific causes are specially operative in the case of the offspring of lepers, who are commonly beggars, and living under the most unfavourable conditions for health. Nothing has struck the Commission more forcibly during this enquiry than the improvement in the general health of the sufferer which follows residence in a well-conducted asylum, where cleanliness, regular diet, and sanitary principles are insisted upon and maintained.

CHAPTER VII.

The Treatment of Leprosy.

DURING the stay of the Commission in India little opportunity occurred of personally investigating the effect of treatment on leprosy, for only a short time was spent in any station except Simla, and the latter place was too far from a leper asylum to allow of systematic observations being made. Information as to treatment had, therefore, to be drawn from Indian and other Government reports, from various works on leprosy, and from replies of officers in charge of leper asylums, and of Civil Surgeons who had treated the disease in district hospitals and dispensaries.

It has already been pointed out that the incurability of leprosy by any means yet known is unfortunately certain. The following remarks must, therefore, be understood to apply to palliative treatment.

This may be conveniently considered under three heads—

1. Hygienic.
2. Medicinal.
3. Surgical.

Hygienic.

The amount of hygienic treatment possible naturally varies with the circumstances of the patient. What may be practicable for the rich man will be unattainable by the poor one. This is a truism for all diseases, but it is peculiarly applicable to leprosy. For example, it has been found beneficial in some cases of leprosy

to remove the patient to a temperate climate where leprosy is not endemic. This proceeding is, of course, inapplicable to the great majority of Indian lepers, and can, therefore, be dismissed at once. Similarly, if all the lepers of India could be made to wash regularly, to live in roomy and well-ventilated houses, to eat only fresh and nourishing food, to engage in gardening or other healthy outdoor occupation as far as their crippled state would permit, and to observe the greatest care with reference to drainage and sewage disposal, there is no doubt but that their disease would run a much milder course. For this reason, apart from any question of segregation on other grounds, the establishment of voluntary asylums under careful supervision throughout India would do much to lessen the sufferings of the leper, for in them he would be taken away from his usual insanitary surroundings.

The difference between the condition of the lepers examined by the Commission in well-organised asylums, and that of outcasts, in many cases without home or friends, collected by the civil authorities, was sufficiently striking.

Medicinal.

Many drugs have been used in the treatment of leprosy and widely different opinions have been expressed as to their utility. The many secret remedies employed from time to time may be at once dismissed, for they cannot concern a scientific inquiry. Nor would space permit a dissertation on all the known medicines which have been given for leprosy. Only those will be discussed which have attracted attention, or which have appeared to be of some value.

The oils of India may be conveniently considered in the first

place. Dr. George Watt[1] mentions the following oils as having been used in the treatment of leprosy—

1. Aibizzia Lebbek.
2. Anacardium occidentale.
3. Cynometra ramiflora.
4. Dipterocarpus turbinatus.
5. Gynocardia odorata.
6. Hydnocarpus Wightiana.
7. Hydnocarpus venenata.
8. Pongamia glabra.
9. Psoralea corylifolia.
10. Semecarpus Anacardium.

Dr. Vandyke Carter[2] also says that the oil of Arachis hypogæa, the ground-nut, has been used, but that its utility is not apparent.

Of the above eleven oils only four need be discussed at length. They are Anacardium occidentale or Cashew-nut oil, Dipterocarpus turbinatus or Gurjun or Kanyin oil, Gynocardia odorata or Chaulmoogra oil, and Hydnocarpus Wightiana or Kowti oil. They will be referred to in the order in which they occur on the list.

ANACARDIUM OCCIDENTALE.

This oil played an important part in the plan of treatment adopted by Dr. Beauperthuy at Cumana in Venezuela. Several cures having been alleged; Dr. Bakewell was deputed to examine and report on the cases treated. His official report was presented to both Houses of Parliament in 1871, and spoke highly in favour of the results obtained by Dr. Beauperthuy.

The main features of the treatment are these—

1. Hygienic Rules.

The patient must have pure air and nourishing food, including

(1) Dictionary of the Economic Products of India, Vol. IV, page 309; Vol. V, Part I, pages 462 and 463; Vol. V, Part II, page 354.
(2) Reports on Leprosy, Second Series, page 33.

a moderate amount of fresh meat daily, with a sufficient quantity of fresh vegetables. He must abstain from salted meat or fish.

2. External Applications.

Frictions over the whole of the skin with cocoanut oil or olive oil are to be employed twice a day. These are to be followed by soap and water baths. The oil of cashew-nut is then applied by means of a small piece of sponge to the diseased parts. The skin must not be broken, and the exudation must be allowed to dry on, so as to form a crust. After about twelve days this will fall off, leaving the skin beneath clear and free from ulceration. It is not advisable to paint at one time an area greater than that represented by the skin of the leg or fore-arm. The applications should not be repeated at intervals of less than a week.

3. Internal Medicines.

These are perchloride of mercury in doses of one-fifteenth to one-twentieth of a grain twice a day for adults, and in cases where mercury is contra-indicated, sodium carbonate, given in doses of ten to twenty grains twice a day.

There is no doubt that this was a rational mode of treatment, and one which afforded distinct relief to the patient. The mistake made was in regarding it as curative. Whatever merit the treatment possessed lay in the strict regimen which had to be observed by the patient, and in the attention to the functions of the skin which was insured by frequent baths and frictions. The oil of cashew itself merely acts as a caustic, and the same result can be

less laboriously obtained in tuberculated leprosy by free excision of tubercles, as will be mentioned below.

The influence of the medicines given internally by Dr. Beauperthuy may fairly be disregarded. According to Dr. Bakewell a patient taking sodium carbonate progressed quite as favourably as one taking mercury perchloride.

Internal medication has always been the difficulty in leprosy. It is not hard to treat the skin complications and even to attain some measure of success, but so far no drug has been discovered which taken by the mouth will bear the relation to leprosy which mercury and potassium iodide bear to syphilis. Fresh crops of tubercles, or anæsthetic patches, soon make their appearance, and as was the case at Cumana, the condition of patients becomes as bad as, or even worse than, it was before they were treated.

DIPTEROCARPUS TURBINATUS.

The use of this oil in the treatment of leprosy was first recommended by Surgeon-Major J. Dougall[3] at Port Blair, Andaman Islands. Two emulsions were made by direction of Dr. Dougall. The first was to be taken internally and consisted of three parts of lime water and one part of gurjun oil. The second emulsion for external use contained equal parts of gurjun oil and lime water.

The lepers rose every morning at daybreak and bathed, using dry earth as a detergent, so as to rub off the previous day's inunction. They afterwards drank half an ounce of the first emulsion.

(3) Surgeon-Major J. Dougall, M.D. Report on the Treatment of Leprosy with Gurjun oil. Calcutta, 1875: page 6.

The second was then rubbed in for two hours. At 3 P.M. the same dose was given, and another two hours' friction practised.

Dr. Dougall tried the effect of this treatment on twenty-five lepers for a period of six months and says[4]: "the time has been long enough to show that leprosy, both tubercular and anæsthetic, can not only be arrested, but the condition of the lepers can be greatly ameliorated; and men here who have not for years been able to do more than drag out a miserable helpless existence are now able and willing to work, and every sore is quite healed."

This report was sufficiently encouraging to justify investigations into the gurjun oil treatment of leprosy. The Madras Government in the course of the next year published an account of the results obtained with gurjun oil in the hospitals and asylums of that Presidency. Surgeon-General E. G. Balfour, in his covering letter, arranges the medical reports in three groups[5]—

(1) *Those of officers in charge of the three Leper Hospitals.*—In these reports the opinion is expressed that the oil does not possess any particular efficacy;

(2) *Those of officers in charge of Civil Dispensaries and Gaols.*—These on the whole are favourable, and several cases of improvement are reported;

(3) *Those of Regimental Medical Officers.*—The cases under this head are necessarily few, as declared leprosy is considered to unfit the subject for further service and he is discharged. Gurjun oil is reported to be useful in alleviating the more prominent symptoms of leprosy, and one case is said to have been cured.

(4) Op. cit., page 14.

(5) Report on the Treatment of Leprosy with Gurjun oil and other Remedies in Hospitals of the Madras Presidency. Madras, 1876: page ix.

On the whole, Dr. Balfour considers the results of the trial of gurjun oil to be encouraging, but points out that in the supposed cases of cure it will be necessary to watch the future medical history of the patient.

In Dr. Watt's Dictionary[6] a summary is given of answers received from nineteen medical men in India who were specially questioned as to the utility of gurjun oil in leprosy. Of these answers thirteen were positive, three negative, and three neutral.

Answers were received by the Commission from a large number of stations in which gurjun oil had been used for leprosy. Many of these answers reported that more or less relief had been obtained, but in no instance was there any allusion to a cure. In some cases the relief afforded was very slight. From other stations the answers stated that no effect whatever had been observed. Some answers were neutral, the patients having disappeared from observation, or the time during which they had been under treatment having been too short to draw conclusions as to the efficacy of the oil.

Dr. Hillis, who tried this treatment in the Mahaica Leper Asylum, says[7]: "I can claim for gurjun oil that, given a suitable case, the disease can be arrested by it, and that in a few instances there has been no return of the disease for over two years, but it may nevertheless be premature to say a cure has taken place."

This experience was not borne out in the Trinidad Asylum,[8] where a trial of the oil showed only such temporary relief as would follow frictions with cocoanut oil or any other simple application.

(6) Op. cit., Vol. III, page 169.

(7) Leprosy in British Guiana, page 224.

(8) Report on the Trinidad Leper Asylum for 1884.

Dr. Vandyke Carter[9] also failed to find that gurjun oil exerted any specific effect on the course of leprosy. He used the drug internally and externally in selected cases for months, without obtaining any perceptible improvement.

From the above review of the various trials which have been made with gurjun oil, it appears that though a certain amount of temporary relief may follow its use, it is very doubtful whether this is more than can be obtained with any common oil. Dr Dougall's success at Port Blair was probably due to the fact that he was dealing with a colony of convict lepers under prison discipline, and was, therefore, able to carry out his plan of treatment far more rigorously than in an asylum where, as a rule, patients are averse to any therapeutic measures which involve the least exertion. It is quite possible that the prolonged frictions with dry earth, and the regular sea-bathing, were quite as potent in affording relief as the use of the gurjun oil.

GYNOCARDIA ODORATA.

This oil has long been known in the East as a remedy for leprosy, but it is only of late years that it has attracted notice in Europe. Like gurjun oil it is used both internally and externally.

Of eight answers recorded by Dr. Watt[10] from medical men who had used chaulmoogra oil in leprosy, four were favourable, two denied that any result was obtained, while two were neutral and expressed no opinion as to the value of the oil.

(9) Reports on Leprosy, Second Series, 1876, page 36.
(10) Op. cit., Vol. IV, page 194.

Dr. Vandyke Carter[11] selected cases for treatment with chaulmoogra oil and arranged them under as follows:—

(*a*) Young fairly nourished* subjects with early disease. In them the result was decidedly beneficial;

(*b*) Confirmed, but not disabled lepers. The effect of the oil on these was moderately beneficial.

(*c*) Confirmed, disabled, or broken-down subjects. No result was obtained in these patients.

Dr. Carter goes on to say that the longer the oil is used, the more favourable will the result be, both in degree and permanence. The good effect may last for three, four, or five years after prolonged use of the oil, but the disease is never eradicated even in the most suitable cases.

Of the first forty-seven answers received by the Commission from asylums and stations in which chaulmoogra oil had been used, thirty reported more or less relief after the use of the oil. In some the relief was very slight, while in others the disease was said to be arrested. In no case was a cure reported. From twelve stations the reports showed that there was no effect after chaulmoogra treatment, while five answers did not express an opinion one way or the other.

Mr. Sakharam Arjun,[12] who was in charge of the ward for incurables in the Jamsetjee Jeejeebhoy Hospital, Bombay, gives his opinion of the value of chaulmoogra oil as follows:—

"Under the prolonged and continuous use of this oil the progress of the disease is arrested, the skin becomes soft and supple, the discolorations vanish, the different morbid sensations leave the

(11) Op. cit., page 34.
(12) Report on Leprosy, 1873.

patient, the mental hebetude passes away, the impaired sensibility is completely or partially restored, the ulcers heal and cicatrise though ever prone to break out again, and the general nutrition of the tissues improves, patients crippled before being known to walk about unassisted and to gain in strength and weight."

In the Trinidad Leper Asylum[13] many of the more intelligent patients asked for chaulmoogra oil of their own accord and continued its use for several months.

One man used the oil internally and externally for seven years. The tubercles disappeared from his face and extremities and the anæsthesia was lessened.

The fact that these lepers persevered in any plan of treatment is in itself strong presumptive evidence that they derived some benefit from the drug employed, for, as was pointed out earlier in this chapter, such patients soon tire of any systematic medication.

The chief results obtained in eighteen cases were—

1. Increase of perspiration.
2. Decrease of tubercles.
3. Improved appetite and sense of well-being.
4. Increase of sensation.
5. Increased suppleness of skin and lessening of pains in joints.

A similar result was observed in a highly intelligent private patient who had taken chaulmoogra capsules for six years, and who had also tried residence in Europe. At one time he took seventy-five drops of the oil a day. When last seen the face was clear, but there were one or two nodules in the lobes of the ears, and a few small reddish patches on the thighs. The intervals between

(13) Report on Trinidad Leper Asylum for 1889, pages 11 and 12.

the tubercular outbreaks were much greater, and the patient was able to do a great deal of riding.

From the above experiences derived from various sources it seems that the action of chaulmoogra oil in leprosy, though at the best palliative, is nevertheless more marked than that of gurjun oil. Indeed it is probable that a prolonged and regular use of this oil may in some cases arrest the progress of the disease, though for how long must still be doubtful.

HYDNOCARPUS WIGHTIANA.

This oil was employed together with chaulmoogra oil in the treatment originated by the late Mr. Bhao Daji, which attained such notoriety in Bombay about twenty years ago. Apparently the first results obtained were encouraging, but Dr. Vandyke Carter,[14] who was living in Bombay at the time, and who had, therefore, ample opportunities of observing the effect of this treatment, writes as follows:—"I have before me notes of three or four cases which were duly treated here, and in none has a cure been effected, for either no good whatever resulted, or the symptoms are simply quiescent." Dr. Carter, however, considers that kowti oil is a useful aid in the treatment of leprosy.

The most noteworthy oils used in the treatment of leprosy have now been discussed. From what has been said above it will be evident that any oil which is thoroughly rubbed into the skin may afford more or less relief. Hence it will not be necessary to discuss the other seven oils mentioned at the beginning of this section, for no specific action can be claimed for any of them.

(14) Reports on Leprosy, Second Series, 1876, page 36.

Two common Indian plants, which have attained a considerable reputation in the treatment of leprosy, may here be mentioned. They are the Madar (Calotropis gigantea) and the Asiatic Pennywort (Hydrocotyle Asiatica). From evidence collected, however, it appears that their value has been overstated.[15] The first is said to be temporarily useful as a tonic in recent cases of leprosy, while the stimulant effects of the second on the circulation are most marked in the preliminary anæsthetic stages of the disease. These drugs have clearly no specific action in leprosy.

Arsenic is a drug which has been largely used in the treatment of leprosy, and sometimes with good result. Dr. Bowerbank[16] writing from Jamaica says: "In only one case did medical treatment seem to keep the disease in check. During eighteen or twenty years the patient, a female, had repeated attacks of apparently intermittent fever, and on each occasion the characteristic spots made their appearance; she had also anæsthesia and slight enlargement of the eyebrows and lobules of the ears. The use of Fowler's solution always checked the disease."

Dr. W. Nicholson[17] of Antigua also remarks: "Arsenic is the only remedy which in my practice has had any effect in arresting the disease, and that only for a time. I have seen the tubercles disappear under its use, sensation restored to fingers that were incapable of feeling and using a needle, so that the patient was enabled to sew, yet the disease returned and proved fatal."

In the Trinidad Asylum[18] arsenic was given during several

(15) Dictionary of the Economic Products of India, by Dr. G. Watt, Vol. II, page 45; Vol. IV, page 313.

(16) Report on Leprosy by the Royal College of Physicians, 1867, page 14.

(17) Op. cit., page 21.

(18) Report on Trinidad Leper Asylum, 1884.

months to two lepers who were particularly subject to frequent acute outbreaks of tubercles. One of these patients was a German, the other a slightly coloured Barbadian. Both had very delicate skins, and the result observed after prolonged treatment with liquor arsenicalis was a very marked diminution in the frequency of the tubercular eruptions. The German, in fact, was free from an attack for about a year, whereas before he began taking arsenic the exacerbations had occurred at intervals of a few weeks.

The use of ichthyol and resorcin in the treatment of leprosy was recommended by Dr. Unna of Hamburg in 1886. Since that time they have been used in various parts of the world, but the results obtained have hardly fulfilled expectations. It is true that many of the cutaneous complications of leprosy may be relieved or removed by the use of these drugs, but the same result can be obtained with a simple application such as vaseline. Ichthyol and resorcin have lately been issued by the Government for trial in the various Indian stations. Reference to the table at the end of this chapter will show that little effect has followed their use in leprosy.

Still more recently Dr. Lutz in Hawaii has brought to notice a new group of drugs for the treatment of leprosy. In a report, dated June 30th, 1890, he says: "in general we have had no instance of the disease making progress in patients treated by the most efficacious means, that is, salol and salicylate of soda internally, and chrysarobin and pyrogallic acid externally."

Some members of the Commission tested the action of salol on two lepers in Simla who happened to present themselves for treatment, but observed no result. Surgeon-Major H. D. Cook kindly treated a number of patients in the Madras Leper Asylum with salol, and his report is appended.

From an ambulatory treatment little or nothing could be expected, as this naturally excludes all clinical observation. Dr. Cook's verdict confirms the opinion of Dr. Lutz, that as a palliative, salol is of decided value in leprosy. The results, even after a course of five weeks, are so promising that the treatment is being continued at the Madras Asylum. No doubt, prolonged and critical observation is necessary before a decided opinion can be given; and this Dr. Cook has fully realised. There can, however, be no question as to the temporary benefit derived from the salol treatment. Doses of twenty grains were given three times a day for two weeks, and then increased to thirty grains three times a day. Apparently large doses must be given for a long time. Most of the cases subjected to the treatment suffered from tubercular or mixed leprosy. The greatest testimony in favour of the drug is, that the patients professed to feel better and gladly persisted in the treatment, while the native as a rule is easily disheartened and will not subject himself to therapeutic measures which after a short trial give him no relief. Bad effects were hardly ever observed, even after large doses of salol. On the other hand, tubercles seem to decrease in size, ulcerate, and then subside or heal up: a fresh crop of tubercles follows and goes through the same phases. Sensation was often restored, and anæsthetic areas, in fact, at times became hyperæsthetic; patches improved in colour, and the scaly, shiny appearance of the skin which is so characteristic of certain forms of leprosy, assumed a healthier look, and the skin became moist and more sensitive. The general condition of the patient often improved greatly.

With such favourable results after so short a period, it is justifiable to persevere. Accordingly Dr. Cook is continuing the treatment, and thus better means will be given to fully estimate the value of salol. If future experience confirms these early observations, then Dr. Lutz has indeed found in salol a valuable drug for the palliative treatment of leprosy.

A brief allusion only need be made to such drugs as perchloride of iron, potassium iodide, and cod-liver oil. These are often very useful adjuncts in the treatment of leprosy, but their action is exactly the same as in other diseases. Mercury may be used when the leper is suffering from syphilis. The latter disease will be rapidly brought under control, while the leprosy remains uninfluenced. The risk of giving mercury in leprosy, which some writers have insisted on, appears to be overrated. There is no doubt that with ordinary care this drug may be as freely given in leprosy as in any other disease.

From the above evidence it would appear that chaulmoogra oil and arsenic are the most valuable drugs at present known for the palliative treatment of leprosy; and if further experience and observation justify the hopes expressed by Dr. Cook at the beginning of his treatment of lepers with salol, then the latter must rank high among the drugs used and given for the amelioration of the general and local condition of these patients. It must, however, always be remembered that spontaneous subsidence of tubercles, or even arrest of the disease, may take place without any treatment, hence it is always difficult to estimate the part contributed by drugs to any good result which may follow their use.

Surgical.

Much may be done to relieve the leper by surgical interference.

It may be stated as a general rule that any operation which would be undertaken on another patient may be performed on one suffering from leprosy. The rapidity with which the tissues of lepers heal after operation is remarkable. This may, perhaps, be due to the excess of fibrin which has been found in the blood of these patients, a fact which was first pointed out by Danielssen and Boeck,[19] and which has since been confirmed by Hillairet[20] and at the Trinidad Asylum.[21] In the last-named institution an examination of the blood of fifty lepers, tuberculated, anæsthetic, and mixed, showed the average quantity of fibrin to be ·7 per cent.

An operation which has attracted considerable attention as a means of treatment in leprosy is nerve-stretching. This has yielded good results as practised by Surgeon-Major Lawrie,[22] and by Drs. Downes,[23] Neve,[24] Mitra,[25] and others in India. In Trinidad[26] out of one hundred cases operated on, more or less relief was obtained in about half. The chief results noticed were relief of pain in the course of the nerves operated on, healing of perforating ulcers in the area supplied by these nerves, and, more rarely, some decrease of anæsthesia. Unfortunately none of these effects were permanent, and the operation had sometimes to be repeated on the same nerve. There is no doubt, however, that nerve-stretching is a valuable aid, especially in the treatment of the distressing neuralgia which often complicates leprosy. But the operation cannot be claimed as in any way curative.

(19) Traité de la Spedalskhed. Paris, 1848.
(20) Annales de Dermatologie et Syphilographie.
(21) Report on Trinidad Leper Asylum for 1887.
(22) Lancet, 1881.
(23) Lancet, 1886.
(24) Edinburgh Medical Journal, 1884-85.
(25) Indian Medical Gazette, 1888.
(26) British Medical Journal, 1888.

Free excision of cutaneous tubercles is a procedure which is followed for a time by good results, and if a drug could be found which given internally would prevent recurrence of these growths, there would be a fair hope of ultimate success.

By cutting deeply it is possible to eradicate single tubercles or circumscribed masses of tubercle and to obtain a clean cicatrix, but after a few weeks or months the growths begin to recur in the sound skin around these cicatrices, and the patient relapses as rapidly as after the Beauperthuy treatment.

Much pain is often experienced in perforating ulcers and in sinuses leading to dead bone, especially by patients who are otherwise anæsthetic. Free incision down to the bone gives great relief in these cases. The effect of this treatment is appreciated by the patients, who eagerly beg to be operated on. There is also no doubt that early incision in such cases does much to prevent the gangrene which is so common in leprosy. When this gangrene has actually set in, amputation high up will often save life, and the patient will regain flesh and strength in a remarkable manner.

Dyspnœa, due to invasion of the larynx by tubercles, is not uncommon in leprosy, and sometimes necessitates tracheotomy. Life may be greatly prolonged after this operation. Dr. P. S. Abraham,[27] after a visit to the Norwegian Leper Asylums in 1888, writes: "I was shown numerous cases of amputations and tracheotomies. One patient at Bergen had worn a tube for three years, another for seven years, another at Trondhjem for ten years, and a great many others in all the asylums for shorter periods. In one case at Trondhjem the larynx subsequently became functionally useful, and the tube was discarded and the opening closed."

(27) Epidemiological Society's Transactions, Vol. VIII, page 135.

Lastly, reference must be made to the ophthalmic complications so common in leprosy. Leloir[28] noted during his visit to Norway that of sixty-four lepers at Molde, forty-one had ophthalmic lesions, in thirty-seven both eyes were affected, while six were absolutely blind.

The two commonest affections of the eye in leprosy are invasion of the cornea by tubercles, and paralytic ectropion, the former occurring in the tuberculated, the latter in the anæsthetic form of the disease.

To arrest the progress of tubercles across the cornea various methods of treatment have been adopted. Kaurin has attained considerable success by the performance of keratotomy. Danielessen and Hansen have produced similar results by cauterizing the conjunctiva or cornea round the tubercle. In the Trinidad Asylum ligature of the vessels supplying the tubercle has arrested its growth for a time. Iridectomy will in some cases temporarily avert total blindness, though it cannot affect the progress of the disease. It must, however, be undertaken before the iris becomes involved in the leprous growth, otherwise it will be found impossible to excise sufficient to produce any appreciable effect.

For paralytic ectropion Kaurin practises tarsoraphy with good results. Epiphora is greatly reduced, and closure of the lids is effected.

Enough has now been said of the surgical treatment of leprosy to show that in any well-organised asylum no operation need be refused. Indeed the leper may be treated like an ordinary surgical patient in a general hospital.

This chapter will be concluded by the following table showing

(28) Traité Pratique et Theorique de la Lépre, page 316.

the medical and surgical treatment adopted in various Indian stations with the results obtained :—

*Results of Treatment of Leprosy in India.**

Station or Asylum.	Medical Officer.	Treatment.	Result.
Assam.			
Sylhet . .	Dr. T. O. Partridge	Gurjun oil and Lime water. Liquor arsenicalis.	Not satisfactory.
Bengal.			
Balasore . .	Assistant Surgeon Moti Lal Mukerjee.	Gurjun and Carbolic oils.	Slight relief, but no cure.
B ckergunge .	Surgeon G. Jameson	Gurjun oil internally and externally; Ichthyol and Resorcin internally and externally.	Anæsthetic patches disappear and ulcers heal.
Bankoora . .	Dr. Umes Chunder Mukerjee.	Gurjun and Neem oils; also Gurjun oil and Lime water.	Temporary relief.
Bhagalpur . .	Surgeon-Major W. Beatson.	Gurjun oil locally, Chaulmoogra oil internally and externally.	No permanent benefit.
Beerbhoom .	Dr. W. Forsyth .	Arsenic . . .	No effect.
Burdwan . .	Surgeon-Major G. Price.	Ichthyol and Resorcin	Improvement, but no absolute cure.
Calcutta . .	Dr. T. Nath Bose .	Chaulmoogra oil internally and externally; Madar powder, Neem oil, Salol and Dr. Unna's treatment.	No permanent benefit.
Chittagong .	Surgeon-Major W. Flood Murray.	Gurjun oil . .	Beneficial.

* Several stations have been omitted, as the answers to the questions sent out by the Commissioner arrived too late for the press.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Bengal—*contd.*			
Chittagong Hill Tracts.	Assistant Surgeon Kali Nath Banerjee.	Chaulmoogra oil internally and externally; Cod-liver oil, Iron, Iodine and Arsenic with local antiseptic treatment of ulcers.	Improvement in several cases.
Cooch Behar .	Dr. J. L. Hendley .	Gurjun, Carbolic and Chaulmoogra oils.	Tubercles appear to fade away; and relief to ulcers.
Dacca . .	Surgeon-Major F. C. Nicholson.	Gurjun, Chaulmoogra, and Carbolic oils.	Temporary improvement.
Eastern Bengal State Railway.	Dr. C. C. Bose .	Gurjun and Chaulmoogra oils.	Progress of the disease arrested and sensibility restored.
Gya . . .	Surgeon-Major R. D. Murray.	Gurjun oil and Arsenic	Some improvement, but never cure
Hooghly . .	Surgeon-Major B. Gupta.	Gurjun oil . .	Improvement in general health and healing of ulcers.
Lohardugga (Leper Asylum).	Reverend F. Hahn .	Liquor arsenicalis, Chaulmoogra and Gurjun oils.	The ulceration stops, the sores heal up, and general condition of patient improves.
Malda . .	Apothecary J. Kelly	Gurjun oil . .	No permanent relief.
Midnapore .	Surgeon-Major R. L. Dutt.	Arsenic and Potassium iodide where there is constitutional syphilis; Neem or Gurjun oils with Lime water.	The eruptions, tubercles and ulcerations disappear in most cases and in some great improvement, but not a radical cure.
Monghyr . .	Assistant Surgeon Upendra Nath Sen.	Iron, Strychnia, Quinine, Cod-liver and Neem oils internally; Perchloride of mercury lotion (1—1000), Carbolic oil (1—40), Gurjun and Neem oils externally.	Improvement of general health, healing of ulcers, and in some cases the disease checked and the symptoms ameliorated.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Bengal—*contd.*			
Mozufferpore .	Surgeon F. S. Peck	Hydrochloric acid internally; Ichthyol, Resorcin, Pyrogallic acid, Chrysarobin, Salicylic acid and Neem oil externally.	No perceptible improvement.
Mymensingh .	Surgeon-Major D. Basu.	Gurjun and Chaulmoogra oils with Lime water internally; and Gurjun and Chaulmoogra oils externally.	Much improvement, but no cure.
Patna . .	Surgeon-Major E. G. Russell.	Aristol externally. Gurjun, Chaulmoogra and Cashewnut oils and Liquor arsenicalis.	Amelioration in many cases.
Pooree . .	Surgeon E. H. Brown.	Chaulmoogra and Gurjun oils; Unna's treatment (Resorcin and Ichthyol).	Temporary relief.
Purulia . .	Surgeon-Major H. W. Hill.	Pyrogallic acid ointment. Ichthyol pill and ointment. Resorcin ointment: Aristol: Iodoform dressings: Carbolic oil.	Ulceration is arrested, but it breaks out again, if the individual partakes of fish diet.
Sealda (Campbell Medical School and Hospital).	Surgeon-Major R. Cobb.	Arsenic, Potassium iodide and Gurjun oil internally; Cashewnut, Chaulmoogra and Gurjun oils and Carbolic acid externally.	Temporary and local improvement.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Bengal—*concld.*			
Shahabad . .	Assistant Surgeon Nritto Gopal Mittra.	Gurjun oil internally and externally. Fowler's solution, Donovan's solution, Iron preparations, Resorcin and Ichthyol.	Little or no effect.
Sonthal Pergunnahs.	Assistant Surgeon Gopal Chunder Dey.	Gurjun and Chaulmoogra oils internally and externally; also Arsenic and Potassium iodide.	No permanent relief.
Tipperah . .	Surgeon R. R. H. Whitwell	Gurjun oil internally and externally.	None.
24-Pergunnahs .	Surgeon-Major J. F. P. McConnell.	Potassium iodide or Arsenic, or both internally, and Gurjun ointment externally.	Marked, but temporary amelioration of symptoms.
Bombay.			
Ahmedabad .	Surgeon-Major M. L. Bartholomeusz.	Ichthyol, Aristol and Resorcin; also Chaulmoogra and Gurjun oils.	No permanent benefit.
Belgaum . .	Surgeon-Major J. P. Greany.	Chaulmoogra and Gurjun oils, also Unna's treatment.	Slight relief.
Broach . .	Surgeon K. H. Mistri.	Chaulmoogra and Gurjun oils, both internally and externally.	Effects not noted.
Dharwar . .	Surgeon-Major W. McConaghy.	Chaulmoogra oil, both internally and externally, Cashewnut oil externally, Arsenic, also Gurjun oil.	No permanent relief.
Dhulia . .	Surgeon-Major K. A. Dalal.	Gurjun oil, both internally and externally.	No effect

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Bombay—*contd.*			
Hyderabad (Sind)	Surgeon-Major J. F. Keith.	Chaulmoogra oil.	No effect.
Jamsetjee Jejeebhoy Hospital.	Surgeon-Major W. K. Hatch.	Chaulmoogra and Gurjun oils.	Beneficial in anæsthetic form.
Kurrachee . .	Surgeon-Major J. McCloghry.	Gurjun oil.	None.
Mahableshwar .	Surgeon H. P. Dimmock.	Arsenic.	Marked improvement.
Matoonga . .	Dr. N. H. Choksy.	Chaulmoogra and Gurjun oils.	Too early yet to obtain any appreciable results.
Poona . .	Surgeon W. H. Burke.	Chaulmoogra and Gurjun oils, internally and externally, Antiseptic dressings to ulcers.	Improvement in general health and temporary relief to ulcers.
Rajkot (Kathiawar Agency).	Surgeon-Major F. C. Barker	Tonics, Alteratives, Chaulmoogra and Gurjun oils internally. Gurjun oil and other ointments to the ulcers.	No specific effect on the disease.
Ratnagiri . .	Surgeon-Major H. McCalman.	Gurjun, Chaulmoogra and Cashewnut oils, recently Resorcin, Ichthyol, Fish oil and Pyrogallic acid. Local antiseptic and stimulating dressings to ulcers, also Carbolic oil, Iodoform, Eucalyptus, Iron, Arsenic and Cod-liver oil.	No specific effect.

Results of Treatment of Leprosy in India —continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Bombay—*concld.*			
Sawantwadi .	Dr. D. G. Dalgado.	Arsenic pills internally. Chaulmoogra and Gurjun oils externally.	Invariably improvement in general health and partial subsidence of the disease in many cases, but no cure.
Thana . .	Surgeon-Major K. R. Kirtikar.	Chaulmoogra, Gurjun and Kowti oils, internally and externally, Hydrocotyle Asiatica, Aristol, Tonics, Massage.	In many cases diminution of size of tubercles, and some return of sensation in anæsthetic patches. No absolute cure.
Yerrowda Prison .	Surgeon-Major S. M. Salaman.	Alteratives, Tonics, Cooling lotions to the face, Chaulmoogra and Gurjun oils, internally and externally.	No effect.
Central Provinces.			
Damoh . .	Surgeon A. C. Deare.	Gurjun oil internally and externally. Carbolic oil.	Temporary benefit.
Narsingpur .	Brigade-Surgeon P. Cullen.	Chaulmoogra and Gurjun oils. Quinine and Iron. Antiseptic dressing to ulcers.	Benefit in a small number of cases.
Chhindwara .	Surgeon W. L. Price	Chaulmoogra oil. Iron and arsenic.	Slight improvement.
Sambalpur .	Assistant Apothecary D. O'Connell Murphy.	Internally — Tonics and Alteratives. Liquor arsenicalis. Bitter Infusions. Chaulmoogra oil and Lime water. Externally—Before ulcerative stage Chaulmoogra oil, after Carbolic ointment and oil and Antiseptic dressings.	Some of the ulcers healed, but no visible effects were noticed on the constitution.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Madras.			
Anantapur .	Assistant Surgeon B. F. Gonsalves.	Arsenic, Chaulmoogra and Gurjun oil. Potassium iodide. Tonics.	Temporary benefit.
Calicut . .	Surgeon-Major L. Beech.	Gurjun and Carbolic oils for ulcers.	Temporary relief.
Chingleput .	Surgeon S. C. Sarkies.	Gurjun oil and Tonics.	Relief, but no permanent cure.
Chitoor . .	Assistant Surgeon A. J. Hesterlow.	Gurjun oil internally and externally.	It ameliorates the symptoms, but no cure has been noticed.
Coimbatore .	Surgeon-Major H. J. Hazlett.	Gurjun oil . .	It certainly has a curative effect, but probably this is not permanent.
Coonoor . .	Surgeon-Major F. C. Smith.	Gurjun and Chaulmoogra oils.	Unknown.
Cuddapah .	Surgeon A. T. O'Hara.	Gurjun and Carbolic oils.	Only temporary relief after continued treatment.
Kistna . .	Assistant Surgeon C. Munisawmy.	Gurjun and Chaulmoogra oils. Arsenic. Cod-liver oil. Blood tonics.	Symptoms abate, but no single case cured.
Kurnool . .	Brigade-Surgeon L. C. Nanney.	Gurjun oil . .	Prolonged treatment with good food and hygiene; if it does not cure, it at any rate checks progress of disease.
Madras . .	Surgeon-Major H. D. Cook.	Chaulmoogra oil internally and externally.	No effect whatever on the disease.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Madras—*concld.*			
Madura . .	Surgeon W. B. Browning.	Gurjun and Chaulmoogra oils.	Any good result is due to increased cleanliness and improvement in nutrition under better hygiene and diet.
Nellore . .	Surgeon-Major G. L. Walker.	Internally—A mixture of Potassium iodide, Liquor arsenicalis and Liquor strychniæ, also Gurjun and Chaulmoogra oils externally. For ulcers Carbolic oil dressings, Iodoform and Mercury ointments.	Under treatment and good food disease markedly ameliorated, general health much improved, the ulcers heal, the raised macular patches became much less prominent but no case was cured.
Palliport . .	Surgeon-Major D. Elcum.	Gurjun, Chaulmoogra, and Carbolic oils. Fumigation with Carbolic acid.	State of ulcers improved slightly. No other result.
Tanjore . .	Surgeon-Major A. F. Nailer.	Chaulmoogra oil.	Improvement in general health.
Tellichery . .	Assistant Surgeon C. A. Lafrenais.	Gurjun and Chaulmoogra oils.	Very little benefit.
Trichinopoly .	Surgeon-Major H. Hyde.	Chaulmoogra oil internally and Gurjun oil externally.	Progress of disease retarded in some cases.
Vellore . .	Surgeon-Major T. J. H. Wilkins.	Sodium salicylate and Arsenic internally with Chrysophanic acid externally. Chaulmoogra oil.	In two cases disease appeared to be almost cured, but in others the symptoms did not seem to be relieved.
North-Western Provinces and Oudh.			
Agra . .	Surgeon-Major A. Willcocks.	Gurjun oil. Aristol. Resorcin and Ichthyol.	More or less improvement but nothing approaching a cure.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
North-Western Provinces and Oudh—*contd.*			
Aligarh . .	Surgeon-Major W. H. Cadge.	Chaulmoogra oil .	Slight relief.
Allahabad . .	Surgeon-Major J. McConaghey.	Ichthyol and Resorcin. Chaulmoogra oil.	Nothing beyond what might be expected after good diet and cleanliness.
Almora . .	Reverend G. H. Bulloch.	Gurjun and Chaulmoogra oils. Carbolic acid. Potassium iodide, Resorcin, and Ichthyol.	Ulcers were healed, but only for a time.
Bahraich . .	Surgeon W. Deane	Chaulmoogra and Gurjun oils, Ichthyol, Resorcin, and Aristol.	Chaulmoogra oil kept the sores healed, but Gurjun oil appeared of no use. No cure has been observed.
Ballia . .	Assistant Surgeon Sheoraj Misra.	Gurjun and Chaulmoogra oils internally and externally.	Relief, but no complete cure.
Banda . .	Surgeon-Major J. Moran.	Tonics, Arsenic, and Cod-liver oil internally and dilute Citrine ointment locally.	No satisfactory result.
Bareilly . .	Surgeon-Major J. Anderson.	Ichthyol internally and Aristol externally (since October 1890).	Ulcerations rapidly heal, but no change for the better in the constitution has been observed.
Benares . .	Brigade-Surgeon W. R. Hooper.	Gurjun oil . .	Only temporary benefit.
Budaun . .	Surgeon-Major J. C. C. Smith.	Chaulmoogra and Gurjun oils, Resorcin, Ichthyol, and Arsenic.	Ulceration in several cases cured and anæsthesia often relieved, but the thickening of skin and other symptoms do not yield to treatment.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
North-Western Provinces and Oudh—*contd.*			
Dehra Dun . .	Surgeon-Major G. G. MacLaren.	Chaulmoogra oil, Aristol, Gurjun oil, Sodium phosphate, Resorcin, and Ichthyol.	Chaulmoogra oil gave general relief. Aristol in many instances has proved a useful and rapid healing agent in external ulceration. No lasting good effect from Gurjun oil, Phosphate of soda, Resorcin, and Ichthyol.
Etawah . .	Surgeon-Major E. S. Brander.	Gurjun oil . .	Results negative.
Fyzabad . .	Surgeon J. Sykes .	Arsenic and juice of Madar internally, Gurjun oil internally and externally, Carbolic oil to ulcers.	Temporary relief.
Ghazipur . .	Surgeon J. F. MacLaren.	Arsenic internally, Chaulmoogra and Gurjun oils internally and externally.	In primary stage tubercles decreased after the use of Gurjun oil, but in advanced stages where there was ulceration, no benefit was observed. Ichthyol and Resorcin had no effect.
Gonda . .	Surgeon-Major C. Cameron.	Gurjun oil . .	Temporary benefit, but no cure.
Hardoi . .	Dr. G. D. McReddie	Arsenic, Gurjun oil, Carbolic acid. Chrysarobin externally and Ichthyol internally are now being tried on a prisoner.	No cure. Carbolic acid arrests ulcerations only. Tubercular appearance of skin decreased.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
North-Western Provinces and Oudh—*contd.*			
Jaunpur . .	Surgeon-Major F. C. Chatterji.	Gurjun and Chaulmoogra oils, Liquor arsenicalis, Tonics, Nerve-stretching.	General improvement, but no cure.
Jhansi . .	Surgeon C. P. Lukis	Chaulmoogra oil, Tonics, Nerve-stretching.	None. Nerve-stretching for anæsthetic leprosy is a complete failure.
Kheri . .	Surgeon-Major M. D. Moriarty.	Gurjun and Chaulmoogra oils, Arsenic, Iron.	Slight improvement.
Lalitpur . .	Dr. G. T. Leopold .	Gurjun ointment and Chaulmoogra oil.	Gurjun ointment seems to check the disease a little.
Mainpuri . .	Surgeon-Major G. M. Nixon.	Chaulmoogra and Gurjun oils. Donovan's solution. Tonics.	Marked improvement in many cases, but no complete cure.
Meerut . .	Brigade-Surgeon W. Moir.	Carbolic acid internally and externally with Arsenic and Donovan's solution. Gurjun oil more recently.	Any tonic treatment produces some temporary improvement, but there is no permanent cure. No success.
Naini Gaol .	Surgeon-Major G. C. Hall.	Arsenic, Mercury, Strychnia, Phosphorus, Chaulmoogra and Gurjun oils, Ichthyol, and Resorcin.	No effect whatever from any special treatment.
Partabgarh . .	Senior Apothecary S. Bond.	Gurjun and Chaulmoogra oils, Liquor arsenicalis, Mercury, Potassium iodide, Tonics.	Considerable relief after prolonged treatment and care.
Roorkee . .	Surgeon-Major J. Young.	Gurjun oil . .	No curative effect.

Results of Treatment of Leprosy in India—continued.

Station or Asylum.	Medical Officer.	Treatment.	Result.
North-Western Provinces and Oudh—*concld.*			
Sitapur . .	Surgeon-Major D. F. Barry.	Nerve-stretching .	Improvement in the area supplied by the nerve operated on. General progress of the disease unaffected.
Unao . .	Senior Apothecary F. W. Saunders.	Arsenic, Chaulmoogra oil, Tar ointment.	Ulcers begin to heal and the symptoms are palliated.
Punjab.			
Baba Lakhan (Sialkot.) .	Assistant Surgeon Mehta Duni Chand.	Liquor arsenicalis internally, Carbolic oil for ulcers, Gurjun oil both internally and externally, Tonics and Stomachics.	Symptoms relieved, but no case has been cured.
Hoshiarpur .	Dr. N. P. Datta .	Gurjun oil . .	Temporary relief. Ulcers begin to heal more quickly than before the use of the oil.
Jullundur . .	Surgeon-Major M. O'Dwyer.	No specific remedies. Carbolic oil and various ointments for ulcers.	No effect.
Ludhiana . .	Assistant Surgeon Bhagwan Das.	Tonics, Chaulmoogra and Gurjun oils.	Result not known.
Peshawar . .	Surgeon H. Hendley	Gurjun oil internally and externally, Chaulmoogra oil, Arsenic, Calcium sulphite, Tonics, Arsenic and Potassium iodide internally, Sulphur and Carbolic Acid externally.	Little or no effect.

Results of Treatment of Leprosy in India—concluded.

Station or Asylum.	Medical Officer.	Treatment.	Result.
Punjab—*concld.*			
Shahpur . .	Assistant Apothecary C. J. Maher.	Gurjun oil and ointment, Chaulmoogra oil and earth baths, Electricity.	No result.
Subathu . .	Reverend J. W. J. Wylie.	Tincture of Iodine, Carbolic acid.	Pain temporarily relieved. No cure.
Tarn Taran .	Assistant Surgeon Gulam Mustafa.	Chaulmoogra and Gurjun oils, Cashew-nut oil externally, Liquor arsenicalis, Potassium iodide, Iodoform, Carbolic acid, Liniments, Resorcin, and Ichthyol ointment externally, Ichthyol internally.	No marked effect. No diminution of anæsthesia of the benumbed part of the body. No change in the appearance of tubercles. When using these remedies, however, the skin remains soft and supple and patients become a little stronger.
Umballa . .	Surgeon G. W. P. Dennys.	Chaulmoogra and Gurjun oils, Ichthyol, Resorcin, and Aristol.	No case is ever cured. Diminution of tubercles. Cessation of several symptoms.
NATIVE STATES.			
Mysore.			
Bangalore . .	Brigade Surgeon T. J. McGann.	Gurjun oil, Carbolic acid, Iodine, Hoangnan, Ichthyol internally and Resorcin externally.	Temporary improvement, but in no case has there been a cure.
Punjab.			
Kashmir . .	Dr. A. Mitra .	Nerve-stretching is largely practised, Gurjun oil lately, Creolin for leprous ulcers.	Nerve-stretching checks the progress of disease in its early stage in a large number of cases.

APPENDIX I.

SURGEON-MAJOR H. D. COOK'S REPORT ON SALOL IN THE TREATMENT OF LEPROSY.

OFFICE OF THE SUPERINTENDENT, GOVERNMENT LEPER HOSPITAL,

Madras, 31st August 1891.

From—SURGEON-MAJOR H. D. COOK, M.B., Supdt., Government Leper Hospital,
To—A. A. KANTHACK, Esq., F.R.C.S., Member of the Leprosy Commission.

I have the honour to inform you that, in compliance with your request, I began the treatment of twelve patients, selected for the purpose, with Salol on the 26th ultimo and continued it up to date (a period of five weeks), strictly adhering to your instructions.

According to directions I administered 3 doses, a dose after each meal daily, beginning with 20 grains and increasing it to 30 grains. A careful diagnosis was made of each case, and duly registered prior to the treatment, and critically scrutinized from time to time during the treatment, and any perceptible reaction duly noted.

So far as can be seen from observation of the twelve cases under treatment Salol has a mitigating effect, ulcers heal and tubercles ulcerate and subside, fresh tubercles crop up and burst, subside, and are followed by another lot, and so on. I am of opinion that sufficient time has not been allowed to enable me to form a correct estimate of the value of salol as a therapeutic agent in leprosy, and that a steady, unremitted course of three months' treatment with it would at least be required. So I will keep up these cases and send you the continuation of the treatment, even after you go home, if you should so wish.

DETAILED REPORT OF TWELVE CASES OF LEPROSY TREATED WITH SALOL.

CASE I.—Hindu, male, aged 25 years; suffering from MIXED LEPROSY for last four years.

Present condition.—Borders and lobes of both ears tuberculated; minute tubercles on the nose and chin; forehead and cheeks slightly thickened.

Minute tubercles on the little fingers of both hands, the remaining fingers slightly thickened. Discoloration of the skin of the trunk and both thighs. Shins of both legs are covered with dark patches and shiny; feet are covered with scaly patches. Loss of sensation in little finger of right hand, and from knee downwards to tips of toes in both lower extremities.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Gaining sensation in the little finger of right hand.

August 10th.—Minute tubercles on the little fingers of both hands ulcerating, discharging freely, and then subsiding. *There is much improvement*, and patient is gaining more sensation in the little fingers of right hand.

August 10th.—Salol, gr. xxx, three times a day.

August 16th.—No change.

August 19th.—Fresh minute tubercles have appeared on both hands.

August 27th.—Some of the tubercles mentioned above have ulcerated and are healing up.

Treatment still continued.

CASE II.—Hindu, male, aged 26 years; suffering from TUBERCULAR LEPROSY for last ten years.

Present condition.—Minute tubercles on the lobes of both ears, nose and chin. Skin of face slightly thickened. Skin of the body discoloured, and covered with dry, scaly patches of a lighter colour than the natural skin; and also psoriasis. Discoloration of the skin of both upper extremities; both legs are covered with brown patches, peculiarly shiny; toes are thickened, and there is an ulcer on the inner side of the right great toe. No loss of sensation anywhere. Both feet are swollen.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Complains of severe pain over the abdomen and burning sensation on the soles of the feet after taking the medicine.

Treatment continued.

August 10th.—No improvement.

Salol, gr. xxx, three times a day.

August 16th.—No improvement.

Treatment continued.

August 19th.—Pain over the abdomen, and burning on the soles of the feet is worse; ulcer broke out on the dorsum of right foot.

August 27th.—No improvement.

CASE III.—Hindu, male, aged 32 years; suffering from MIXED LEPROSY for last nine years.

Present condition.—The skin of the whole face, chin, nose, and ears thickened and tuberculated; skin of trunk and extremities discoloured and thickened by a mass of minute inter-current tubercles. Fingers and feet slightly thickened; brown shiny patches on both shins; toes much thickened, nails wasting away. Slight anæsthesia in hands and legs. This is a more macular form of the disease. Slight loss of sensation on the soles of both feet.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Several tubercles on the trunk and extremities have ulcerated and subsided; gaining sensation in the tubercles of the face, being very painful to the touch.

Treatment continued.

August 10th.—More tubercles on the trunk and extremities have subsided. He is gaining sensation in hands and legs. Tubercles on the face somewhat depressed and not so much elevated, but very painful. *He states he is improving greatly.*

Salol, gr. xxx, three times a day.

August 18th.—Tubercles on the face look somewhat depressed and are painful, and those of the forearms have ulcerated and are subsiding.

Treatment continued.

CASE IV.—Pariah, male, aged 36 years; suffering from ANÆSTHETIC LEPROSY from infancy.

Present condition.—Copper coloured anæsthetic patches on both cheeks and on the neck. Front and back of the trunk are covered with similar circular patches, much lighter than the natural skin, attended by loss of sensation. Similar patches on both upper arms; fingers of both hands exhibiting signs of contraction; little fingers of both hands are already slightly contracted. The buttocks and both lower extremities are covered with similar kind of patches, attended by loss of sensation. Toes of both

feet are swollen, and there is a characteristic deep ulcer under the great toe of right foot.

Total anæsthesia in patches over the trunk and in both extremities.

July 7th.—Salol, gr. xx, three times a day.

August 3rd.—Gaining sensation in the fingers of both hands.

Treatment continued.

August 10th.—No change.

Salol, gr. xxx, three times a day.

August 18th.—Copper coloured anæsthetic patches all over body, several of them becoming darker and regaining sensation.

Treatment continued.

August 27th.—No further improvement.

Treatment continued.

CASE V.—Muhammadan, male, aged 20 years; suffering from TUBERCULAR LEPROSY for last four years.

Present condition.—Slight thickening of the skin of the nose and lobes of both ears; discoloration and desquamation of skin of both upper extremities; peculiar shiny appearance and dark patches on both the legs; both feet slightly swollen; toes of both feet present a peculiar shiny appearance. No loss of sensation anywhere.

July 26th.—Salol, gr. xx., three times a day.

August 3rd.—He complains of muscular pains all over the body which prevent him from sleeping during the night.

Salol, gr. xx, three times a day.

August 18th.—No improvement.

August 21st.—He absconded.

CASE VI.—Muhammadan, male, aged 20 years; suffering from MIXED LEPROSY for last ten years.

Present condition.—Minute tubercles on borders and lobes of both ears, discoloration and slight thickening of the skin of the face. Scaly dark patches on both forearms; fingers of both hands exhibiting signs of contraction, discoloration and dark patches in both lower extremities and a peculiar shiny appearance over shins of both legs; toes of both feet

slightly swollen; nails wasting away. Discoloration of the skin of the trunk; anæsthesia in both upper and lower extremities.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Improvement not to be seen.

Treatment continued.

August 10th.—Tubercles on the ears ulcerating.

Salol, gr. xx, three times a day.

August 18th.—Tubercles on the ears still sore; those of the forehead getting smaller in size.

Treatment continued.

CASE VII.—Hindu, male, aged 30 years; suffering from MIXED LEPROSY for last five years.

Present condition.—Tubercular thickening of the skin of the whole face, minute tubercles on both ears and nose; nose sunk in. Discoloration of the skin of the trunk, and the latter covered with psoriasis. Discoloration and slight thickening of the skin of the upper extremities, which are also covered with psoriasis. The little fingers of both hands are contracted; fingers swollen. Discoloration and slight thickening of the skin of both lower extremities; dark peculiar shiny patches on both shins, and tubercles around both ankles. Joints of both feet swollen, as well as toes; nails of toes wasting away. Loss of sensation over both the upper and lower extremities.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Gaining sensation in both extremities; minute tubercles on both ears and nose absorbed; thickening of the skin remains.

August 10th.—Gaining sensation in the extremities; psoriasis is fading away; thickening of the skin of the face gradually going down; minute tubercles on the ears subsided, but thickening of the skin remains; skin is moist and not so dry as before.

He is much better.

Salol, gr. xxx, three times a day.

CASE VIII.—Hindu, male, aged 18 years; suffering from TUBERCULAR LEPROSY from infancy.

Present condition.—Tubercular thickening of the lobes and pinnæ of both ears; cheeks, chin and nose also affected with a mass of tubercular

thickening. Patches of tubercular thickening over both the front and back of the trunk. Minute tubercles on both forearms; and fingers of both hands swollen. Discoloration and slight thickening of the skin of both lower extremities; dark and peculiarly shiny appearance of both shins and feet. No loss of sensation anywhere.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Several tubercles on the body and extremities have been absorbed; tubercular thickening of the face gradually going down.

August 10th.—Tubercles on the body and forearms have been greatly absorbed; tubercular thickening of the face is gradually going down.

Salol, gr. xxx, three times a day.

August 18th.—Fresh tubercles appearing on both upper extremities.

Treatment continued.

August 27th.—No further improvement.

CASE IX.—Hindu, female, aged 20 years; suffering from TUBERCULAR LEPROSY for last five years.

Present condition.—The whole of the face covered with minute tubercles; lobes of both ears and nose tuberculated. Both upper extremities covered with minute tubercles and skin slightly thickened. Front and back of the trunk also covered with similar tubercles; slight thickening of the skin of both lower extremities; peculiar dark shiny appearance of both shins. No loss of sensation anywhere.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Tubercles on the face and upper extremities have a tendency to subside.

August 10th.—Many of the tubercles on the face, trunk, and extremities have subsided; dark patches on the shins are lighter.

Salol, gr. xxx, three times a day.

August 18th.—A mass of fresh tubercles has appeared over face and extremities.

Salol, gr. xxx, three times a day.

August 27th.—Some of the tubercles on the arms have subsided and fresh ones have appeared.

Treatment continued.

CASE X.—Native Christian, female, aged 14 years; suffering from MIXED LEPROSY for last two years.

Present condition.—Lobes and borders of both ears covered with tubercles; chin, nose, and whole of the face covered with minute tubercles. Slight thickening of the skin of both upper extremities; fourth and fifth fingers of right hand and little finger of left hand slightly contracted. Discoloration of the skin of the trunk and of lower extremities. Dark peculiar shiny patches on both shins; feet somewhat swollen. Left fourth toe and right second toe ulcerated. Complete anæsthesia of both lower extremities.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—Gaining sensation in both lower extremities.

Treatment continued.

August 10th.—Improvement very slight.

Treatment continued.

August 19th.—Itchy sensation all over the body.

Treatment continued.

August 27th.—She is getting good appetite; no further improvement.

CASE XI.—Hindu, female, aged 12 years; suffering from TUBERCULAR LEPROSY for last two years.

Present condition.—Cluster of tubercles on the nose, chin, and ears; skin of the whole face covered with minute tubercles. Both upper extremities covered with tubercles; a large tubercle situated on the dorsal aspect of right middle finger; fingers of both hands slightly swollen. Discoloration of the skin of the trunk. Dark shiny patches on both feet; toes thickened; nails gradually wasting away; ulcer on the under surface of right second toe. No loss of sensation anywhere.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—No change.

Treatment continued.

August 10th.—Tubercles on the face are slightly depressed.

Salol, gr. xxx, three times a day.

August 18th.—Complains of severe pain over abdomen; tongue sore; ulcer on the right foot getting worse.

August 27th.—No improvement. As she is suffering from dysentery the medicine is stopped.

CASE XII.—Native Christian, female, aged 15 years; suffering from TUBERCULAR LEPROSY for one year.

Present condition.—Skin of face somewhat shiny, and lobes of ears slightly thickened. A raised eruptive tubercular patch on the back, also on right upper arm, left forearm and left thigh. No loss of sensation over these maculæ. Peculiar shiny scaly appearance of both shins.

July 26th.—Salol, gr. xx, three times a day.

August 3rd.—No change.

Treatment continued.

August 8th.—Eruption has a tendency to subside; *there is some improvement.*

Salol, gr. xxx, three times a day.

August 8th.—Eruption is getting less and the thickening decreased.

Treatment continued.

August 27th.—There is some itching over the eruptive patches.

Treatment continued.

ADDENDUM.

Since the return of the Commissioners to England Dr. Cook has continued the above treatment, but apparently without success, as may be gathered from his letter.

GOVERNMENT LEPER HOSPITAL,

Madras, 11th November 1891.

From—Surgeon-Major H. D. COOK, M.B., Supdt., Govt. Leper Hospital, Madras;
To—A. A. KANTHACK, Esq., F.R.C.S., St. John's College, Cambridge.

In continuation of my letter of 31st August 1891, I have to inform you that the salol treatment was continued up to 12th October 1891, thereby terminating the three months' treatment suggested by me—a trial sufficiently long to evince the virtue of Salol as a specific in leprosy. A few

of the twelve cases under treatment derived some benefit, while the others improved in parts previously affected, but have broken out in parts formerly unaffected. Tubercles have subsided and already existing ulcers healed up, but after some time fresh tubercles and ulcers appeared. In addition patients complained of loss of appetite, lassitude and a disagreeable burning sensation in the stomach, eyes; in fact throughout the body. Considering that leprosy is a disease, requiring a steady persevering course of treatment, extending over a period of three months, if necessary a year, a specific that would recommend itself and combat successfully with the disease must necessarily possess properties of a progressively beneficial nature, so that its continued use would have no disagreeable counteraction; it must have tonic and alterative properties, and be such that its use could be continued for a long time without the least ill-effect. Salol in my estimation is of no therapeutic value in leprosy; in fact, I consider it a *decided failure.*

APPENDIX II.

EXPERIMENTS ON THE ACTION OF TUBERCULIN IN LEPROSY.

In connection with the treatment of leprosy mention must be made of a series of experiments with Koch's fluid which Surgeon-Major H. D. Cook of Madras kindly performed for the Commission. Twelve lepers were treated with tuberculin, obtained from Dr. A. Libbertz of Berlin. The observations were made, with but faint hope of achieving any therapeutic success, but rather to test the diagnostic value of the medium. For assuming tuberculin to be a specific in tubercular affection, it could *à priori* scarcely be considered probable that as such it could have been of any use in leprosy, even though one must allow a certain pathological resemblance between the two diseases. Besides, by the time the tuberculin reached India the majority of men expressed their doubt as to its efficacy even in tubercular disease. Various physicians,[1] notably amongst them Dr. P. S. Abraham, Dr. Arning, Dr. Goldschmidt and Drs. Babés and Kalindero have tried the injection of Koch's fluid, but in no case has there been any permanent improvement, and hardly even any temporary benefit. Difference of opinion has been expressed as to value as a diagnostic means of tuberculin, that is, whether or no the injection of tuberculin is followed by a general and local reaction of a characteristic type. It was especially to test this point that the Commission requested Dr. Cook to try the effect of tuberculin on a number of lepers, carefully selected, free from any suspicion of tubercular disease, representing the various types of leprosy. No advanced cases were employed.

Dr. Cook's detailed report will be found appended. Here a short extract, with the conclusions based on the experiments, will be given. The patients were all male Hindus, young adults suffering from leprosy in its less advanced stages. Four were affected with the anæsthetic form of the disease, four with tubercular leprosy, and the remaining four with the mixed form. Each patient was observed for a few days previous to the commencement of the injections and the day before, the temperature taken at morning, noon, and night. Then a minimal dose of tuber-

(1) Journal of the Leprosy Investigation Committee, No. 2, February 1890, page 110, *et seq.*

culin was injected (·001cc), sometimes into an apparently healthy part, at other times into the diseased tissues. An interval of twenty-four hours always elapsed between two successive injections, and to begin with, the dose was increased by ·001cc until ·005 was reached, when it was ventured to increase it by ·002cc; ·01 was the maximum dose ever given. The temperature was carefully observed every three hours, and whenever it rose above 102·5° the dose was not increased, but the same or a smaller dose given, until the temperature remained at or below 102·5° for some time. Injections were never continued, until the phenomena of reaction had abated.

No case was in any way, generally or locally, improved, although the treatment was continued for at least three weeks. One or two patients were decidedly the worse for the injections, being reduced in general health. In two instances the patients acknowledged a return of sensation into a primarily anæsthetic region, but this passed off quickly. In another case several ulcers healed up and remained so at the time the treatment was discontinued; in one instance tubercles in the face became less prominent. But with these exceptions the improvements were but slight and momentary, or altogether absent, so that the lepers lost all faith and patience. In four cases the use of tuberculin was followed by a general outbreak of psoriasis all over the body. These were of the mixed or tubercular type.

A general reaction was absent in two anæsthetic cases, one tubercular and one mixed case (or in four out of twelve cases). In seven cases,—one anæsthetic, three tubercular, and three mixed—it was well marked, while in one case it was but weak. The mere absence or presence of a general reaction, therefore, cannot be made the criterion between leprosy or tuberculosis, as was at one time assumed by Dr. Arning.[1] In Dr. Cook's patients tuberculosis was out of the question.

According to Professors V. Babés and Kalindero,[2] the general reaction in the case of leprosy essentially differs from that established in tuberculosis. There is no doubt from a perusal of Dr. Cook's cases that, in accordance with these authors, a stronger dose is required to produce a febrile reaction in leprosy than in tuberculosis. Again the twelve cases confirm

(1) Deutsche Med. Wochenschr, 1890, 50.

(2) Semaine Médicale. Jan. 26, 1891.

the statement of the same writers that in leprosy the general reaction, instead of in about six hours, does not ordinarily appear until twenty-four hours after injection. The cases offer, however, no evidence that the fever and concomitant symptoms last longer than in tuberculosis. But they may be said, on the whole, to confirm the other statement that after the first reaction a second appears on the following day, and at times even a third the day after (*cf.* Case XII). Local reactions failed in almost every case, the ulcers, as a matter of fact, healing in only one instance. Amelioration of the general condition was not observed, but a general weakness was not uncommon.

The Commissioners, therefore, agree with Professors Babés and Kalindero that there are essential differences in the character of the reactions in the case of leprosy and tuberculosis, respectively, where these are typical; and that in a number of cases (as far as it is allowed to argue from so small a number of cases), Koch's remedy might distinguish whether an affection is leprous or tubercular, provided it were proven that a typical reaction follows in all cases of tubercular, but never appears in healthy individuals. But they think that it can hardly be employed in the diagnosis of a suspicious trophoneurotic disease, seeing that of four anæsthetic cases no reaction occurred in two cases, while in a third case it was very weak. The most marked reactions occur in purely tubercular and mixed cases. The Commissioners also have no doubt that as far as leprosy is concerned, the remedy is of no therapeutic value, but, on the other hand, may not be altogether free from danger.

Table to show the Effect of Tuberculin in Leprosy.

Form of Disease.	Number of case.	Reaction.	Remarks.	Improvement.	Other changes.
ANÆSTHETIC .	(1)	Very weak—practically nil . .	What little reaction there was appeared only after large doses were given .	Nil	
	(2)	Practically nil .		,,	
	(3)	Weak . .		,,	
	(4)	Well marked .		,,	

Table to show the Effect of Tuberculin in Leprosy — contd.

Form of Disease.	Number of case.	Reaction.	Remarks.	Improvement.	Other changes.
TUBERCULAR .	(5)	Nil . . .		Nil	
	(6)	Well marked .		,,	
	(7)	Ditto . .		,,	Psoriasis.
	(8)	Ditto . .	when large doses given	,,	Ditto.
MIXED . .	(9)	Ditto . .		,,	Ditto.
	(10)	Practically nil .		,,	
	(11)	Well marked .		,,	
	(12)	Ditto . .	when large doses given	,,	Psoriasis.

SURGEON-MAJOR H. D. COOK'S REPORT ON TUBERCULIN.

GOVERNMENT LEPER HOSPITAL,

Madras, June 1891.

From—Surgeon-Major H. D. COOK, M.D., Supdt., Govt. Leper Hospital, Madras,
To—A. A. KANTHACK, Esq., F.R.C.S., New Club, Simla.

With reference to your letter dated Simla, 22nd April 1891, I have the honour to inform you that in accordance with the directions contained therein, regarding the use of "Tuberculin," I began the treatment with it on twelve lepers selected for the purpose on the 27th ultimo and continued it till the 20th instant.

A minute diagnosis of each of these twelve cases being recorded (as per cases attached), they were inoculated with solutions of tuberculin carefully prepared by the local Chemical Examiner to the minimum strength of ·001, and gradually increasing it at each successive injection performed every alternate day. When the temperature exceeded 102·5°, the minimum strength or one of less strengh than that used at the previous operation, as the case required, was injected, till it reached ·01cc.

From the careful observation of their temperatures with a clinical thermometer, every third hour, five times daily, and duly registered, the only

effects noticeable were an increase of temperature, only temporarily attaining a maximum of 105° in three cases, accompanied with fever; and an average of 100·5° in others, while in three of these cases the temperature slightly exceeded the normal.

These symptoms, unaccompanied with the least mitigation of the disease, were the only results attained, and as the trial given was sufficiently long to exhibit any virtue, if there be, of the remedy, I have decided to discontinue the treatment. The patients who were under treatment got disheartened and one of them got very ill. These cases were typical of the various forms of leprosy, carefully selected, and not in an advanced stage, and I may remark here that every detail of the whole treatment was strictly observed from 27th May till 20th June 1891. The injections and the registration of temperature have been scrupulously carried out, so that I can safely say a fair trial has been given.

From the above remarks it may be seen that so far no mitigation of the symptoms has occurred, and therefore I think the "Tuberculin" for leprosy is a decided failure. It may be said that the injection should be continued for a long time, but the patients would not submit to a painful treatment, seeing no good result therefrom.

The Resident Apothecary, Mr. M. Nur Mahomed, has taken great pains with these cases throughout. Recording temperatures every three hours in twelve cases is of itself an irksome duty, and my thanks are due to him for so cheerfully assisting me with the experiments.

SURGEON-MAJOR COOK'S DETAILED REPORT OF TWELVE CASES OF LEPROSY TREATED WITH TUBERCULIN.

Case I.—Hindu, male, aged 39 years; suffering from ANÆSTHETIC LEPROSY for last five years.

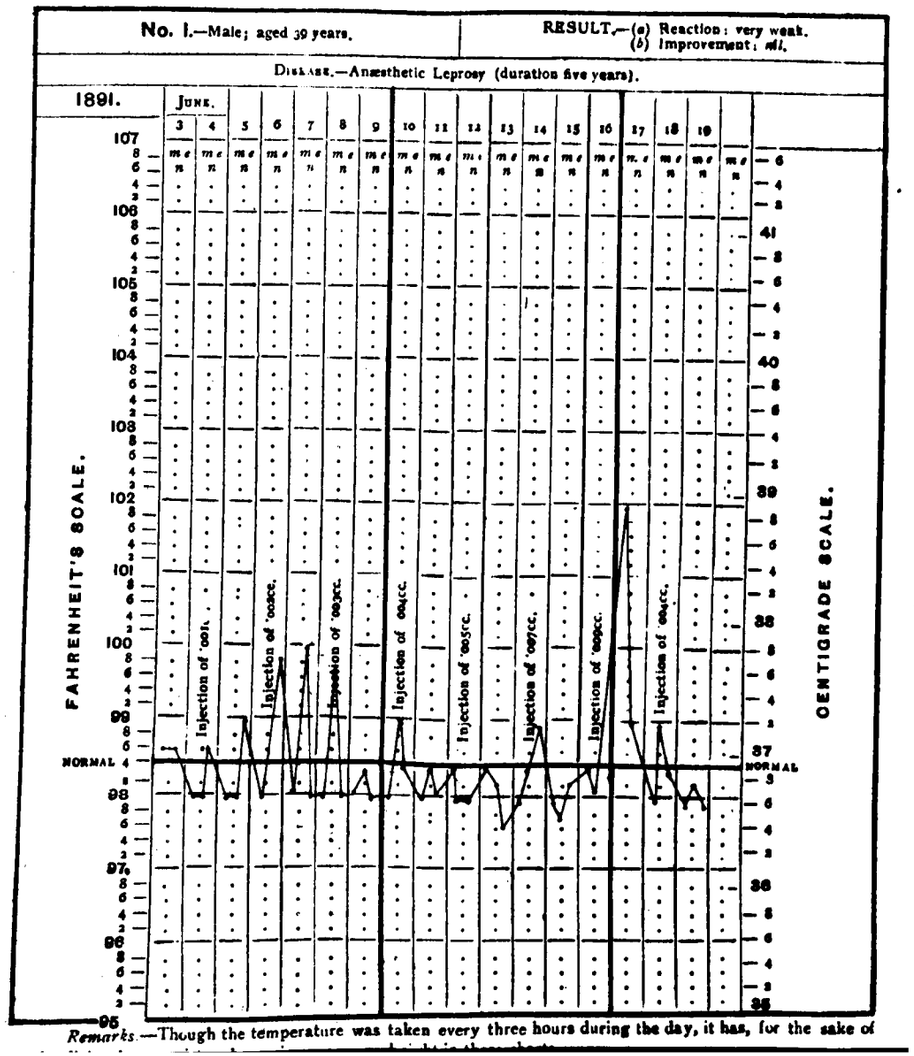
Condition on June 3rd: A circular light patch immediately above the right eyebrow; similar patches on the sides of the lower jaw. A large light-coloured anæsthetic patch on posterior surface of the left elbow extending downwards. A large anæsthetic patch surrounding the right knee and both legs. A small characteristic ulcer on the right heel. Complete anæsthesia of both legs.

June 4th: ·001cc. of tuberculin injected, and injections carried on until June 16th, when ·009cc. was injected.

Practically NO REACTION: a slight rise of temperature at the commencement, and a marked rise when the largest dose was administered.

Improvement: nil.

Weight on June 3rd: 108lb.
" " " *8th:* 94½lb.
" " " *14th:* 93lb.



Remarks.—Though the temperature was taken every three hours during the day, it has, for the sake of

Case II.—Hindu, male, aged 32 years; suffering from ANÆSTHETIC LEPROSY for last six months.

Condition on May 26th: Slightly raised patch on the forehead (macular) and face; a small patch on the right cheek; nose slightly thickened. Two small light patches a little above the right knee; a dark characteristic patch on dorsum of left foot.

Loss of co-ordination of movement of left leg; the foot falls when raised.

Slight anæsthesia of left leg and hyperæsthesia of face.

May 27th: ·001cc. injected, and the usual injections continued.

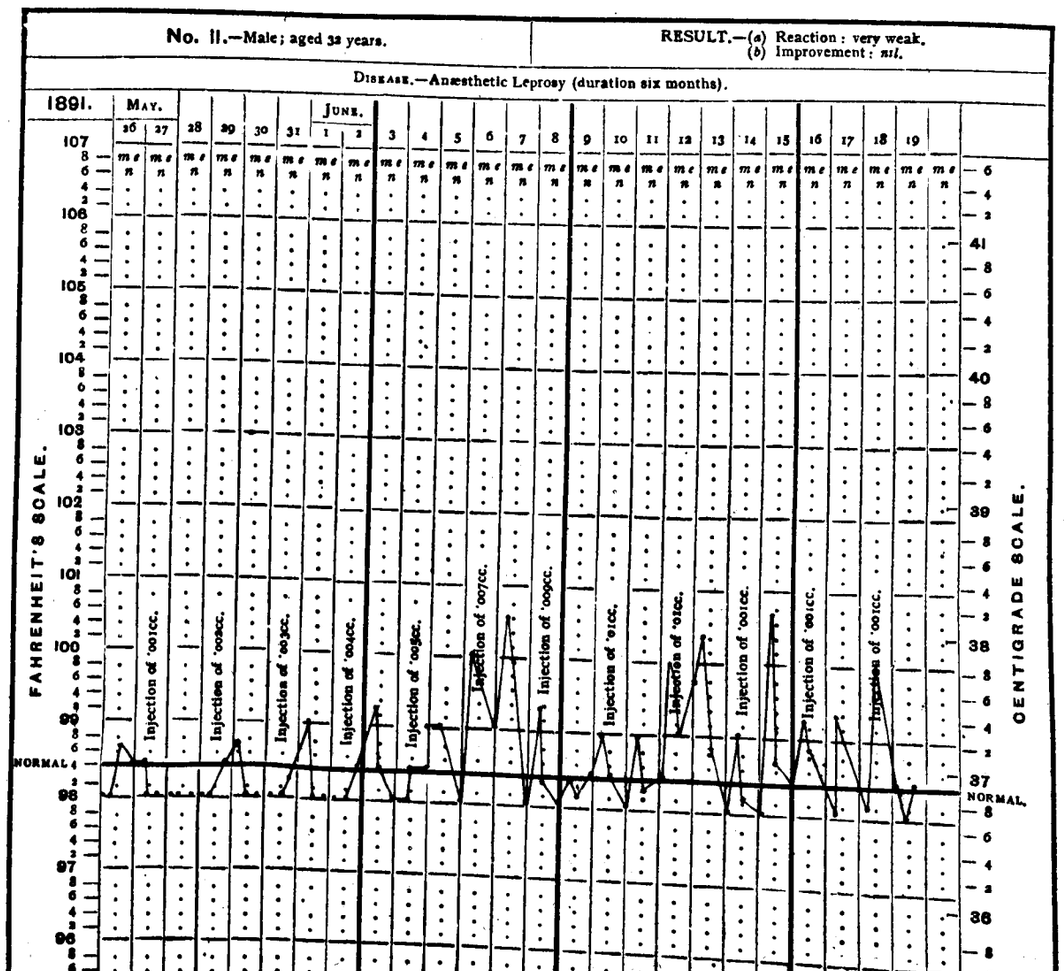
Hardly any Reaction, the temperature never rising above 101°, and then only after the maximum dose ·01 had been given for the second time.

Improvements: June 14th; decrease of anæsthesia of left foot, also a certain amount of increased power in that leg. Patches above right knee are paler.

June 19th: anæsthesia of left foot as before, and loss of strength also as before.

Thus no improvement.

Weight on May 27th: 150½lb.
" " *June 8th:* 154lb.
" " " *14th:* 150lb.



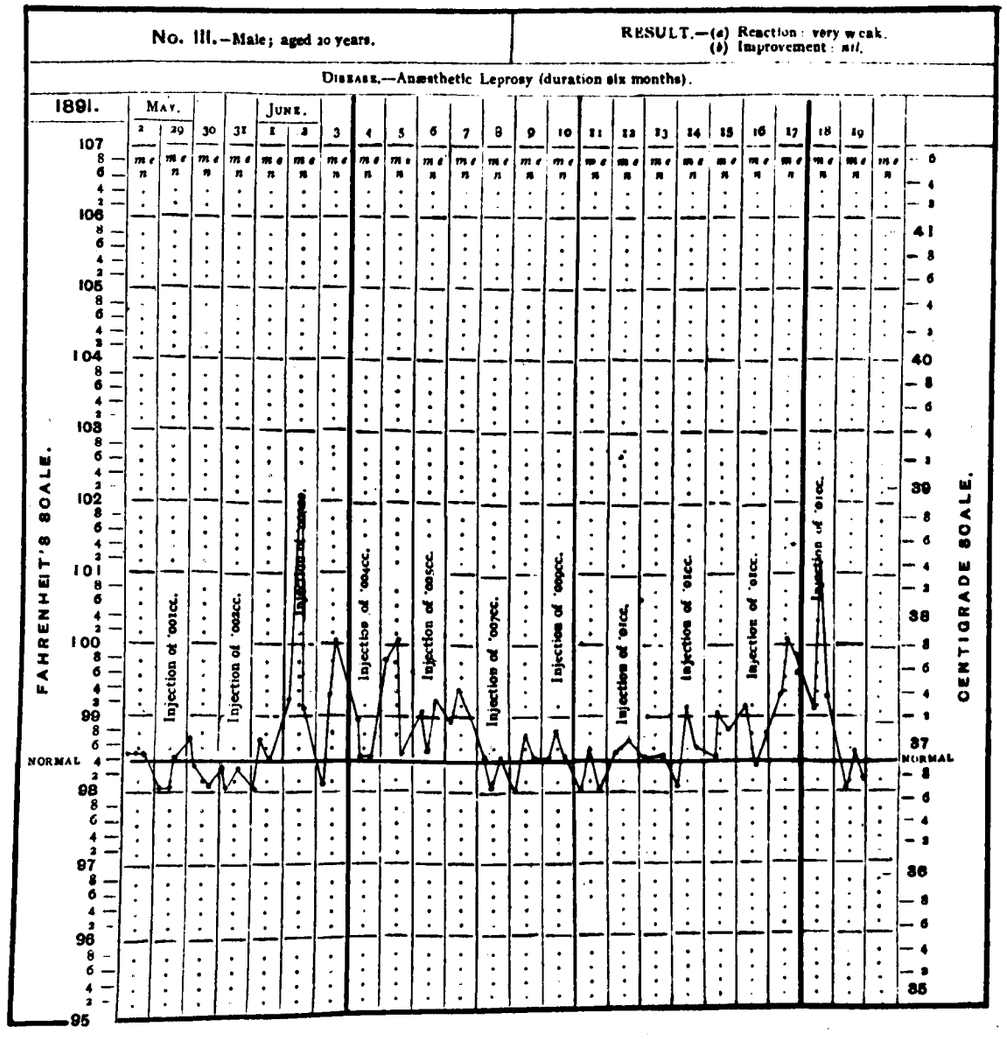
Case III.—Dutchman, male, aged 20 years; suffering from ANÆSTHETIC LEPROSY for six months.

Condition on May 28th: A well developed lad. Very slightly discoloured patches (fairer than his natural skin which is of an olive hue) on the left cheek and on the forehead. A dark patch (darker than natural skin) stretching from right arm to right breast. These patches are not anæsthetic. A raised irregular darkly coloured eruptive patch, raised above the level of the skin, on the abdomen — not anæsthetic. An eruptive anæsthetic patch on the right knee. Anæsthetic patches on right leg and foot.

May 29th: ·001cc. injected, and injections continued until June 18th. No reaction until ·003cc. was reached when temperature rose to 102°. Taking it all round the reaction was weak.

Improvement: nil.

Weight on May 28th: 108lb.
" " *June 8th:* 105lb.
" " " *14th:* 105½lb.
" " " *19th:* 105lb.



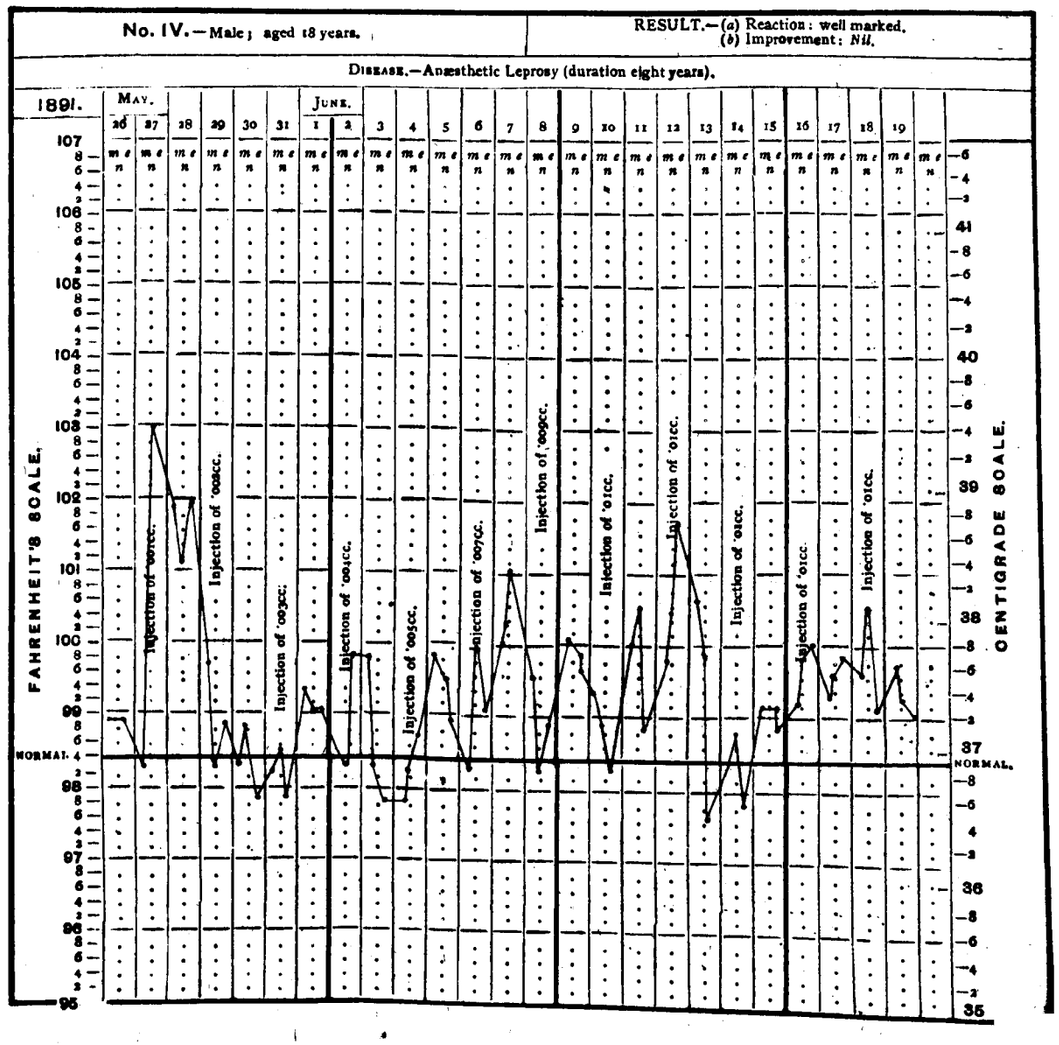
Case IV.—Hindu, male, aged 18 years; suffering from ANÆSTHETIC LEPROSY for eight years.

Condition on May 26th: Small light anæsthetic patch on right cheek and forehead. A large similar patch on the outer surface of left upper arm; another on the anterior aspect of left forearm from elbow downwards. Large light patches on the outer surface of both thighs and buttocks. Peculiar shiny appearance and discoloration of the skin of both shins. A small circular patch immediately below the umbilicus. Loss of sensation in both extremities.

May 27th: ·001 cc. injected, followed by considerable reaction. Injections continued up to ·01cc., and reaction showed itself fairly well throughout.

Improvement: nil.

Weight on May 27th: 75lb.
" " *June 8th;* 72½lb.
" " " *14th:* 72½lb.
" " " *19th:* 72½lb.



Case V.—Eurasian, male, aged 22 years; suffering from TUBERCULAR LEPROSY for ten years.

Condition on May 30th: Slight tubercular thickening of nose, lobes of both ears and chin, and tubercular thickening of the whole face. Dark patches over left shin. No anæsthesia.

May 31st: ·001 of tuberculin injected. The usual injections were carried on every other day, and the maximum dose given on June 14th.

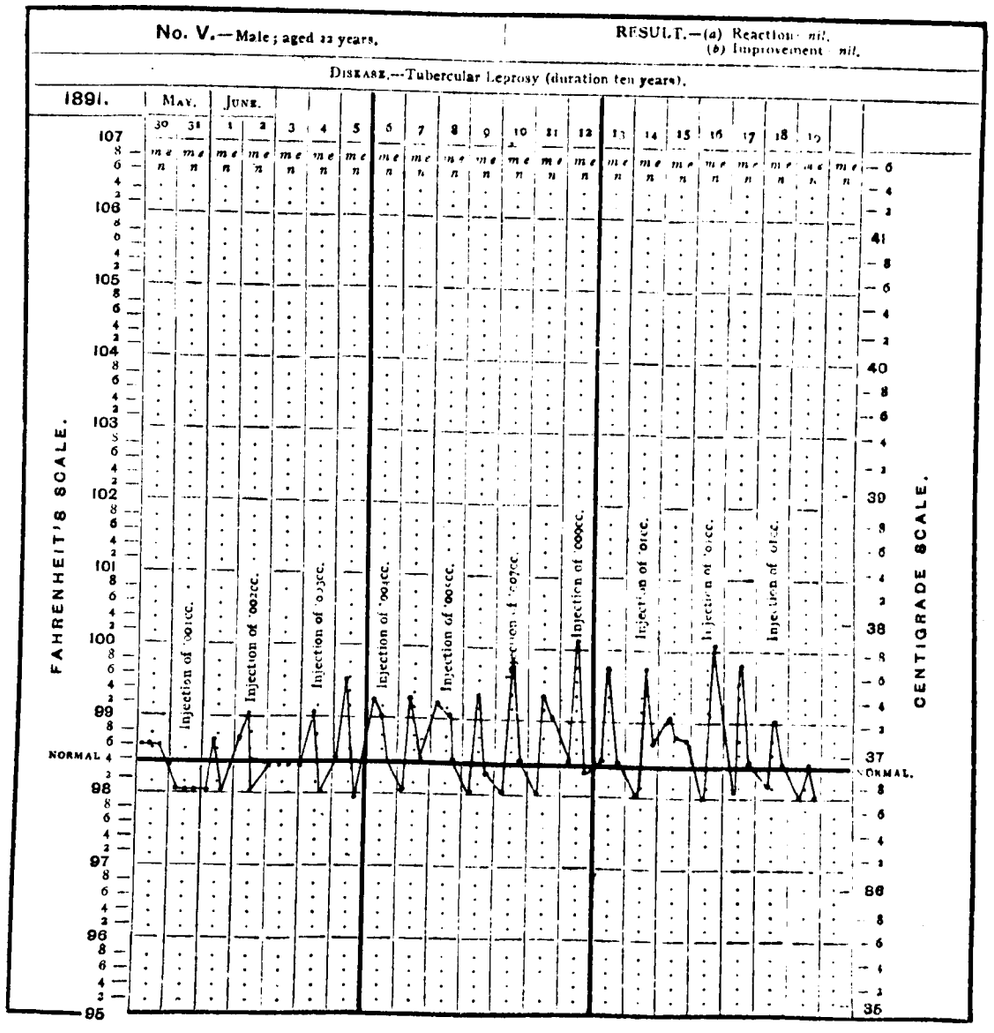
Reaction and rise of temperature practically *nil.*

Improvement: nil.

Weight on May 30th: 110lb.

" " *June 8th:* 106½lb.

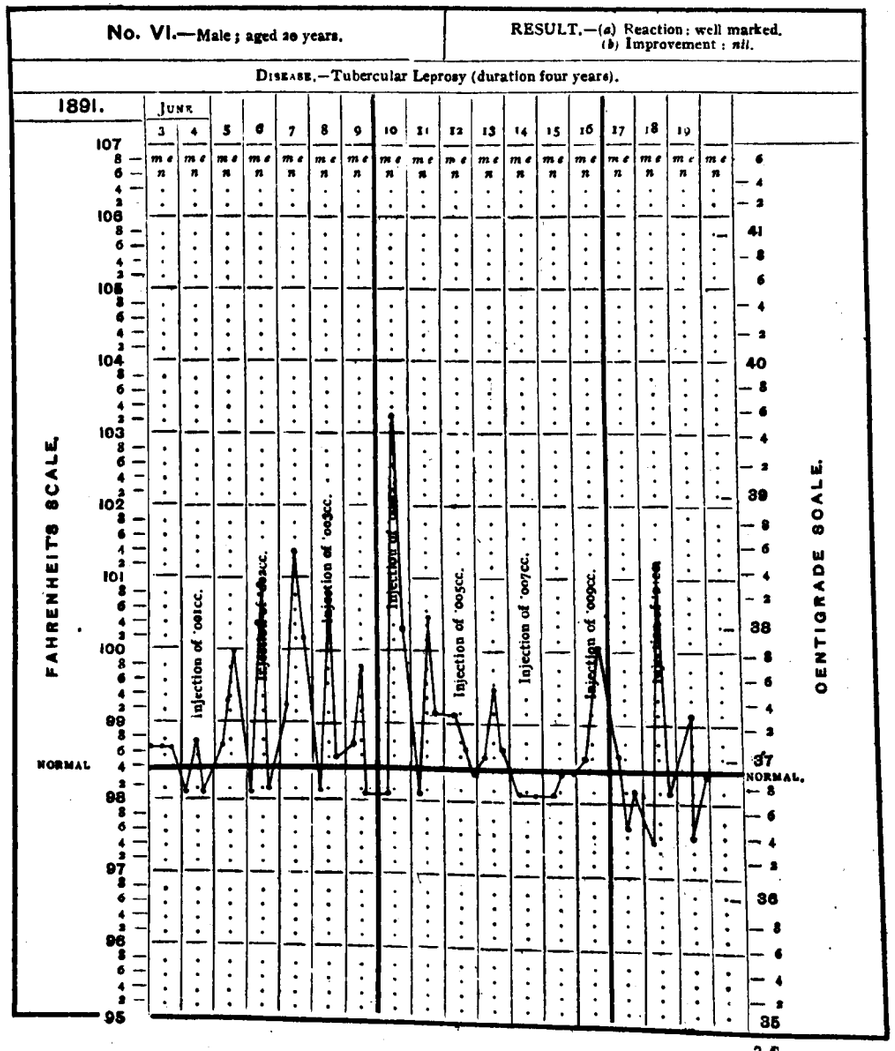
" " " *14th:* 108½lb.



Case VI.—Hindu, male, aged 20 years; suffering from TUBERCULAR LEPROSY; duration of disease: four years.

Condition on June 3rd: Tubercular thickening of lobes and pinnæ of both ears, also of nose and lips; general thickening of face. Discoloration of skin of trunk and extremities. Epidermis scaly and desquamating. Fingers of both hands slightly thickened. Peculiar dark discolorations over both shins. Toes of feet thickened. No anæsthesia.

June 4th: ·001cc. of tuberculin injected, and injections continued until ·009cc. was reached.
Reaction: Well marked.
Improvement: nil.
Weight on June 3rd: ·110lb.
" " " *8th:* ·101lb.
" " " *14th:* ·104lb.



Case VII.—Hindu, male, aged 28 years; suffering from TUBERCULAR LEPROSY for two and a half years.

Condition on June 1st: Tubercular thickening of lobes and pinnæ of both ears; minute tubercles on the edge of the nose. Fingers of both hands thickened. Dark patches over both shins; toes of feet thickened and nails wasting. Psoriasis all over body. No anæsthesia.

June 2nd: ·001cc. injected. When ·003cc. was reached on June 6th the temperature rose to 102·8°, and to 103° the next day.

Reaction has been well marked throughout.

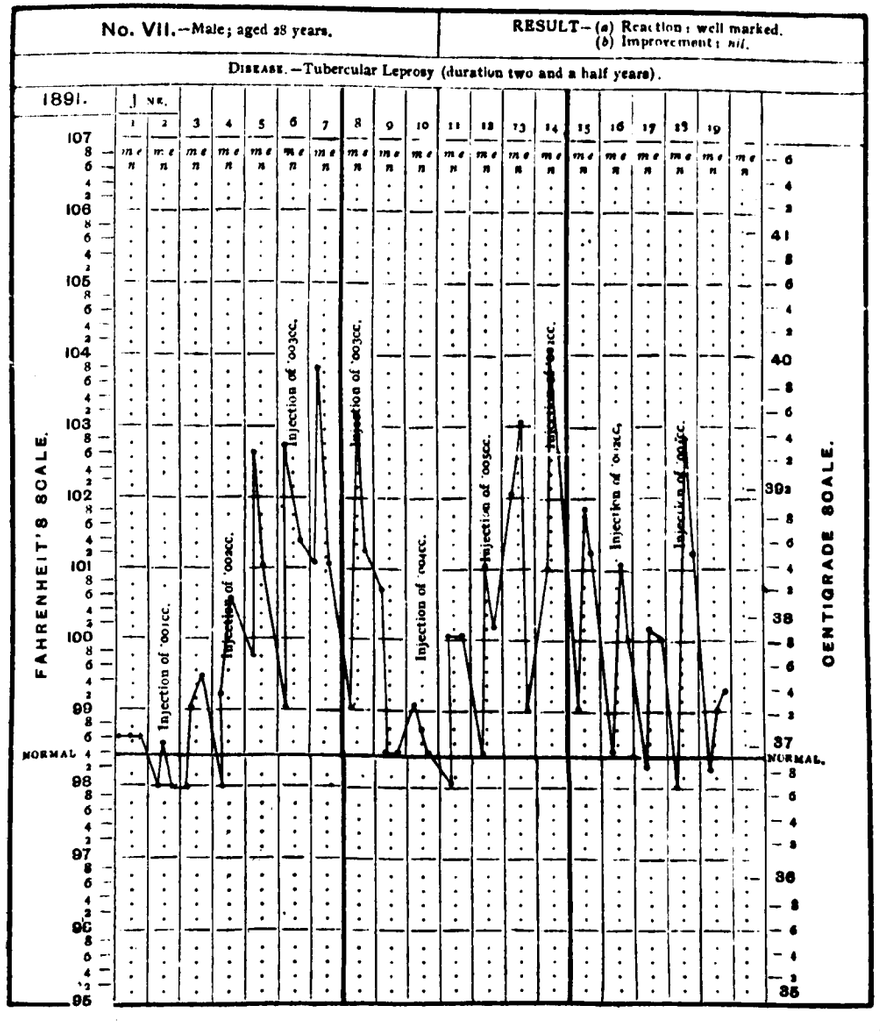
Improvement: nil.

June 19th: Psoriasis much aggravated (cf. Cases VIII, IX, and XII).

Weight on June 1st: 102lb.

,, ,, ,, *8th:* 102½lb.

,, ,, ,, *14th:* 99lb.



Case VIII.—Hindu, male, aged 22 years; suffering from TUBERCULAR LEPROSY; duration of disease: eight years.

Condition on May 28th: Minute tubercles on the chin, lips, nose, and lobes of both ears; slight thickening of whole face. Thickening and peculiar glossy appearance of fingers of both hands. Lower extremities discoloured and partially anæsthetic; toes of feet thickened; ulcer on great toe of left foot. Peculiar glossy appearance of skin over shins. Toes of both feet inclined to retract.

May 29th: ·001cc. injected, and usual injections continued. When ·005cc, was reached, signs of severe reaction appeared.

Reaction: Well marked with larger doses.

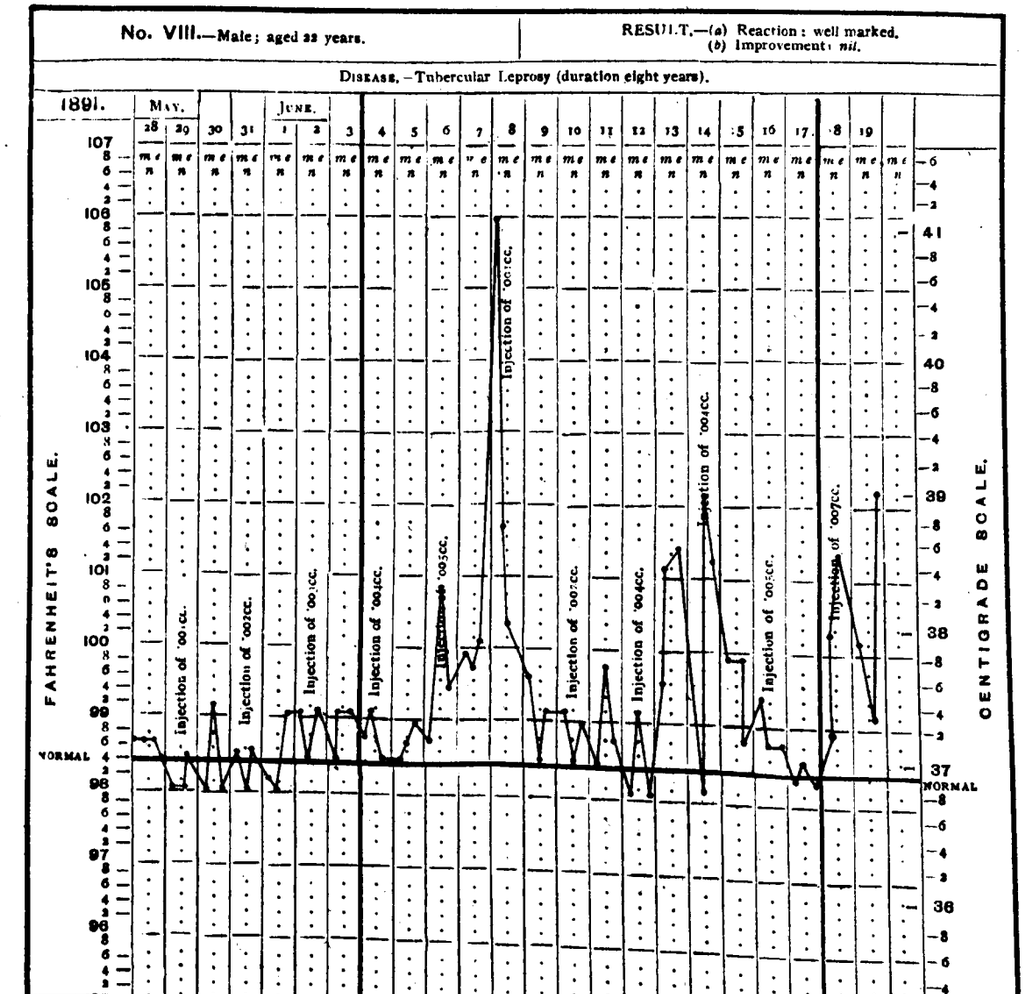
Improvement: At one time there was a decided diminution of the anæsthesia; but this passed off very quickly.

Psoriasis appeared all over the body (cf. Cases VII, IX, and XII).

Weight on May 28th: 119lb.

,, ,, *June 8th:* 115lb.

,, ,, ,, *14th:* 113lb.



Case XI.—Hindu, male, aged 25 years; suffering from MIXED LEPROSY; duration of disease: ten years.

Condition on May 26th: Tubercular thickening of the lobes of both ears, nose, chin; general thickening of face. Discoloration of the skin of the trunk and upper extremities. Fingers of right hand thickened; fourth and fifth fingers of left hand slightly contracted and atrophied; thickening of skin of both upper extremities. Light patches on both thighs and knees; dark patches on both legs, glossy in appearance. Anæsthesia of extremities.

May 27th: ·001cc, injected; when dose was increased to ·003cc, a good reaction appeared.

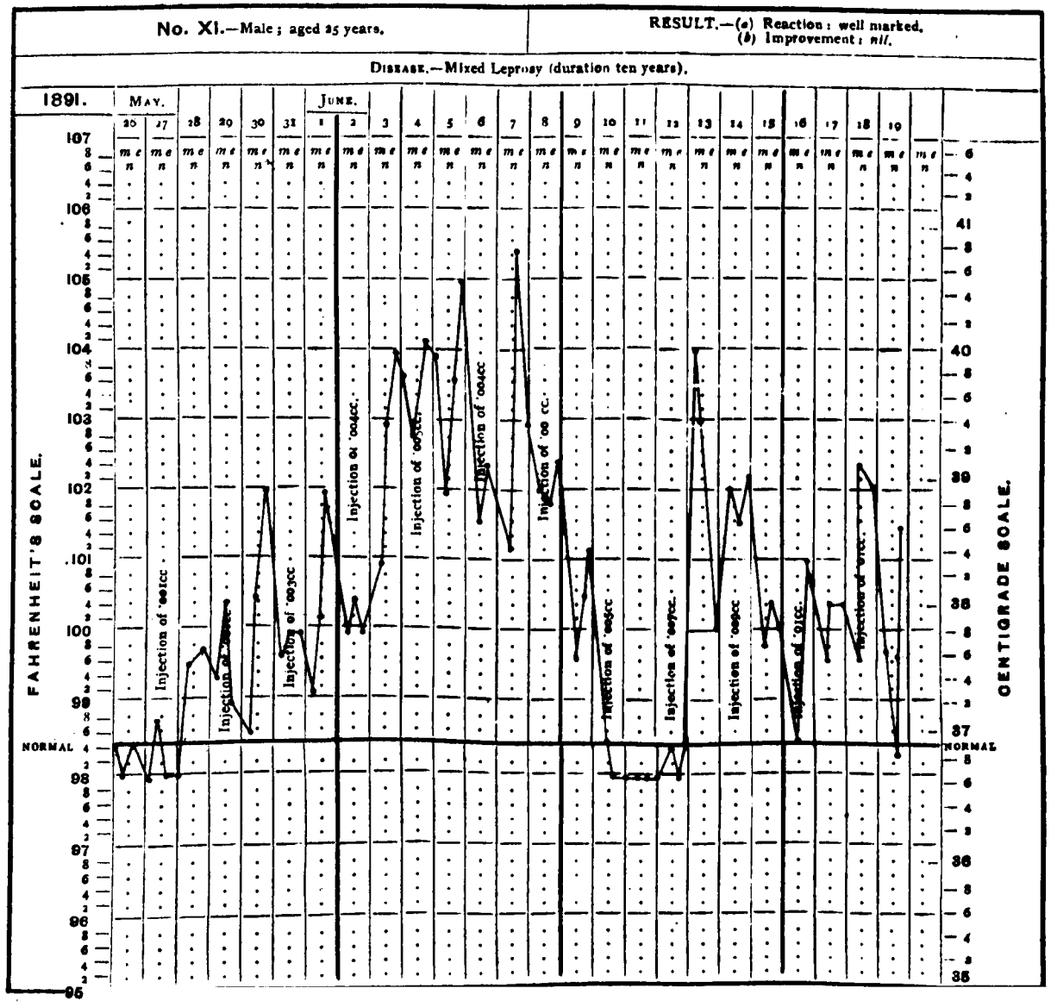
Reaction: well marked.

Improvement: nil; on the other hand, became seriously ill, complaining of pains all over his body and greatly reduced in general health.

Weight on May 27th: 106½lb.

,, ,, *June 8th:* 99½lb.

,, ,, ,, *14th:* 99lb.



Case XII.—Hindu, male, aged 18 years; suffering from MIXED LEPROSY; duration of disease: six years.

Condition on June 1st: Minute tubercles on the edges of both pinnæ; slight tubercular thickening of lobes of ears and nose; discoloration of skin of face. Light anæsthetic patches on both arms; discoloration of skin of both forearms; fingers thickened; fingers of right hand slightly contracted, and also those of left hand. Dark patches above left nipple. Anæsthetic patches on both legs; glossy appearance of skin over both shins; second toe of right foot slightly contracted; second toe of left foot thickened. Total anæsthesia of upper extremities and of both legs.

June 2nd: ·001cc, injected, and usual injections given until ·005cc, was reached, and the temperature began to rise to 103°. Injections reduced then.

June 12th: ·004cc, was injected, but finding that the temperature rose up to 105°, the injections were stopped.

Reaction: Well marked, when ·005cc, was given.

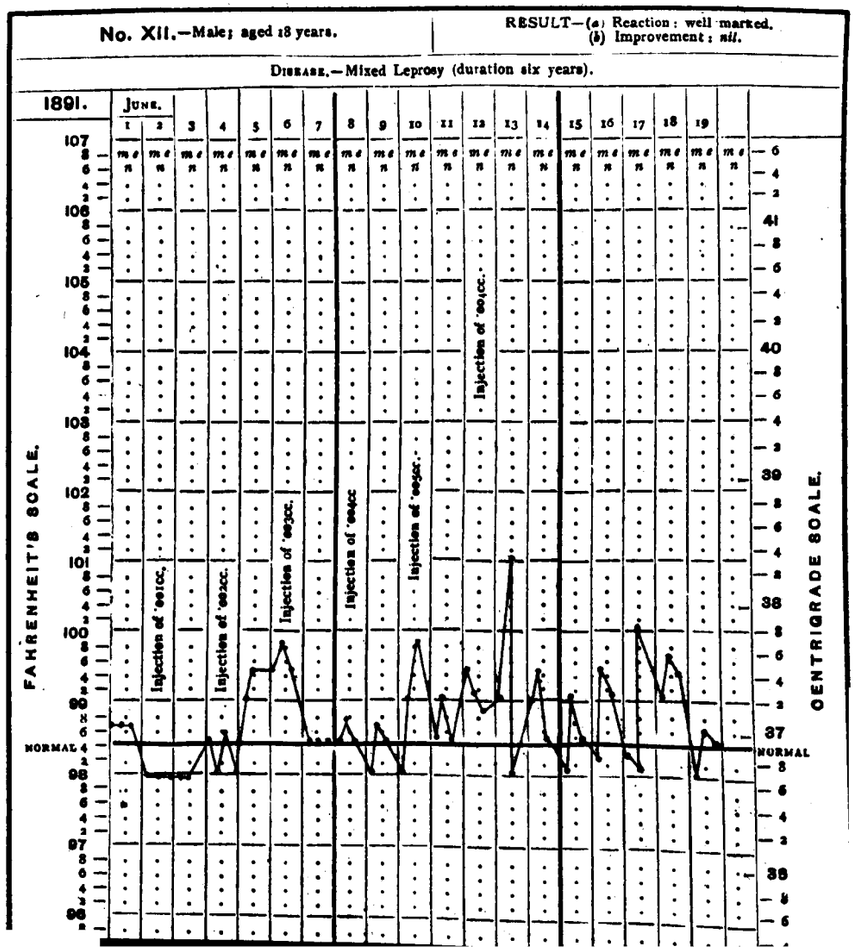
Improvement: nil.

Psoriasis again appeared all over the body (cf. Cases VII, VIII and IX).

Weight on June 1st: 83½lb.

,, ,, ,, *8th:* 84lb.

,, ,, ,, *14th:* 80lb.



Case IX.—Hindu, male, aged 21 years; suffering from MIXED LEPROSY; duration of disease: seven years.

Condition on May 26th: Minute tubercles on chin, lips, nose; thickening of nose; tubercles on eyebrows and middle of forehead; general thickening of face; lobes of ears thickened and nodular. Fingers of both hands thickened and glossy. A big light patch a little above the left knee, which is more or less anæsthetic. Skin over shins glossy and discoloured. Both feet swollen; three inner toes of right foot, and the great and third toes of left foot ulcerated; nails atrophied. Total anæsthesia of left upper arm and left thigh.

May 27th: ·001cc. injected. The dose was never increased beyond ·004cc. on account of the rise of temperature.

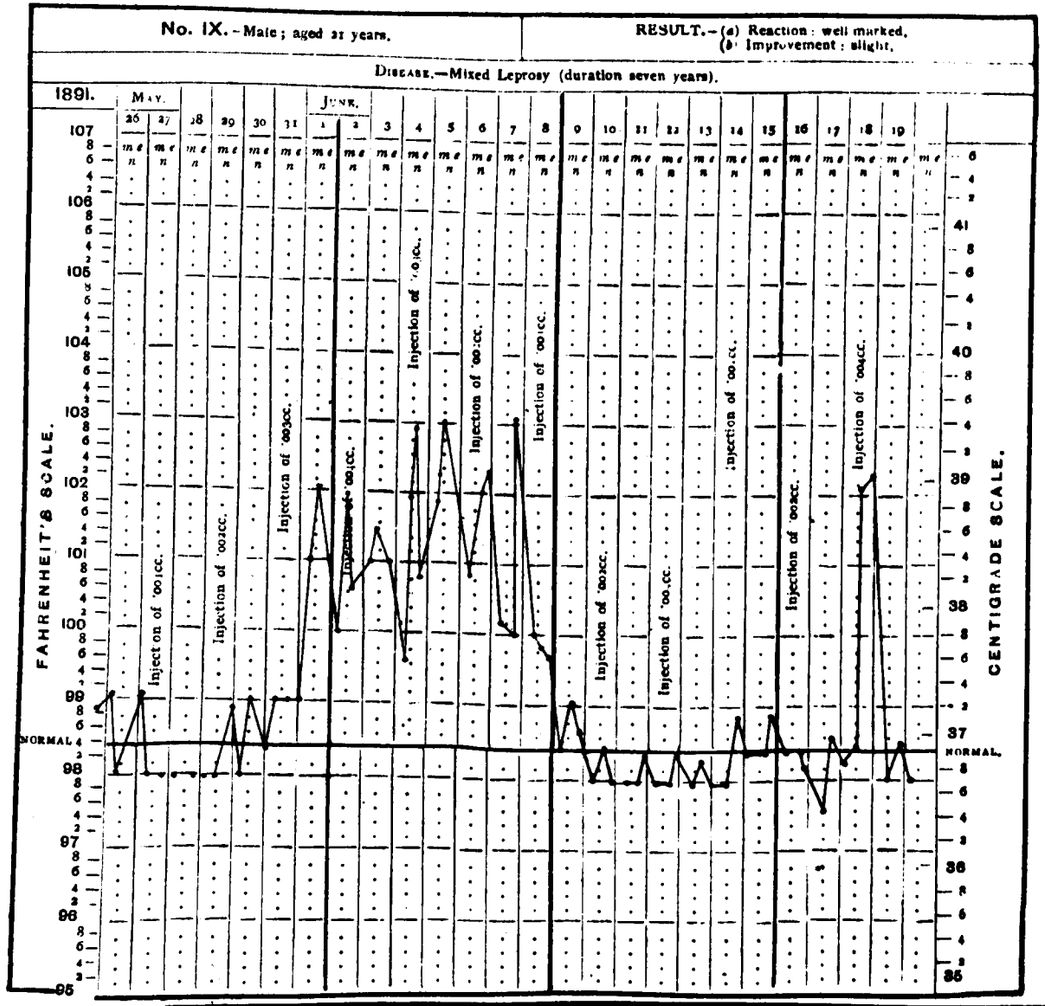
June 12th: There seems to be some improvement: the ulcers on the toes are healed, the swelling of the feet subsided; the anæsthetic patch on left thigh is regaining sensation; the tubercles on the face are not so prominent.

June 19th: The anæsthetic patch has again lost its sensation and the feet are as swollen as before. *Psoriasis* appeared all over the body cf.) Cases VII, VIII, and XII).

Reaction: Well marked.

Improvement: Slight and of passing nature.

Weight on May 26th: 94lb.
" " *June 8th:* 93lb.
" " " *14th:* 83lb.



Case X.—Hindu, male, aged 25 years; suffering from MIXED LEPROSY; duration: seven years.

Condition on June 1st: Tubercular thickening of lobes and pinnæ of both ears; nose very nodular; general thickening of face. Skin over whole of trunk discoloured. Mammary glands much enlarged. Raised anæsthetic patches on both lower extremities. Fingers thickened; left fingers retracted. Total anæsthesia of the extremities.

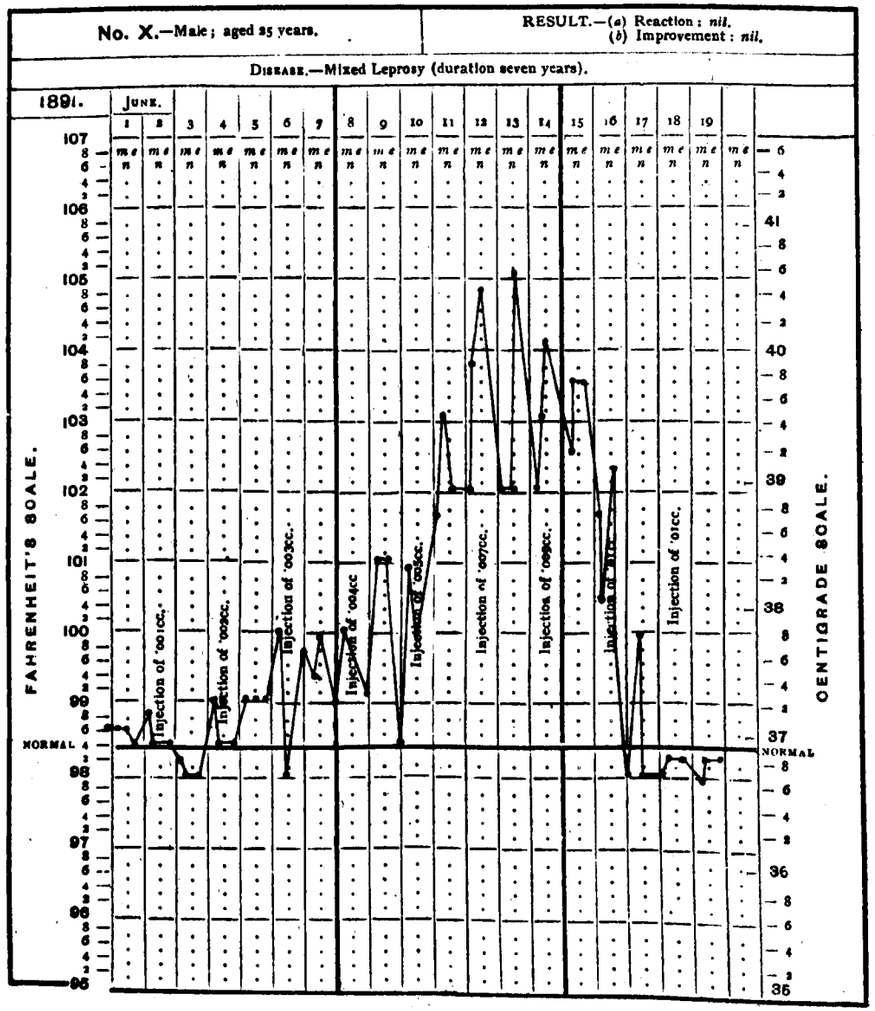
June 2nd: ·001cc. injected. The usual injections administered every other day until ·007 was reached and the temperature rose to 101°.

June 16th, the maximum dose was given and temperature did not rise above 101°.

Reaction: practically nil.

Improvement: nil.

Weight on June 1st: 100lb.
" " " *8th:* 91lb.
" " " *14th:* 100lb.



CONCLUSIONS.

The following conclusions are based upon the observations and arguments contained in the foregoing report:—

1. Leprosy is a disease *sui generis;* it is not a form of syphilis or tuberculosis, but has striking ætiological analogies with the latter.
2. Leprosy is not diffused by hereditary transmission, and for this reason, and the established amount of sterility among lepers, the disease has a natural tendency to die out.
3. Though in a scientific classification of diseases leprosy must be regarded as contagious and also inoculable, yet the extent to which it is propagated by these means is exceedingly small.
4. Leprosy is not directly originated by the use of any particular article of food, nor by any climatic or telluric conditions, nor by insanitary surroundings; neither does it peculiarly affect any race or caste.
5. Leprosy is indirectly influenced by insanitary surroundings, such as poverty, bad food, or deficient drainage or ventilation, for these by causing a predisposition increase the susceptibility of the individual to the disease.
6. Leprosy in the great majority of cases originates *de novo*, that is, from a sequence or concurrence of causes and conditions, dealt with in the report, and which are related to each other in ways at present imperfectly known.

Practical Suggestions.

Segregation may be voluntary or compulsory, and in either instance partial or complete. Complete segregation has never yet been possible. Both in the Sandwich Islands and in Norway it has failed.

"It appears that there has always been great difficulty in the Sandwich Islands[1] in carrying out a full measure of segregation. This fact comes out in several of the reports; and in the latest of them we see it stated that for some period during the administration as President of the Board of Health of Mr. Gibson, who apparently doubted its efficacy, the work of segregation had been practically brought to an end. At the present time there is an endeavour to enforce rigorously the isolation of lepers."

Again, "the isolation of lepers in the Norway[2] asylums is not absolute. The doors and gates of the institutions are not locked, and more than once I met some of the inmates in the neighbouring roads. The doctors informed me that they allowed those to go out who showed no ulcerations, and that no cases of consequent infection have ever been traced. The lepers, however, are usually kept in on market days, and at Trondhjem at least, and I believe elsewhere, they are not permitted to enter houses and churches, and to come in close contact with other people."

"To attribute the decline of leprosy in Norway[3] to compul-

(1) P. S. Abraham. Leprosy: A Review of some Facts and Figures. Epidemiological Society's Transactions, Vol. VIII., page 132.
(2) Op. cit., page 136.
(3) W. J. Collins. Note on the Leprosy Revival, Lancet, May 17, 1890, page 1064.

sory isolation is entirely erroneous. In the first place no such powers exist or are likely to be sanctioned by the Norse democracy; if they did exist it would be impossible without further accommodation to segregate even the reduced number of lepers in Norway at the present time. Indeed, I met many lepers in the streets of Bergen and on the quay going about their usual vocations. It is not that segregation is stamping out leprosy in Norway, but the increased material prosperity of the people, the growth of foreign trade, the intercommunication with town life, and the opportunities these give for better and more varied subsistence, which have doubtless effected beneficent changes in this direction."

The amount of legislation which at present exists in Norway may be best expressed in the following words of Dr. G. A. Hansen[4]: "Though the disease was steadily diminishing I found that something ought to be done to check the spread of the disease in the country, besides what might be done by the entrance of lepers into our asylums. In consequence I proposed a law which was enacted in 1885. This law gives the Sanitary Commission or Board of Health in each district the right to order a leper, if he will live at home, that he must have his own room, at least his own bed, that his clothes ought to be washed, separately, that he must have his own eating apparatus,—spoon, fork, knife, etc. If he cannot, or will not comply with this regimen, he is obliged to enter an asylum."

From the above quotations it is at once evident that even in the two countries in which segregation of lepers is commonly believed to be complete, the actual procedure falls far short of this. For India complete compulsory segregation may be considered to be

(4) G. A. Hansen. Journal of the Leprosy Investigation Committee, No. 2, pages 65-66.

absolutely impracticable. Neither do the conclusions given above as to the nature of the disease justify any recommendation for absolute segregation. The presence of a leper in a healthy community is a source of danger no greater than the presence of an individual suffering from tuberculosis. Both diseases are contagious in an equal and minimal degree. The amount of ulceration which exists in both diseases is to some extent a measure of the danger of contagion.

It is impossible for the same reasons to advise compulsory partial isolation. Voluntary isolation is, therefore, the only measure left for consideration. Among civilised communities the separation of those suffering from many diseases other than leprosy is encouraged. The voluntary isolation of the leper is, therefore, no exception to this custom. For this reason the Commissioners recommend the adoption of a voluntary isolation as extensive as local circumstances allow. Further, by permitting marriages among lepers, the plan suggested might be the more easily carried out.

In accordance with instructions received from the Committee of the National Leprosy Fund, the Commissioners are required in their final report to describe minutely what they believe to be the best plans for ensuring the efficient carrying out in practice of their recommendations relating to the treatment of lepers.

Conformably with these directions the Commissioners, though they are fully aware that the extent and varying social conditions of the empire may present great practical difficulties, venture to suggest the following schemes.

The Commission are of opinion that the sale of articles of food and drink by lepers should be prohibited, and that they should be prevented from practising prostitution, and from following such occupa-

tions, as those of barber and washerman, which concern the food, drink, and clothing of the people generally, quite apart from the dread of a possible infection.

Vagrant and indigent lepers living in the villages and scattered about the country are probably sources of little or no danger. Disgust, and to a certain extent fear, rarely permit their relations to the general community to be intimate, and no harm is likely to result from their soliciting alms by the roadside. The tendency at the present time, however, is for mendicant lepers to leave their homes, to crowd into the large centres of population, and this in the opinion of the Commission should be discouraged. In the cities and towns they herd together, living under circumstances of extreme poverty and filth, and forming communities which are not only offensive to public decency, but constitute from many points of view a menace to the public health. Moreover, in such leper colonies promiscuous and casual alliances are the rule, and the result is that children are born to struggle for existence under circumstances most painful to contemplate. The Commission consider that the best policy in dealing with this matter is to discourage this concentration of lepers in towns and cities, and to this end would suggest that municipal authorities be empowered to pass by-laws preventing vagrants suffering from loathsome diseases from begging in or frequenting places of public resort, or using public conveyances.

The large Presidency towns and the capitals of provinces in many cases already possess leper asylums, which might be enlarged by municipal funds or private subscriptions. Asylums should be built near towns where they do not already exist, and the authorities should have the power of ordering lepers infringing the regulations either to return to their homes or to enter an asylum.

Competent medical authority should always be consulted before action is taken under such by-laws.

In no case would the Commissioners suggest an Imperial Act, especially directed against lepers as such, for these are far less dangerous to a community than insane or syphilitic people.

The effect of such by-laws in large towns would be an emigration into the surrounding country, and a furtherance of the scheme now to be proposed for establishing experimental leper colonies or farms in rural districts.

The experiment of the leper farm in Cyprus[5] has succeeded. Though unwilling to argue from a small island to a large empire, the Commission are of opinion that the success of leper colonies such as that of Sialkot, which is isolated in the centre of a large agricultural district, and where lepers with their wives and families cultivate the soil, leads to the belief that similar farms scattered over the country would be productive of practical good.

Land might be granted, cheap buildings raised, seeds distributed, and work supplied. The produce might partially support the colony, and a small fixed money allowance might be given, or a small bounty be paid on the produce raised by each leper.

Comparatively few children would be born, and these should, if possible, be removed to Orphanages. Only a certain number of the latter would be required, as the inmates might be discharged as soon as they were old enough to support themselves. The advantages of such a method are fully illustrated in the Almora Orphanage.

In conclusion, the Commissioners believe, from the considerations and arguments adduced in the foregoing report, that neither com-

(5) Dr. Heidenstam, C.M.G. Report on Leprosy in Cyprus, 1890, page 13.

pulsory nor voluntary segregation would at present effectually stamp out the disease, or even markedly diminish the leper population, under the existing conditions of life in India. It can only be hoped that by means of improved sanitation and good dietetic conditions a diminution of leprosy will result. The Commission agree with most authorities in believing that the decline of leprosy in Europe has been due principally to improved hygienic habits and surroundings, and increased material prosperity.

English Members.

BEAVEN RAKE.

GEORGE A. BUCKMASTER.

ALFRED A. KANTHACK.

Indian Members.

ARTHUR BARCLAY, *Surgeon-Major, Bengal Medical Service.*[6]

SAMUEL J. THOMSON, *Surgeon-Major, Bengal Medical Service.*

SIMLA,
August 21st, 1891.

(6) The name of the late Surgeon-Major A. Barclay is appended to this report, as the conclusions and suggestions were drawn up previous to his death, and were known to be in accordance with his views.

Appendices I and II are printed separately for the following reasons. In the interests of the investigation, one section of the Commission commenced enquiries at Almora, while the other began work at Simla. Subsequently, when the two sections of the Commission met, it was found that the enquiry had proceeded so far that it would be preferable to continue experiments separately, especially as there was not sufficient room in the laboratory erected by the Government of India at Simla for the entire Commission to work together with facility. The two sections met at intervals, compared their observations, and discussed details of their work. Each Appendix consequently represents independent observations for which each section is separately responsible.

APPENDIX I.

Laboratory Work done in Almora and Simla.

By
DR. BEAVEN RAKE,
DR. GEORGE ALFRED BUCKMASTER,
SURGEON-MAJOR S. J. THOMSON.

Explanation of Plate I.

FIG. 1.—Culture on May 28th from bouillon inoculated on May 14th, with capillary tube containing blister fluid in which were bacilli. In the specimen the bacilli are bright red. Stained with Ziehl's solution for six hours, then 25 per cent. nitric acid and 60 per cent. alcohol. Drawn with Camera Lucida. Winkel Homog. Immersion $\frac{1}{14}$, ocular 4.

FIG. 2.—Culture on Agar on June 10th, 4th generation inoculated on June 8th. Stained and drawn as above. Winkel Homog. Immersion $\frac{1}{14}$, ocular 4.

FIG. 3.—Culture on plate, 2nd generation of June 18th, from plate culture of two days earlier which was inoculated from 4th generation on Agar of June 8th. Stained and drawn as above. Winkel Homog. Immersion $\frac{1}{14}$, ocular 4.

Explanation of Plate II.

FIG. 1.—Surface pellicle on bouillon. This was the original culture from blister fluid in which bacilli had multiplied. The broken capillary tube containing this fluid is seen at the bottom of the culture. A surface pellicle, general slight turbidity, and just a faint bottom deposit are seen. Appearance on twelfth day after introduction of the capillary tube.

FIG. 2.—First generation on gelatine three days after inoculation from culture of Fig. 1. Central pellicle, and commencing fluidity of gelatine. Absence of growth in the needle track to any extent.

FIG. 3.—Appearance of the gelatine culture of Fig. 2 twenty days later, showing a thick tenacious surface scum which is capable of being drawn out into threads, and a clear liquid gelatine with deposits of ropy sticky culture.

FIG. 4.—Culture on Agar. Piece of tubercle from centre of the lobe of the ear. The inoculation was made on May 22nd. Appearance six weeks subsequently. No trace of bacilli of any kind, but a rich growth starting from the tubercle of *staphylococcus albus* and *cereus*.

FIG. 5.—First generation on Agar from original bouillon. Appearance of growth on third day.

FIG. 6.—Same culture as Fig. 5, showing appearance at end of sixth day.

Fig 1.

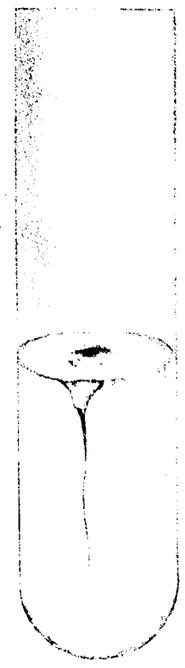
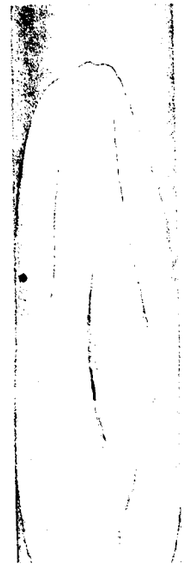


Fig 2.

Fig. 3.



Explanation of Plate III.

FIG. 1.—Culture of bacillus in bouillon on eleventh day (original culture). Carbol-fuchsin; water; 20 per cent. nitric acid. Winkel Homog. Immersion $\frac{1}{14}$, ocular 4.

FIG. 2.—Same culture as Fig. 1.; five days later, showing hull Gram's Method, Winkel Homog. Immersion $\frac{1}{14}$, ocular 4.

FIG. 3.—Second generation on gelatine; three days old. Carbol-fuchsin; water, and subsequent 20 per cent. nitric acid. Winkel Homog. Immersion $\frac{1}{14}$, ocular 4.

FIG. 4.—Second generation on Agar from first generation on Agar; two days old. Carbol-fuchsin; water and subsequent 20 per cent nitric acid. There is a hull round a few bacilli, and being drawn in water, these appear rather large.

FIG. 5.—First generation on Agar from original; three days old, showing the commencing grouping together of the bacilli owing to the development of a mucin (?) hull. Magnification as above.

FIG. 6.—A small colony of first generation on Agar; five days old, showing peculiar grouping of bacilli into heaps very comparable to the grouping in tissues. Methyl Blue. Winkel $\frac{1}{14}$, ocular 3.

FIG. 7.—Culture of bacillus resembling the vacuolated bacilli figured by Neisser. Magnification as above.

FIG. 8.—Appearance exhibited by bacilli described by Neisser as Kugel Bacteria. Magnification as above.

All the figures were drawn with the Camera Lucida.

The following is a record of Laboratory work which was done at Almora and Simla by Drs. Rake, Buckmaster, and Surgeon-Major Thomson.

I.—The Bacillus Lepræ.

The microscopical examination of the pathological neoplasms or tubercles which are characteristic of one form of leprosy was first made by Virchow in 1859.[1] The appearances presented by the nerves in the anæsthetic form are also figured by the same observer. In both these forms of the disease typical nucleated lepra-cells were seen which contained granular matter. Vandyke Carter by his researches, carried on independently in Bombay, constantly found these specific structural elements present in leprosy,[2] and Klebs[3] in 1873 drew attention to the fact "that in freshly extirpated tubercles, groups of bacteria, which in form and arrangement totally differed from those met with in other diseases, could be seen without difficulty." Armauer Hansen in 1868 and 1873 described movable rod-shaped bodies in fresh tubercles, and yellow spherical masses which possibly were zooglœa masses in older ones. In 1880 the Norwegian observer, who during the preceding seven years had shown his preparations to numbers of medical men, published a record of his experiments and observations accompanied with figures, in which the granular masses within the lepra-cells were shown to be heaps of bacilli.[4] At this period the work of Koch[5] and Weigert,[6] which is the foundation of scientific bacteriological method, was all but unknown, and with the exception of the Bacillus Anthracis[7] and some ill-defined bacteria connected with Pyæmia and Septicæmia,[8] scarcely a single definite pathogenic organism had been discovered.[9] Among the great number of observers who have verified the accuracy of Hansen's discovery, the work

(1) Die Krankhaften Geschwülste, Bd. II, p. 494.
(2) On Leprosy and Elephantiasis; Vandyke Carter, 1874, pp. 169,170, Plate XII.
(3) Archiv. f. Experiment. Path. und Pharm., 1873 (quoted from Carter).
(4) Virchow's Archiv. 1880, Bd. 79, p. 31.
(5) Cohn's Berträge zur Biologie der Pflanzen Bd. II, Heft 3; and Wundinfectionskrankheiten, 1873.
(6) Sitzung der Schles. Gesellsch. für vaterl. Cultur, 1875.
(7) Seen by Rayer and his pupil Davaine in 1851.
(8) Burdon Sanderson, Report of the Medical Officer of the Privy Council, Series II, No. 3.
(9) The Bacilli of Septicæmia in mice and rabbits, and also certain pathogenic micrococci were fully described by Koch in 1878.

of Neisser, who by the aid of the Koch-Weigert methods, placed this discovery beyond question, must be mentioned.[10] Sufficient time has now passed for it to be possible to confidently affirm that the presence of the Bacillus Lepræ in the new growths of leprosy is absolutely characteristic of the disease. Indeed, this bacillus has a specific relationship in the causation of leprosy. No leper is free from this organism, and in the bodies of those suffering from other diseases it never occurs. For these reasons the Bacillus Lepræ has been a subject of much study to those who have concerned themselves with the disease, and the most important results which are at present known will be briefly given.

The Bacillus Lepræ is a rod-like parasite, the length of which is one-half to three-quarters the diameter of a human red blood-disc;[11] in breadth about one-fifth of the length. It is straight, or slightly bent, with extremities that are pointed or slightly round. The bacillus possesses a protoplasmic content, in which clear spaces may at times be seen (endogenous spores?), surrounded by a delicate membrane. According to De Bary this membrane is only the most compact internal layer of a hull of material which may swell up, take a gelatinous character, and form an envelope to the bacillus. With certain reagents, such as iodine solution, the coherent rod may present the appearance of a row of minute spheres or cocci (kokkothrix). Whether these, morphologically, are true sphærobacteria or only a granular thread is uncertain. This appearance can also be produced by treatment with strong sulphuric acid, by staining with borax-methyl-blue and subsequent decolorisation in water or alcohol, and further by staining with hæmatoxylin or osmic acid. Neisser[12] holds the view that the Bacillus Lepræ is rod-shaped and possesses a hull, the innermost layer of which retains such a stain as fuchsin better than the outer layers, and this obscures the finer structure of the bacillus. The same observer states that the contents of a bacillus are (1) highly refracting oval spores; (2) ordinary protoplasm which scarcely stains at all, or if it does is easily bleached by acids; (3) granules. These latter become visible under the influence of the reagents mentioned above and give rise to the kokkothrix aspect. The bacillus has been described as both motile and non-motile, possibly this is true for young and old bacilli respectively. The bacillus multiplies by

(10) Breslauer Aerztl. Zeitschr. 1879, Nos. 20, 21, and Virchow's Archiv. 1881, Bd. 84, p. 514.
(11) A human red blood disc is 7·8 micro-millimeters ($\frac{1}{3200}$ mm.).
(12) Neisser, Archiv. f. Dermat. und Syph. 1889. Quoted from Baumgarten's Jahresbericht, 1889.

fission; a bacillus grows to a certain length and then divides into two, and the process is repeated. Isolated spores which have become free have never yet been seen.

In form and size Bacillus Lepræ greatly resembles Bacillus Tuberculosis. A distinction is considered to exist between these in their behaviour to staining reagents, and this rests upon a micro-chemical reaction dependent upon the behaviour of the investing membrane of the bacillus towards acids, alkalies, and aniline dyes.[13] The staining procedure employed as diagnostic of the Bacillus Lepræ is as follows: 1st, treatment of the section of tissue, or film fixed upon a cover-glass, with warm Ziehl's solution[14] for twelve minutes; 2nd, decolorisation of the specimen in 25 per cent. nitric acid; 3rd, washing in 60 per cent. alcohol; 4th, washing in distilled water. Cover-glass specimens are at once examined in water, or after drying, in xylol-balsam. Sections are treated with absolute alcohol and removed to bergamot oil before mounting in balsam. A saturated solution of acetate of potash is the best medium in which to mount specimens, as the colour disappears less rapidly. The Bacillus Tuberculosis is not stained by so short an immersion in Ziehl's solution, and all other bacilli that were stained would have been wholly decolorised by the acid, while the Bacilli Lepræ resist this bleaching[15] and stand out in the field of the microscope as bright red rods (Baumgarten). The following also stain well by the above method and exhibit a resistance to strong mineral acids which is almost as great as that shown by Tubercle and Leprosy Bacilli; (1) keratin tissue; (2) the spores of a large kind of bacillus found in the intestinal canal and described by Koch; (3) the spores of mucor; (4) certain pseudo-bacilli figured by Celli and Guarnieri which are probably crystals of fatty acid; (5) the Syphilis, or Smegma Bacillus, which to some extent behaves similarly, due

(13) Ehrlich's early papers are in Zeitschrift f. klin. Med. Bd. I, 1880; Ibidem Bd. II, 1881; Deutsch. Med. Wochen. 1882, No. 19; Charité Annalen 1886. The views of Ehrlich with reference to the behaviour of Bacillus Lepræ and Bacillus Tuberculosis towards aniline dyes and also to acids, are, that, firstly, the membrane of these bacilli is only slightly permeable to acids; and, secondly, that the acid chemically changes this membrane, so that the colour held by the protoplasm cannot diffuse outwards. The value of the addition of alkalies, aniline or phenol to the dye depends upon the facts that the membrane becomes more permeable, and a true combination of these reagents with the dye ensures a more brilliant staining effect.
(14) Water 100 cub. cent.; Crystallized Carbolic Acid 5 grammes; Alcohol 10 cub. cent.; Fuchsin 1 gramme (Ziehl-Neelsen).
(15) As a fact, the bacilli do lose their colour wholly or in part in the acid, but regain it in the treatment with alcohol and water. A piece of silk treated with aqeous methyl-violet similarly reacts by losing colour in acid and recovering this in water.

to the presence of a hull of fatty material. Leprosy Bacilli may also be differentiated from those of Tubercle by treatment with potash solution 1 : 12. The bacilli appear as clear rather thick rods. If a drop of watery methyl-violet be now added to the specimen, only the Bacillus Lepræ stains, while the other remains colourless (Baumgarten). However, the Bacillus Tuberculosis can also be stained with watery aniline dyes[16] and the Bacillus Lepræ with alkaline methyl-blue.[17] The method with Bor-fuchsin[18] has not succeeded in our hands; the Leprosy Bacilli remained stained. The staining behaviour of Bacillus Lepræ, according to Neisser,[19] may be summarised briefly as follows: fuchsin, gentian and methyl-violet, dahlia, especially in weak acid solution, stain the bacillus well; methylene-blue, nigrosin, aurantia, methyl-green and eosin have no staining effect; Bismarck brown does not stain whether in acid or alkaline solution, though Koch has obtained a slight coloration with vesuvin. An eosin-alum-hæmatoxylin solution (Ehrlich) stains the nuclei of tissues blue, the cell protoplasm rose, and the bacilli orange. The coloration of the bacilli is always less than that shown by micrococci, and at times the colour disappears in a day or so. The methods of Kühne and Unna having reference chiefly to the manner of disposition of the bacilli within the tissues and organs of the body, have not been employed by us. Gram's method stains the bacilli well, and the kokkothrix appearance is admirably seen.

Though the work of Weigert, Ehrlich, Kühne, Unna and others has to some extent changed the technology of staining from an empirical to a scientific basis, much is still required before a definite staining procedure can be considered as a definite micro-chemical reaction. At present the differential diagnostic method for the Bacillus Lepræ and Bacillus Tuberculosis given above is generally recognized as the best means for detection of the Leprosy Bacillus; but it may be considered an open question whether this is absolute. The grouping of the bacilli in clumps in cells of

(16) Baumgarten, Path. Mykologie, Vol. II, page 645. Wesener, Centralbl. f. Bac. u. Parasit, Vol. II, 1887.

(17) Koch, Berlin, klin. Woch. 1882. According to Bordoni-Uffreduzzi a distinction exists between Bacillus Lepræ and Bacillus Tuberculosis in that only the latter stains with methyl-blue.

(18) Lübimoff. Abstract of paper in Baumgarten's Jahresbericht, Vol. I, 1888. Stain in the following solution: Fuchsin 0·5 gramme; Boracic Acid 0·5 gramme; Absolute Alcohol 15·0 cub. cent., water 20 cub. cent.; decolorise in 1 : 5 Sulphuric Acid. The Bacillus Lepræ decolorizes, but not the Bacillus Tuberculosis.

(19) Virch. Archiv. 1881.

specific shape and structure[20] is, however, characteristic of the Bacillus Lepræ within the tissues of the body. This aggregation may be due to the nature of the hull of the bacillus, for though, according to Lutz and Unna, the Bacillus Tuberculosis possesses a similar hull, this is rarely grouped into heaps, and even the arrangement within giant cells in no way resembles the grouping that is presented by the Bacillus Lepræ

Bacilli which have been in alcohol stain better than when seen in fluid just expressed from a tubercle. A very prolonged treatment with warm Ziehl's solution will also enable bacilli, other than Bacillus Lepræ or Bacillus Tuberculosis, to resist the effect of acids. Masses of micrococci also retain the stain. Spina's statement[21] that putrefactive bacteria treated with 2 per cent. tannin stain like the above bacilli we did not succeed in confirming.

II.—DISTRIBUTION OF THE LEPROSY BACILLUS WITHIN THE BODY.

In the absence of autopsies this subject could not be dealt with as fully as might have been wished. From the records of various observers, it is however by this time well known that the bacillus of leprosy has been found in most of the tissues and viscera of the body, though it occurs in some, such as the liver and spleen, far more frequently than in others, such as the kidney and brain.

Observations on the following fluids, secretions, and excretions of the body were made as opportunity offered at Almora, Simla, and Hyderabad.

Fluids.

Blood.	Juice from leprous tubercle.
Blister fluid.	Discharge from leprous ulcer.
Material from thickened nerve.	

Secretions.

Saliva.	Vaginal mucus.

Excretions.

Urine.	Sputum.
Fæces.	Menstrual discharge.

(20) With the question whether the bacilli are within cells, or collected into groups lying exterior to these, we have not concerned ourselves. The latter view which is held by Unna and exhibited by his "Trocken Methode" is also that of Kühne. The iodine-pararosaniline-method also shows the rod-like bacilli to be a coccus series, similar to that figured by Zopf for Bacillus Tuberculosis (*vide* Die Spaltpilze).

(21) Quoted by Stricker in Infectionskrankheiten.

FLUIDS.

Blood.

Köbner is the only pathologist who claims to have found Leprosy Bacilli in the blood.

All other observers have failed, even during acute outbreaks of the disease, and it may fairly be doubted if the bacillus can live in the blood.

Twenty-three cover-glasses prepared from blood obtained from six lepers failed in any case to show Leprosy Bacilli. It is worthy of note that even where the blood was obtained by section of a tubercle, the results were negative. The following table shows the results of examination of blood:—

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Source of Blood.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Amarua . .	25	...	M	13	Tuberculated hand .	25-3-91	4	1, 2, 3, 4. Small rods stained blue. No Leprosy Bacilli.
2	„ . .	25	...	„	13	Anæsthetic skin of arm.	25-3-91	2	1. Rods of various lengths, not taking fuchsin 2. Rods stained blue.
3	Phaganiya .	32	...	A	12	Vein of anæsthetic hand.	25-3-91	4	1, 2, 3, 4. Debris only.
4	Jaikishen . .	35	...	M	29	Normal skin of fore-arm.	30-3-91	3	1, 2. Cocci. 3. Cocci and short rods. No Leprosy Bacilli.
5	Fakira . .	30	...	M	10	Normal skin of fore-arm.	30-3-91	2	1, 2. Blood disc. No bacilli.
6	Puniya . .	30	...	M	22	Normal skin of fore-arm.	30-3-91	2	1. A few red cocci. 2. Negative.
7	Bishan Singh .	30	...	T	10	Back of finger between two tubercles.	7-4-91	2	1, 2. Debris. No bacilli.
8	„ .	30	...	„	10	Incised tubercle of hand.	7-4-91	2	1, 2. Debris. No bacilli.
9	„ .	30	...	„	10	Incised tubercle of wrist.	13-4-91	2	1, 2. No Leprosy Bacilli.

Blister Fluid.

The observations and experiments made on blister fluid are fully explained in another part of the report, for it was by the application of blisters over tubercles that it appeared possible to separate living Leprosy Bacilli from the tissues, and subsequently to grow them in blister serum and other media. Little more than an enumeration of the observations need, therefore, be here given. The subjoined table shows that blisters on various parts of the body were raised on five different patients. Fifty-nine cover-glasses were prepared, and of these, nine specimens from three different patients showed Leprosy Bacilli in greater or less quantities. It must be noted that all these positive cases were in fluid obtained from blisters over actual tubercles. In blisters raised over anæsthetic patches or normal skin no bacilli were in any case found.

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Material examined.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Puniya . .	30	...	M	22	Fluid from blister over tubercle of cheek.	27-3-91	4	1. Swarms of bacilli of various lengths; some very long; some in nests; deeply stained with fuchsin. 2, 3, 4. A few red bacilli.
2	„ . .	30	...	„	22	Fluid from blister over right superciliary tubercle.	27-3-91	3	1. Weigert's threads stained red. No bacilli seen. 2, 3. A few red bacilli.
3	Phaganiya .	32	...	A	12	Fluid from blister over anæsthetic patch of back.	28-3-91	2	1. Nothing beyond a few pieces of shreddy material. 2. A few red bacilli.
4	„ .	32	...	„	12	Fluid from blister over normal skin.	22-3-91	2	1. No bacilli. 2. A few red bacilli.
5	Fakira .	30	...	M	10	Fluid from blister over tubercle of nose.	31-3-91	2	1. Quantities of Leprosy Bacilli. 2. A few red bacilli.
6	„ . .	30	...	„	10	Fluid from blister over non-tuberculated back.	31-3-91	2	1. Numerous leucocytes. No bacilli. 2. A few red bacilli.
	Jaikishen .	35	...	M	29	Fluid from blister over non-tuberculated back.	31-3-91	2	1. Debris. No bacilli. 2. A few red bacilli
8	„ . .	35	...	„	29	Fluid from blister of back.	1-4-91	3	1. Large bacteria. No bacilli. 2. Debris. No bacilli. 3. A few red bacilli.

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Material examined.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
9	Jaikishen . .	35	...	M	29	Fluid from blister of back.	1-4-91	2	1. Chain of short thick bacilli. No Leprosy Bacilli. 2. Bacilli and cocci. No Leprosy Bacilli.
10	Puniya . .	30	...	,,	22	Fluid from blister on tuberculated ear.	1-4-91	2	1. Debris. No bacilli. 2. Bacilli and cocci. No Leprosy Bacilli.
11	,, . .	30	...	,,	22	Fluid from blister on tuberculated ear.	1-4-91	2	1. Few other bacilli. No Leprosy Bacilli. 2. Bacilli and cocci. No Leprosy Bacilli.
12	Jaikishen . .	35	...	M	29	Fluid from blister of back.	1-4-21	2	1. Few bacilli stained blue. No Leprosy Bacilli. 2. Chain of bacilli stained blue. No Leprosy Bacilli.
13	,, . .	35	...	,,	29	Fluid from blister over tuberculated ear.	3-4-91	2	1. Few suspicious bacilli taking pink stain. 2. Leucocytes. No bacilli.
14	Fakira . .	30	...	M	10	Fluid from blister over tuberculated nose.	4-4-91	2	1. Few Leprosy Bacilli, also a number of unstained bacilli. 2. Debris. No bacilli.
15	Bhaguli . .	...	...	...	...	Fluid from blister over anæsthetic patches on back.	4-4-91	2	1. Numerous leucocytes. No bacilli. 2. Debris. No bacilli.
16	Fakira . .	30	...	M	10	Fluid from blister over tubercle of face.	6-4-91	1	1. Numerous unstained bacilli. No Leprosy Bacilli.
17	Bishan Singh .	30	...	T	10	Fluid from blister over tubercle of nose.	7-4-91	2	1. Few Leprosy Bacilli. 2. Debris. No bacilli.
18	Bishan Singh .	30	...	,,	10	Fluid from blister over tubercle of wrist.	8-4-91	4	1. Few micrococci and bacilli stained blue. 2, 3, 4. Debris. No bacilli.
19	,, .	30	...	,,	10	Fluid from blister over tubercle of cheek.	8-4-91	2	1. Few micrococci and bacilli stained blue. 2. Debris. No bacilli.
20	,, .	30	...	,,	10	Fluid from blister over tubercle of forehead.	11-4-91	2	1. Leucocytes. No bacilli. 2. Debris. No bacilli.
21	,, .	30	...	,,	10	Fluid from blister over tubercle of cheek.	11-4-91	2	1. Leucocytes. No bacilli. 2. Debris. No bacilli.
22	Puniya . .	30	...	M	22	Fluid from blister over tubercle of forehead.	11-4-91	2	1. Leprosy Bacilli and leucocytes. 2. No Leprosy Bacilli.
23	,, . .	30	...	,,	22	Fluid from blister on non-tuberculated back.	11-4-91	2	1. Quantities of leucocytes. No bacilli. 2. No Leprosy Bacilli.
24	Fakira . .	30	...	,,	10	Fluid from blister over tubercle of lip.	14-4-91	2	1. Pus cells and bacteria. 2. Debris. No bacilli.

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Source of Saliva.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
25	Fakira . .	30	...	M	10	Fluid from blister over healthy back.	14-4-91	1	1. No bacilli.
26	Puniya . .	30	...	,,	22	Fluid from blister over tubercle of forehead.	14-4-91	2	1. No Leprosy Bacilli. 2. Debris. No bacilli.
27	,, . .	30	...	,,	22	Fluid from blister over healthy back.	14-4-91	1	1. Leucocytes. No bacilli.
28	Bishan Singh .	30	...	T	10	Fluid from blister over tubercle of face.	14-4-91	1	1. Debris. No bacilli.
29	,, .	30	...	,,	10	Fluid from blister over healthy back.	14-4-91	1	1. Leucocytes. No Leprosy Bacilli.

Juice from leprous tubercle.

It may safely be affirmed that Leprosy Bacilli are always found in cutaneous and other tubercles at some period or other of their existence. The failure to find them is due either to error of preparation or observation, to the fact that no bacilli have happened to be expressed on to the cover-glass, or to the fact that the tubercle is old and degenerating, and the bacilli are consequently dead. That it is not always easy to obtain bacilli from leprous tubercles is shown by the observations recorded above that no bacilli were found in blood from tuberculated tissue. Evidently more than simple section of the tubercle is usually required to extract the bacilli. Compression must also be employed as recommended by Manson, or the blister method detailed above. So well known is the association of bacilli with leprous tubercles, that it was hardly considered necessary to make observations on this point. Such as were made were for the purpose of controlling other work, such as blister or inoculation experiments. The results are given in the next table, and it will be seen that only three observations absolutely failed to show them, while in another they were doubtful.

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Source of Juice.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Puniya . .	30	...	M	22	Tubercle of ear .	25-3-91	4	1, 2, 3. Quantities of Leprosy Bacilli free and in cells. 4. Negative.

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Source of Juice.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
2	Puniya . .	30	...	M	22	Tubercle of ear .	31-3-91	2	1, 2. Quantities of Leprosy Bacilli.
3	,, . .	30	...	,,	22	,, ,, .	14-4-91	1	1. Clusters of Leprosy Bacilli.
4	Fakira . .	30	...	,,	10	,, ,, .	14-4-91	1	1. Doubtful bacilli taking fuchsin.
5	Bishan Singh .	30	...	T	10	,, of wrist .	14-4-91	1	1. Debris. No bacilli.
6	,, ,, .	30	...	...	10	,, of finger .	15-4-91	1	1. Swarms of Leprosy Bacilli.
7	,, ,, .	30	...	...	10	,, of wrist .	15-4-91	1	1. Cocci stained blue. No bacilli.
8	,, ,, .	35	...	T	...	,, of face .	29-6-91	1	1. Few Leprosy Bacilli.
9	Rahim Khan .	35	...	,,	2½	,, of ear .	12-7-91	2	1. Clusters of Leprosy Bacilli.

Discharge from leprous ulcers.

In the discharge from tuberculated ulcers Leprosy Bacilli, both free and in cells, are more beautifully brought out by staining than perhaps in any other fluid or tissue. Whether it is that the bacilli in this discharge have been so macerated as to be divested of their mucin-like hull is a question, but the fact remains. In the discharge from anæsthetic ulcers, on the other hand, they could not be found.

At Almora an experiment was made in which a leper washed his tuberculated and ulcerated feet in a basin of water. Examination of cover-glass preparations from this water showed Leprosy Bacilli in every specimen. The experiment was repeated at Simla with like results, but plate cultivations from this water failed to produce any colonies of Leprosy Bacilli. It is possible to argue from these experiments that though the bacilli are thrown off in great numbers from tuberculated ulcers, they are not living. It must, however, be remembered that discharges from open ulcers are so full of microbes of every description, that the Leprosy Bacillus, which does not grow rapidly or very vigorously, might easily be overpowered in the struggle for existence with other organisms. An account of the different discharges examined is given in the next table.

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Source of Discharge.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Phaganiya .	32	...	A	12	Ulcer on anæsthetic hand.	25-3-91	2	1. Doubtful bacilli, probably not pathogenic. 2. Debris only.
2	Fakira . .	30	...	M	10	Tuberculated ulcer on dorsum of foot.	3-4-91	2	1, 2. Quantities of Leprosy Bacilli singly and in groups.
3	,, .	30	...	,,	10	Tuberculated ulcer on side of sole.	3-4-91	2	1, 2. Quantities of Leprosy Bacilli, but not so numerous as in last specimen.
4	Bishan Singh .	30	...	T	10	Water in which tuberculated ulcers of feet had been washed.	13-4-91	4	1, 2, 3, 4. Epithelial squamous nucleated cells, dirt, and Leprosy Bacilli. [In many cases the bacilli were in distinct clumps, *i. e.*, the cells were loaded with them.]
5	Fakira . .	30	...	M	10	Water in which tuberculated ulcers of feet had been washed.	13-4-91	4	1, 2, 3, 4. Leprosy Bacilli as in last four specimens.
6	Rahim Khan .	35	...	T	2½	Water in which tuberculated ulcers of feet had been washed.	10-6-91	4	1, 2, 3. Leprosy Bacilli. 4. Negative.

Material from thickened nerve.

Four cover-glass preparations from a leprous ulnar nerve, operated on by Surgeon-Major Lawrie at Hyderabad, were examined, but no bacilli were found. Comparatively little value can, however, be attached to this observation, for bacilli in leprous nerves often escape notice unless sections are made. This, of course, was impracticable in the above case.

SECRETIONS.

Saliva.

In many of the cases in which bacilli were found in the saliva, the tongue was tuberculated and in some cases ulcerated. It is, therefore, not a matter of surprise that bacilli should be present in this secretion. Indeed, so frequently and rapidly do leprous tubercles of the tongue, throat, and

larynx ulcerate, that the wonder is that bacilli are not more frequently found in saliva.

The following table shows the number of specimens of saliva examined, and it will be seen that in only eight out of twenty-two observations were characteristic bacilli seen :—

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Source of Saliva.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Puniya . .	30	...	M	22	Over tuberculated tongue.	27-3-91	2	1, 2. Epithelioid cells in which lie spherical masses filled with Leprosy Bacilli in stages of apparent disintegration.
2	„ . .	30	...	„	22	Over tuberculated tongue.	28-3-91	2	1, 2. Debris. No bacilli.
3	Jaikishen . .	35	...	„	29	Apparently normal mouth.	28-3-91	2	1, 2. Small blue rods and cocci.
4	„ . .	35	...	„	29	Apparently normal tongue.	30-3-91	2	1. Squamous epithelium and leucocytes. 2. Bacteria and leucocytes.
5	Puniya . .	30	...	„	22	Over tuberculated tongue.	30-3-91	2	1, 2. Debris, Squamous epithelium, leptothrix and bacteria.
6	Fakira . .	30	...	„	10	Apparently normal mouth.	30-3-91	2	1, 2. Quantities of small blue rods and cocci.
7	Puniya . .	30	...	„	22	Over tuberculated tongue.	3-4-91	2	1, 2. No Leprosy Bacilli.
8	Bishan Singh .	30	...	T	10	Over tuberculated ulcer of tongue.	10-4-91	2	1, 2. Several Leprosy Bacilli lying over blue masses of epithelium.
9	Bishan Singh .	30	...	T	10	Over tuberculated ulcer of tongue.	11-4-91	2	1, 2. Some Leprosy Bacilli, also leptothrix and salivary cells. 2. No bacilli.
10	Rahim Khan .	35	...	„	24	Over tuberculated ulcer of tongue.	22-5-91	4	1, 2, 3. Showed clumps of Leprosy Bacilli. 4. Negative.

Vaginal Mucus.

Surgeon-Major H. D. Cook kindly sent specimens from two cases in the Madras Asylum.

The first specimen was taken on 24th June, 1891, from a patient, aged 16, suffering from mixed leprosy. Ten cover-glass preparations were examined on 17th July, 1891, and showed an immense number of decolorised bacilli

with a few faintly stained ones. There were also staphylococci and beaded rods, sometimes stained pink. In one specimen were a few spore-bearing bacilli, the spores being a deep red. There were no undoubted Leprosy Bacilli.

The second specimen was taken on 24th June, 1891, from a tuberculated leper, aged 18. Ten cover-glasses were examined on 17th July, 1891, and showed numerous staphylococci, isolated bacteria, and micrococci. In one specimen was a mass of six or seven highly stained short rods which were possibly Leprosy Bacilli.

Leprosy Bacilli have been described in vaginal secretion by one or two observers (Kalindero and Babés). The bearing of these observations on the question of inoculability of leprosy is obvious.

EXCRETIONS.

Urine.

The urine of six lepers was examined, two of them being mixed cases, three anæsthetic, and one tuberculated. Four cover-glasses from each case were taken, making twenty-four observations in all. In no case were Leprosy Bacilli found. This is not surprising, for very rarely are Leprosy Bacilli found in kidneys examined after death.

The results obtained from urine are given below:—

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Material examined.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Puniya . .	30	...	M	22	Urine . . .	10-4-91	4	1, 2, 3, 4. Epithelium. No Leprosy Bacilli.
2	Fakira . .	30	...	„	10	„ . . .	10-4-91	4	Ditto ditto.
3	Bishan Singh .	30	...	T	10	„ . . .	10-4-91	4	Ditto ditto.
4	Ratanah . .	18	...	A	13	„ . . .	10-4-91	4	Ditto ditto.
5	Gumaniya .	35	...	„	19	„ . . .	10-4-91	4	Ditto ditto.
6	Phagania. .	32	...	„	12	„ . . .	10-4-91	4	Ditto ditto.

Fæces.

This excretion was examined in five lepers, two anæsthetic, two mixed, and one tuberculated. Sixteen cover-glass preparations were made, and in one from a mixed case Leprosy Bacilli were found. In this patient, however, bacilli had previously been found in the saliva, so that it was quite possible to expect to find them in the fæces.

The observations were as follows: —

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Material examined.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Puniya	30	...	M	22	Fæces	27-3-91	4	1, 2, 3, 4. Quantities of bacteria. No Leprosy Bacilli.
2	Jaikishen	35	...	,,	29	,,	27-3-91	4	1, 2. Quantities of bacteria. No Leprosy Bacilli. 3, 4. Numerous decolorised rods and cocci.
3	Phaganiya	32	...	A	12	,,	11-4-91	2	1, 2. Swarms of bacilli and cocci, also bright oval fuchsin-spores (described by Koch).[22]
4	Gumaniya	35	...	,,	19	,,	11-4-91	2	1, 2. Swarms of bacteria stained blue and brightly stained oval fuchsin-spores.
5	Bishan Singh	30	...	T	10	,,	13-4-91	4	1. A few Leprosy Bacilli. 2. Swarms of cocci and large bacteria stained blue, also oval fuchsin-spores. 3, 4. No Leprosy Bacilli.

Sputum.

Sputum from six lepers was examined, four cover-glasses being taken from each case. Five of the cases were mixed, and one tuberculated. All the patients had raucous voices, indicating leprous infiltration of the larynx. It was therefore to be expected that bacilli would be found in the sputum. It appears to us that it is absolutely impossible to confuse Leprous and Tubercular sputum. In the former case isolated bacilli are very rarely seen, but large typical cells filled with bacilli to such an extent that it is difficult to recognize them individually, abound in advanced cases. In the absence of autopsies during the laboratory work, we are unable to speak

(22) Koch, Mit. a. d. Kaiser. Gesundh. Bd. II, 1884, page 34.

definitely as to the existence of leprous phthisis. Leprosy Bacilli were present in four out of the six patients, or in seventeen out of the twenty-eight specimens, as shown below:—

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	Material examined.	Date.	No. of cover-glasses.	Result of Microscopic Examination.
1	Harkua	40	...	M	31	Sputum	15-4-91	4	1, 2, 3, 4. No bacilli.
2	Saduli	...	38	,,	10	Sputum mixed with blood.	15-4-91	4	1, 2, 3, 4. No bacilli.
3	Kunka	...	28	,,	10	Sputum	15-4-91	4	1, 2, 3. Clumps of Leprosy Bacilli in cells. 4. Negative.
4	Puniya	30	...	,,	22	,,	15-4-91	4	1, 2. Clumps of Leprosy Bacilli in cells. 3, 4. Negative.
5	Fakira	30	...	,,	10	,,	15-4-91	4	1, 2, 3, 4. Quantities of Leprosy Bacilli aggregated in cells.
6	Rahim Khan	35	...	T	2½	,,	22-5-91	8	1, 2, 3, 4, 5, 6, 7, 8. Clusters of Leprosy Bacilli in cells.

Menstrual Discharge.

This was sent by Dr. Cook from Madras. The first specimen was from an anæsthetic leper, aged 40. It was taken on June 28, and examined on July 17, 1891. Ten cover-glasses were prepared, and all gave negative results.

The second specimen was from a patient, aged 19, suffering from mixed leprosy. It was taken on June 27, and examined on July 17, 1891. Ten cover-glasses showed shreds of epithelium, bacteria, micrococci, but no Leprosy Bacilli.

These results are similar to those obtained from vaginal mucus.

III.—DISTRIBUTION OF THE LEPROSY BACILLUS OUTSIDE THE BODY.

Little is yet known of the natural history of the Leprosy Bacillus outside the human body. Arning[23] claims to have found it in the earth of a leper's grave in Hawaii, but failed to find it in mosquitoes which had sucked the blood of lepers. Kaurin[24] failed to find it in earth and in the dust

(23) Report on Leprosy in Hawaii, 1886.
(24) Journal of the Leprosy Investigation Committee, No. 2, p. 68.

and air of rooms inhabited by lepers. Observations in Trinidad failed to show the bacillus in the earth of the asylum cemetery, or in salt fish, salt pork, or urhur dal (*Cytisus cajan*).[25] The attention of the Commission in India was chiefly directed to the following possible habitats of the bacillus outside the human body :—

Earth.	Fish and crustacea.
Water.	Flies.

Earth.

The occurrence of Leprosy Bacilli in earth has already been referred to in the paper on contagion. It will be sufficient to repeat here that a hundred cover-glass preparations were made from earth taken from four of the most likely sites for Leprosy Bacilli within the Almora Asylum compound. Only ten bacilli were found in all these observations, and they occurred on seven cover-glasses. Gelatine plates inoculated from this earth failed to show any colonies of Leprosy Bacilli. It will be seen from the table given below that the number of bacilli to be found in earth habitually trodden by lepers is extremely small, and it is open to doubt whether the few bacilli found were alive.

Observations on Earth at Almora Leper Asylum.

Source of Earth.	No. of cover-glasses examined.	No. of Leprosy Bacilli found.
1. Path in front of office . .	25	3 on 1 cover-glass. 2 „ 1 „ 1 „ 1 „ 1 „ 1 „
2. Path down hill to leper quarters . .	25	1 on 1 cover-glass. 1 „ 1 „
3. Path under bank where lepers sit .	25	1 „ 1 „
4. Bank where lepers sit . . .	25	No bacilli. (One suspicious.)
TOTAL .		10 bacilli in 100 observations.

(25) Report on Trinidad Leper Asylum, 1889, pp. 9 and 10.

Water.

The readiness with which Leprosy Bacilli can be washed out of tuberculated ulcers and detected in basins or other vessels of water has been shown in the preceding section. The object of the present inquiry is to ascertain whether these bacilli can be found distributed in larger collections of water.

For this purpose water was examined from the Almora Leper Asylum and also from the holy tank at Tarn Taran Leper Asylum. This tank is stagnant and overgrown with algæ, and if Leprosy Bacilli are found anywhere in water, they ought to be present here, for this tank is supposed to have remarkable healing properties, and lepers are constantly in the habit of bathing in the water, and swimming through it in the hope of getting rid of their disease.

An examination of fifty cover-glasses prepared from this water failed, however, to show any unequivocal Leprosy Bacilli. Four of the specimens gave several short pink rods closely resembling the bacilli sought for, but after careful and repeated examination by different members of the Commission, it was finally decided that they could not be regarded as Leprosy Bacilli.

The following table shows the observations made on water:—

Source of Water.	No. of cover-glasses.	Result of Microscopic Examination.
1. Water from tank at Almora Asylum, used for supplying bath-room.	2	1, 2. No Leprosy Bacilli.
2. Water from puddle outside spring of drinking water often visited by lepers.	2	1, 2. Debris. No Leprosy Bacilli.
3. Water from tank in which lepers bathe at Tarn Taran Asylum.	50	1 to 50. No positive Leprosy Bacilli. Four specimens showed very suspicious rods taking pink stain.

Fish and Crustacea.

A full discussion of the fish theory is given in the chapter on food in relation to leprosy. Though, as will be seen in that part of the report, abundant evidence unfavourable to this theory was collected in the Punjab

and the Asylums of Almora and Dehra Dun, still it was thought that the laboratory work ought to include the examination of as many varieties of fish and crustacea as possible. For this purpose five different varieties of nga-pi were collected while part of the Commission was travelling in Burma, and specimens of dried fish and shrimps were kindly sent from Darjeeling by Surgeon-Major O'Brien. Numerous species of dried and fresh fish, with some shrimps and crabs, were also obtained from Bombay through the kindness of Dr. J. H. Choksy, and these were supplemented with a few recent specimens of fresh-water fish from the neighbourhood of Simla.

It was considered advisable to examine fish from Darjeeling, for some members of the Commission when making enquiries in this station found to their surprise that dried fish was consumed by almost every native inhabitant. The expenditure of a trifling sum was sufficient to procure enough dried fish for a month, and this was eaten with each meal more as a condiment, like the nga-pi in Burma, than as a food.

Though leprosy is not unduly prevalent in Darjeeling, still it was thought that an examination of the article of diet in question derived from a locality presenting the unusual feature of a hill district where fish is almost universally consumed, would not be without interest. Two hundred cover-glass preparations were made from five varieties of nga-pi, and nineteen species of fish and crustacea, and the material was most carefully stained and examined, but in no case could Leprosy Bacilli be found. As might have been expected, numerous bacteria and other micro-organisms were present, and it is noteworthy that these in most instances retained the pink stain. This only serves to emphasise the fact which has been remarked elsewhere in this report that the staining reaction is after all only of partial importance in diagnosing different bacilli.

The results are given in the appended short table. Unfortunately the species of the various fish and crustacea could not be determined, but this is of less importance since the results were negative.

No.	Description.	Locality.	No. of cover-glasses.	Result of Microscopic Examination.
1	Raw-eaten nga-pi .	Burma .	20	Negative.
2	Zayah nga-pi . .	„ .	20	„ One curved stained bacillus, but not Bac. Lep.

No.	Description.	Locality.	No. of cover-glasses.	Result of Microscopic Examination.
3	Gnagyee nga-pi (putrid, infested with maggots).	Bombay .	20	Pink bacilli [26] in four specimens, one in each. Numerous micrococci. No Leprosy Bacilli.
4	Gnaphayoung nga-pi	„ .	20	Several bacilli, well and badly stained; some curved. No Leprosy Bacilli.
5	Gnakhoo nga-pi .	„ .	20	A few pink bacilli, straight and curved. No Leprosy Bacilli.
6	Dried Bombay duck .	Darjeeling	5	One slightly curved pink bacillus. No Leprosy Bacilli.
7	„ fish . .	„ .	5	One or two pink bacilli.
8	„ „ . .	„ .	5	Large bacilli taking pink stain.
9	„ „ . .	„ .	5	A few pink bacilli.
10	„ shrimps . .	„ .	5	About a dozen large bacilli taking pink stain.
11	„ Bombay duck .	Bombay .	5	Micrococci and one large curved bacillus staining pink.
12	„ pomfret . .	„ .	5	Considerable numbers of large bacilli taking pink stain.
13	„ fish . .	„ .	5	Large thick stained bacilli, not Bac. Lep.
14	„ „ . .	„ .	5	Ditto ditto.
15	„ „ . .	„ .	5	Plenty of large thick bacilli, long bacilli, and micrococci, all taking pink stain.
16	„ „ . .	„ .	5	Ditto ditto.
17	„ „ . .	„ .	5	Ditto ditto.
18	„ „ . .	„ .	5	Ditto ditto.
19	„ „ . .	„ .	5	Ditto ditto.
20	Dried fish . .	Bombay .	5	Rods of various sizes taking pink stain. No Leprosy Bacilli.
21	„ „ . .	„ .	5	Ditto ditto.
22	„ „ . .	„ .	5	Ditto ditto.
23	„ „ . .	„ .	5	Ditto ditto.
24	Fresh fish . .	Simla .	5	Large bacilli.
25	„ crabs . .	Bombay .	5	Negative.

([26]) In many of the samples of fish and nga-pi, stained bodies, which had a crystalline appearance were seen. Possibly these are crystals of fatty acid and similar to the pseudo-bacilli described by Celli and Guarnieri. The paper of these authors is in Accadem. dei Lincei, June 1883.

Flies.

The part played by flies in the spread of certain diseases has been recognised for some time, and it seems reasonable to suppose that they may be capable of conveying the Leprosy Bacillus. An opportunity of putting this to the test occurred at the Almora Asylum. Three lepers were caused to sit on the ground and expose the tuberculated ulcers of their feet. These were soon visited in large number by common house flies, which settled on the ulcers, and fed on the discharges. Several of these flies were caught on the ulcers, their abdomens were opened and cover-glass preparations were made of the intestine and contents. No Leprosy Bacilli could be detected in the flies, though the discharge on which they were feeding was shown in two of the lepers to be rich in Leprosy Bacilli. Indeed, the third leper had been proved to have numerous bacilli in other parts of his body, though by an oversight these particular ulcers were not examined.

Only ten cover-glasses were prepared from ten flies, and it is quite possible that a more extended series of observations might show Leprosy Bacilli. Still considering the immense quantities of Leprosy Bacilli found in the discharge from these tuberculated ulcers, it is strange that they are not more readily discoverable in flies which have fed on these discharges.

A short table follows:—

No.	Name.	Age. M.	Age. F.	Form.	Years afflicted.	On what flies had fed.	No. of flies.	No. of cover-glasses.	Result of Microscopic Examination.
1	Puniya . .	30	...	M	22	Discharge from tuberculated ulcer of foot	7	7	1—7. No Leprosy Bacilli.
2	Fakira . .	30	...	,,	10	Ditto ditto	2	2	1, 2. Ditto.
3	Bishan Singh .	30	...	F	10	Ditto ditto	1	1	1. Ditto.

IV.—VACCINATION.

Of late years much attention has been directed to the possibility of infecting persons with leprosy through vaccination. Gairdner in 1887 published a case, in which a doctor in a tropical island vaccinated his own child from a native child who afterwards became leprous. Another white child

was vaccinated from the doctor's child, and both these children also developed leprosy. The case excited much interest, and many people assumed that it proved the possible transmission of leprosy by vaccination. A perusal of the case, however, shows that the details are very few, and insufficient to establish any conclusion. Moreover, leprosy was endemic in the island, so that the possibility of infection from outside sources was always present.

Direct experiments bearing on the subject have not been wanting. Arning in Hawaii vaccinated lepers and examined the lymph. He found bacilli in the vesicles raised in cases of extensive tuberculated leprosy in which the skin contained bacilli. In cases of pure anæsthetic leprosy he found no bacilli.

A series of observations made in the Trinidad Asylum on vesicles raised on tuberculated, anæsthetic, and mixed lepers failed to show any Leprosy Bacilli, and animals vaccinated with lymph from these vesicles did not develop leprosy.

Daubler's observations[27] refer to two women on Robben Island,[28] aged 15 and 35 years, respectively, both of whom he believed contracted leprosy through vaccination. Both individuals were vaccinated with the lymph obtained from a patient who subsequently died of tubercular leprosy. In the first patient the site of inoculation swelled a few days after the puncture and had a brown hue, but no true vaccine vesicle formed. Subsequently after the swelling had subsided, fever and rigors occurred, and by the eighteenth week swelling and discoloring of the right frontal eminence was seen. The second patient, two months after the vaccination, showed the typical leonine aspect of a tuberculated leper. These cases are frequently quoted, but Arning's experiments, which are in complete accordance with the clinical experience of all observers, do not permit of the belief that such an essentially chronic disease as leprosy is produced so soon after inoculation.

Quite recently Dr. Simpson has vaccinated lepers in Calcutta, and claims to have found Leprosy Bacilli in the vaccine lymph.

(27) Daubler, Ueber Lepra und deren Contagiosität., Monat für prakt. Dermat. Bd. VIII, 1889, p. 123. Abstract in Baumgarten's Jahresbericht, 1889, p. 243.

(28) An island in Table Bay where the lepers are removed from Cape Town.

In view of these conflicting statements, it seemed that further and more numerous observations on vaccine lymph taken from lepers would be of value.

A suitable opportunity occurred at the Almora Asylum, where all the lepers who were well enough were vaccinated, amounting to some eighty-seven in all.

Of these forty (twenty-one males, nineteen females) developed vesicles, which were examined. Of the forty cases, thirty-four were anæsthetic, five mixed, and one was tuberculated.

The condition of the skin where vaccinated was as follows:—

In 14 cases it was normal.
„ 13 „ there was an anæsthetic patch.
„ 12 „ sensation was diminished.
„ 1 case it was tuberculated.

The condition of the vesicle was as follows:—

In 31 cases normal.
„ 2 „ purulent.
„ 2 „ purulent and mixed with blood.
„ 1 case normal one arm and purulent on the other.
„ 1 „ normal, but mixed with blood in taking.
„ 1 „ immature.
„ 1 „ immature and mixed with blood in taking.
„ 1 „ immature and the crust taken.

Crusts also were taken from two vesicles which had been normal.

Ninety-three cover-glasses were prepared and stained with Ziehl's solution, passed through acid and then treated with methyl-blue. In no case were Leprosy Bacilli found. Suspicious-looking rods taking fuchsin were found in one case in vesicles raised over tuberculated ears, and in another case in vesicles over anæsthetic patches.

But even if these cases be considered as positive, and the utmost value allowed to them, they would have little or no importance in the question at issue, for no vaccinator would be likely to vaccinate a leper over a tubercle or anæsthetic patch, and use the lymph obtained from such vesicles for vaccinating healthy individuals.

It is therefore our opinion that assuming the presence of the bacillus to be necessary to produce leprosy, this series of observations, which is the most extensive yet made, conclusively shows that no danger need be apprehended from the vaccine lymph of even an actual leper, provided

he has been vaccinated on normal skin. *Á fortiori*, therefore, there is no danger of transmitting leprosy by using as a vaccinifer a child born of a family in which leprosy exists, provided reasonable care be taken.

The following table gives a detailed account of the observations made in Almora:—

No.	Name.	ALMORA ASYLUM. AGE. M.	AGE. F.	Form.	Years afflicted.	Condition of skin where vaccinated.	VACCINATION. Condition of vesicle.	Date of vaccination.	Date of taking lymph.	No. of cover-glasses.	Result of Microscopic Examination.
1	Amarna .	25	...	M	13	Normal . .	R. arm purulent	4-3-91	22-3-91	2	1. Numerous diplococci stained blue. 2. Numerous diplococci stained blue: also few small rods stained blue.
2	„ .	25	...	„	13	„ . .	L. arm normal	4-3-91	22-3-91	4	1. Few small badly stained rods and diplococci. 2, 3, 4. Negative.
3	Bachi .	25	...	A	20	Sensation diminished.	Normal, but the lymph was mixed with blood in taking.	19-3-91	25-3-91	2	1. Few pale rods: no Leprosy Bacilli. 2. Debris and rods: no Leprosy Bacilli.
4	Mongolia .	26	...	A	15	Anæsthetic patch	Normal . .	19-3-91	25-3-91	4	1. No bacilli. 2. Few small rods stained blue. 3. Few small rods not stained. 4. Debris and rods: no Leprosy Bacilli.
5	Teliga .	...	35	M	20	„ „	„ . .	19-3-91	25-3-91	2	1. Three badly stained rods; no Leprosy Bacilli. 2. No Leprosy Bacilli.
6	Jitna .	30	...	M	3	„ „	R. arm normal	21-3-91	27-3-91	2	1. Suspicious rods taking fuchsin. 2. Suspicious rods taking fuchsin.
7	Jitna .	30	...	M	3	Anæsthetic patch	L. arm normal .	21-3-91	27-3-91	2	1. Suspicious rods and cocci taking fuchsin. 2. Suspicious rods and cocci taking fuchsin.

No.	Name.	ALMORA ASYLUM. Age. M.	F.	Form.	Years afflicted.	Condition of skin where vaccinated.	VACCINATION. Condition of vesicle.	Date of vaccination.	Date of taking lymph.	No. of cover-glasses.	Result of Microscopic Examination.
8	Jogia .	45	...	A	33	Sensation diminished.	Normal . .	24-3-91	30-3-91	2	1. Small blue rods, epithelial cells, with nuclei taking methyl-blue readily. 2. Small blue rods: epithelial cells, with nuclei taking methyl-blue readily.
9	Rhagia .	35	...	,,	2½	Normal . .	,, . .	24-3-91	30-3-91	2	1. No Leprosy Bacilli: epithelial cells with nuclei. 2. Epithelial debris, lymph cells, rods, and micrococci. No Leprosy Bacilli.
10	Gumyana .	35	...	,,	19	Sensation diminished.	,, . .	24-3-91	30-3-91	1	1. Wholly negative.
11	Jaikishen .	35	...	M	29	Tuberculated (ear).	,, . .	25-3-91	1-4-91	2	1. One or two bacilli taking fuchsin. 2. Debris: no bacilli.
12	,, .	...	...	,,	29	,, . .	,, . .	25-3-91	1-4-91	2	1. One or two badly stained bacilli. 2. Debris: no bacilli.
13	Chandra .	...	60	,,	1	Normal .	,, . .	28-3-91	1-4-91	2	1. Few small blue cocci: no bacilli. 2. Debris: no bacilli.
14	Bhaisaki .	...	36	A	20	,, . .	,, . .	28-3-91	1-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
15	Heroli .	...	32	,,	15	Sensation diminished.	,, . .	28-3-91	3-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
16	Deluli .	...	45	,,	12	Normal . .	,, . .	28-3-91	6-4-91	2	1. No Leprosy Bacilli. 2. No Leprosy Bacilli.
17	Jaikishen .	35	...	M	29	Tuberculated (ear).	Crust from broken vesicle.	25-3-91	6-4-91	2	1. No bacilli. 2. Few bacilli stained blue: no Leprosy Bacilli.
18	Gumyana .	35	...	A	19	Sensation diminished.	Crust from broken vesicle.	24-3-91	6-4-91	2	1. Numerous cocci stained blue: no Leprosy Bacilli. 2. Numerous cocci stained blue: no Leprosy Bacilli.

No.	Name.	ALMORA ASYLUM. Age. M.	F.	Form.	Years afflicted.	Condition of skin where vaccinated.	VACCINATION. Condition of vesicle.	Date of vaccination.	Date of taking lymph.	No. of cover-glasses.	Result of Microscopic Examination.
19	Gulavi .	...	50	A	17	Anæsthetic patch	Normal . .	30-3-91	4-4-91	2	1. Unstained bacilli: no Leprosy Bacilli. 2. Debris: no Leprosy Bacilli.
20	Bhuri .	...	50	,,	35	Normal . .	,, . .	30-3-91	4-4-91	2	1. Blue rods: no Leprosy Bacilli. 2. Numerous cocci and diplococci: no Leprosy Bacilli.
21	Bhaguli .	...	2[illegible]	,,	10	,, . .	,, . .	30-3-91	4-4-91	2	1. Few blue rods: no Leprosy Bacilli. 2. Few blue rods: no Leprosy Bacilli.
22	Ganguli .	...	50	,,	22	Sensation diminished.	,, . .	30-3-91	6-4-91	2	1. Group of vaccine cocci stained blue: no Leprosy Bacilli. 2. Few bacilli stained blue: no Leprosy Bacilli.
23	Dikvi .	...	40	,,	8	Anæsthetic patch	,, . .	31-3-91	6-4-91	2	1. Short thick bacilli taking pink stain: no Leprosy Bacilli. 2. No bacilli.
24	Bachali .	...	40	,,	5	,, ,,	,, . .	31-3-91	6-4-91	2	1. Debris: no Leprosy Bacilli. 2. Debris: no Leprosy Bacilli.
25	Parmati .	...	40	,,	34	Sensation diminished.	,, . .	30-3-91	6-4-91	2	1. Debris: no bacilli. 2. Few bacilli taking blue stain: no Leprosy Bacilli.
26	Naduli .	...	50	,,	20	,, ,,	,, . .	30-3-91	6-4-91	2	1. Numerous cocci stained blue. 2. Numerous leucocytes: no Leprosy Bacilli.
27	Gopuli .	...	30	,,	10	,, ,,	,, . .	30-3-91	6-4-91	2	1. Debris: no bacilli. 2. Few cocci; no bacilli.
28	Khimuli .	...	50	A	20	Sensation diminished.	Normal . .	30-3-91	6-4-91	2	1. Rods of various sizes: no Leprosy Bacilli. 2. Rods of various sizes; no Leprosy Bacilli.

No.	Name.	ALMORA ASYLUM. Age. M.	F.	Form.	Years afflicted.	Condition of skin where vaccinated.	VACCINATION. Condition of vesicle.	Date of vaccination.	Date of taking lymph.	No. of cover-glasses.	Result of Microscopic Examination.
29	Himatna	60	...	A	12	Sensation diminished.	Normal	1-4-91	7-4-91	2	1. Debris: no Leprosy Bacilli. 2. Debris; no Leprosy Bacilli.
30	Jasuli	...	41	,,	19	Normal	Purulent and mixed with blood.	30-3-91	7-4-91	2	1. Debris: no bacilli. 2 Cocci and diplococci: no bacilli.
31	Dhania	25	...	,,	4	Anæsthetic patch	Normal	1-4-91	7-4-91	2	1. Cocci and bacilli stained blue: no Leprosy Bacilli. 2. Cocci and bacilli stained blue: no Leprosy Bacilli.
32	Karakna	25	...	,,	13	Sensation diminished.	Imperfectly developed.	3-4-91	10-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
33	Khimuli	18	...	,,	13	Normal	Purulent	3-4-91	10-4-91	2	1. Pus cells and developing fibrous tissue: no bacilli. 2. Pus cells: no bacilli.
34	Partima	...	55	T	10	,,	Normal	5-4-91	11-4-91	2	1. Bacilli stained blue and shreds of tissue. 2. Bacilli stained blue and shreds of tissue.
35	Motiya	20	...	A	10	,,	,,	8-4-91	13-4-91	2	1. Numerous cocci: no Leprosy Bacilli. 2. No Leprosy Bacilli.
36	Kamalua	40	...	,,	28	Sensation diminished.	,,	8-4-91	13-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
37	Budhia	32	...	,,	1	Anæsthetic patch	Purulent and mixed with blood.	8-4-91	13-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
38	Telegu	...	60	,,	30	,, ,,	Immature vesicle and mixed with blood.	9-4-91	13-4-91	2	1. Cocci and bacteria: no Leprosy Bacilli. 2. Cocci and bacteria: no Leprosy Bacilli.
39	Sundarsai	40	...	,,	7	Normal	Normal	9-4-91	13-4-91	2	1. Cocci: no bacilli. 2. Cocci: no bacilli.
40	Gujia	45	...	A	17	Normal	Normal	8-4-91	13-4-91	2	Debris: no bacilli. 2. Debris: no bacilli.

No.	Name.	ALMORA ASYLUM. Age. M.	F.	Form.	Years afflicted.	Condition of skin where vaccinated.	VACCINATION. Condition of vesicle.	Date of vaccination.	Date of taking lymph.	No. of cover-glasses.	Result of Microscopic Examination.
41	Kalangit	28	...	A	3	Normal	Normal	8-4-91	13-4-91	2	1. Bacteria: no Leprosy Bacilli. 2. Bacteria: no Leprosy Bacilli.
42	Gumani	28	...	,,	20	Anæsthetic patch	Purulent	10-4-91	13-4-91	2	1. Pus cells: no Leprosy Bacilli. 2. Pus cells: no Leprosy Bacilli.
43	Harna	54	...	,,	30	,, ,,	Normal	8-4-91	13-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
44	Maina	...	60	,,	30	,, ,,	,,	9-4-91	14-4-91	2	1. Debris: no bacilli. 2. Debris: no bacilli.
45	Dhamia	50	...	,,	12	,, ,,	Crust of imperfect vesicle.	9-4-91	14-4-91	2	1. Large rods taking blue stain: no Leprosy Bacilli. 2. Debris: no bacilli.

V.—CULTIVATION EXPERIMENTS.[29]

Hansen, in his original paper, described both rod-formed bodies (Bacillus Lepræ) in tubercles, and jointed threads obtained in specimens of blood which, when kept in a moist chamber, developed into a mycelial growth, as probable specific microscopical features of true leprosy. He also figured these granular mycelial masses, and spoke of them as a culture, but it is almost certain that this must be looked upon as a contamination of the specimen, and since the Bacillus Lepræ never occurs in blood, this growth could not have been a development of these. It is clear also that Hansen himself did not regard the mycelium in this light.

The cultivation experiments of Neisser[30] are the earliest, and the details may be given in his own words.

1. An unbroken tubercle was thoroughly cleansed with alcohol, then removed with every possible precaution against foreign infection, and finely

(29) The references available in India for this section are exceedingly few.
(30) Virchow's Archiv. 1881, 1886.

divided up with needles made sterile in a flame. Particles of this broken-up material were employed in the following three culture methods:—

(*a*) With blood serum in hollow-ground microscope slides. The hollow spaces were very capacious and contained much air.

(*b*) In test tubes, carefully cleansed by heat and acids, which were partly filled with blood serum or with alkaline sterilized flesh-extract-solution.

(*c*) Under cover-glasses with blood serum, in a space where evaporation was checked. All these preparations were kept permanently in a warm chamber at 35°—39°C.

2. Cultivations from pus and juice of leprous nodule which were protected in capillary glass tubes from accidental fouling. The tubes contained much air and were sealed.

Neisser describes three bacilli: first, movable rods which are perfectly free from distortion of any kind; second, motile rods with a minute ball-like swelling at one or both extremities or in the middle; these rods are somewhat longer than the first variety and frequently sharply bent; third, bacilli which are characterised by being distinctly longer and broader than either the first or second kind; each of these rods contains one to four clear unstained spaces (vacuolation). It is possible, according to this observer, that the breaking up of the rod into granules (kokkothrix) which lie in a row adhering together because of the hull which invests the bacillus, is a retrogressive metamorphosis, and that the bacilli of the second and third kinds exhibit veritable spore-formation. Lastly, both in cultures and in microscopical preparations of leprous tubercles which, after experimental introduction, had been encapsuled in the peritoneal cavity, the bacilli grew into a thread occasionally four times the normal length, but in these no sign of anything which could be called a spore-formation was evident.

From a careful examination of Neisser's figures, especially 1 and 10, it appears not unlikely that, as he claims, a veritable culture was obtained. Further, the three kinds of bacilli described by him are to be seen in juice from a leper tubercle. Of the actual existence of two—the first and third kinds—there can be no question, but the relation of these to each other is not so clear; both stain excellently with the Koch-Ehrlich method and resist decolorization with acids; and also Gram's method clearly defines both of them, especially the latter. Neisser's first kind is the normal Bacillus

Lepræ; the second we believe to be possibly a distortion, for a swelling upon a rod can easily be caused to appear and disappear, as the bacillus slowly turns under the cover-glass; and the appearance seen is figured on Plate III, Fig. VIII; the third form, as far as we can judge from his Figs. 7 and 13, we have frequently seen and have also, we believe, cultivated. Whether it is a bacillus possessing pathogenic power it is impossible to say. This form, which it is very probable is the same as that described by Neisser, we shall speak of as the vacuolated bacillus. In a later paper Neisser,[31] as the result of extended observations, inclines to the view that the vacuoles, or spaces in the bacillus, are veritable spores, though in no instance has he seen an isolated spore. The ball-like swelling in the second kind he does not regard as a spore-formation. He also confirms the presence of the hull of the bacillus, stating that it can easily be seen after staining a preparation with watery dye. The presence of this hull is botanically of interest; it occurs also in the Bacillus Tuberculosis,[32] Pneumococcus,[33] and several other forms.

Further culture experiments by Neisser consisted in using solid blood serum and hard-boiled fowl or duck eggs as a nutrient medium, and minute pieces of excised tubercle, or blood from a tubercle, as material for inoculation. The culture was maintained at a temperature of 37°—38°C. The growth was exceedingly slow. Cultivation in generations was not successful. Specimens from detached bacillary islets on the medium yielded what the author held to be a genuine cultivation of the Bacillus Lepræ.

Hansen [34] in 1882 described a culture on serum of bacilli which were motile. He used methylene-blue as a stain, but according to Neisser this will not even tinge the genuine Bacillus Lepræ. This is not a strong argument against Hansen's culture, for in our hands methyl-blue stains the Bacillus Lepræ, though great differences exist in the samples of this dye, so that the discordant results of various observers are conceivable.

It is impossible to be quite certain that a veritable cultivation of the Bacillus Lepræ was obtained by Neisser. On Plate XII,[34] Figs. 1, 5, 6, and especially 10, have the appearance of true cultures, but when it is considered that this observer wholly failed to cultivate the bacillus in

([31]) Virchow's Archiv. 1886.
([32]) Lutz and Unna. Dermatol. Studien Heft I. 1886.
([33]) Friedländer. Fortschr. d. Med. Bd. III, 1885.
([34]) Virchow's Archiv. 1881.

generations, an actual multiplication in fluids which probably did occur is all that can be conceded.

Manson,[35] some years before Hansen had discovered the Bacillus Lepræ attempted by incubation of leper juice to discover the then unknown germ. Sealed capillary tubes were inserted into eggs which were placed under a hen. Beyond the development of streptococci, both free and in the zoogloea state, these experiments gave wholly negative results.

Arning[36] states "that there is no difficulty whatever in growing the Leprosy Bacillus, by simply allowing pieces of leprous tissue to macerate in ordinary water, at ordinary temperatures, giving but sufficient time; but the resulting growths are not pure cultures in a strict sense."

The introduction by Koch of solid nutrient media has, to a large extent, caused nearly all observers who have attempted to cultivate the Bacillus Lepræ to abandon cultivation in fluid media which in the hands of Pasteur had led to many discoveries. In the history of the attempted cultivation of the specific bacillus of leprosy, the great majority of experiments have consisted in the removal of a piece of leprous tubercle and the direct implantation of this upon a solid nutrient surface, such as agar, glycerin-agar, or solidified serum.[37] The successful growth of Bacillus Tuberculosis by this method is by no means easy, and uniformly negative results with the Bacillus Lepræ have been obtained by the vast majority of observers.[38] Quite recently Sheridan Delépine and Slater[39] record numerous attempts to cultivate the bacillus, which were made independently at two different periods during the life of the patient. On both occasions the results were negative. The latter observer two years previously had attempted identical cultivation experiments.

In 1887[40] Bordoni-Uffreduzzi described at length his culture-experiments with the Bacillus Lepræ. He was as unsucessful as previous observers in the direct implantation of a leprous nodule on to a solid nutrient medium. The chief result of such a proceeding was a growth of Streptococcus Pyogenes.

(35) Journal of the Leprosy Investigation Committee, No. 1, p. 40.

(36) Journal of the Leprosy Investigation Committee, No. 2, p. 117.

(37) Fraenkel and Franke repeatedly failed to cultivate the Xerosis Bacillus in this manner by direct implantation on solid media. Schreiber, however, succeeded in this direction.

(38) Among others *cf.* Trinidad Leper Asylum Reports, 1888.

(39) British Medical Journal, May 23rd, 1891, page 1127.

(40) Zeitschrift für Hygiene, Vol. III, page 178. This important paper was unfortunately not available in India.

He considered that the Leprosy Bacilli which abound in leprous nodules are enclosed in cells, and probably are not alive, though, if the view of Unna and Kühne be accepted, the bacillary heaps lie outside the cells. In the marrow of bones of a dead leper, the Italian observer found free bacilli, and from this material he raised cultivations of the Bacillus Lepræ on peptone-salt, glycerine-serum; it grows also upon glycerine-agar. However, the bone-marrow has been examined by Kaurin[41] in many cases, and he never succeeded in finding the Bacillus Lepræ. It does not appear that many observations on bone-marrow have been made, but at any rate in the Haversian canals and spaces of the bones of the little finger, which presented typical mutilation, masses of bacilli, both discrete, and lodged within undoubted lepra cells, have been found by Delépine and Slater.[42] Sawtschenko[43] has also worked out the histology of bone-leprosis.

Bordoni-Uffreduzzi describes the cultivated bacillus on peptone-glycerine-serum at a temperature of 35°—37°C. as a light-yellow tænia-form stripe with irregular edges along the needle-track. The serum is never liquefied, and no growth ever occurs in the condensation water. On glycerine-agar a similar culture develops if a great quantity of bacillary material be streaked upon the nutrient surface. If a smaller amount be streaked, isolated lens-formed colonies with jagged edges and of varying size develop. On glycerine-agar plates, both on the surface and deeply in the medium, colonies may be seen with a power of 100 diameters, which are grey, net-like growths with irregular edges. It is evident that this growth cannot be confounded with cultures of Bacillus Tuberculosis. The isolated bacilli in form and measurement resemble those of the tissues, but a striking peculiarity is the frequent occurrence, at one or both ends of a bacillus, of a swelling in which an arthrospore may be seen. This appearance may also be recognised in the tissues, but it is an open question whether this does not represent an involution-form. Isolated spores, or the development of a rod from such an arthrospore, have not been observed. The bacillus is quite immotile. The tinctorial behaviour of the cultivated Bacillus Lepræ is characterised, firstly,

(41) Journal of the Leprosy Investigation Committee, Vol. 2, p. 68.

(42) Loc. cit.

(43) Sawtschenko über Osteomyelitis Leprosa. Centralblatt. f. Bact. und Parasitenkunde, Bd. V., 1889. According to Sawtschenko the vacuoles in lepra cells contain a liquid which is secreted by the bacilli. The absorption of bones proceeds from lepra clusters in the bone-marrow and in the dilated Haversian canals.

by remaining absolutely unstained by alkaline methyl-blue,[44] in which it resembles the bacillus of the tissues, and secondly, by staining with much greater difficulty in Ehrlich's fuchsin, indeed, with about the same facility as the Bacillus Tuberculosis does. The Bacillus Lepræ of the tissues, however, stains much more readily than the latter. In a great number of experiments on rabbits and other animals, Bordoni-Uffreduzzi wholly failed to establish the pathogenic nature of his cultivation.

Roux, Nocard, Chantemesse and Cornil[45] have failed to confirm the above cultivation experiments, but Gianturco[46] has succeeded in obtaining a culture which Bordoni-Uffreduzzi admits is absolutely identical with his own. At a temperature of 37° C., after seven days, a colony is developed of feebly motile bacilli, which are more slender when grown on glycerine-blood-serum than when on glycerine-agar. The bacilli also possess terminal swellings (arthrospores). Both Campana[47] and Katz[48] using every precaution, have failed to cultivate the Bacillus Lepræ. The former observer, in the course of two years, made about five hundred experiments and employed various nutrient media and material from leprous tubercle. In consequence of the total failure of these experiments he has thrown doubt on the genuineness of the culture obtained by Bordoni-Uffreduzzi.

Extensive experiments which were made in Trinidad, with total want of success, and consisting in the direct implantation of the centre of a non-ulcerated leprous tubercle, taken with every possible precaution, upon a nutrient solid medium, were repeated in India with similar result. No culture was formed, but a staphylococcus growth spread very slowly from the piece of tissue, and by the end of ten weeks the implanted piece was obscured by this growth.

The following methods were adopted in our experiments:—

1. It had been ascertained that in blisters raised over unbroken leprous tubercles, specific bacilli were frequently found. It is also well known that the pigment particles in tattoo-marks do not pass into such a fluid. It was therefore hoped that by this means a separation could be effected between the living and dead bacilli within the tubercle. Serum from

(44) Koch and Wesener have stained Bacillus Lepræ, and this can be confirmed with great ease.
(45) Extr. du Bulletin de l'Academie de Medicine. Séance du 19th Juin, 1888.
(46) Abstract in Baumgarten's Jahresbericht, Vol. II, 1889.
(47) Abstract in Baumgarten's Jahresbericht, Vol. III, 1889.
(48) Abstract in Baumgarten's Jahresbericht., Vol. III.

blisters over the normal skin of the back of a leper was received into sterile vaccine capillary tubes, and a small quantity of the fluid of a blister raised over a tubercle, was added to this. The tube, which had an air-space for about one-third of its length, was then sealed and maintained at the temperature of the body.

2. Pieces of the centre of a leprous tubercle, removed with all possible precautions, were introduced into bouillon, and maintained at the temperature of the room, which during the day averaged 27°C.

3. An unbroken tubercle was thoroughly cleansed with carbolic soap and perchloride solution. A deep puncture with a sterile needle was made into the tubercle, and perfectly clear leper juice was expressed. This was received into sterile pieces of capillary tube, about half an inch long, and the tube and contents at once dropped into bouillon. This was maintained at the temperature of the room.

In all the above instances microscopical examination showed that the material used as the source of cultures contained Leprosy Bacilli. The results of these three methods will now be given in detail.

From what has been previously mentioned, it would appear that the direct implantation of leprous material upon solid nutrient media gives absolutely negative results. Among the causes of this it may be suggested that the Bacillius Lepræ, like the bacillus of malignant œdema,[49] is exquisitely anäerobic, or may be incapable of becoming a facultative saprophyte, or that in the preparation of the piece of leprous tissue, the hot knives used to make the section cause the formation of a layer of coagulated material, through which the bacilli cannot make their way, or, lastly, because the tissue contains, for the most part, only dead bacilli. The last conjecture appears most probable, since the others admit of direct experiment.

If a section of skin be made through a leprous tubercle the bacilli are found in quantities, and fluid expressed from the neoplasm abounds in bacilli. The epidermis from the corneous to the pigmentary layer in unbroken tubercles is wholly free from bacilli, while the cutis and subcutaneous tissue is crowded with them. A blister raised over a tubercle contains a few leucocytes and at times bacilli. In some cases these latter are absent. We believed that the majority of these bacilli were alive and capable of being cultivated.

(49) Discovered by Koch and first cultivated in bouillon with absence of oxygen by Pasteur.

Cultivation Experiments with Blister Fluid.

Blisters were raised over tubercles on the ears, face and wrists, and at the same time over normal skin between the shoulder blades. After avoiding all sources of fouling, sterilised vaccine capillary tubes were charged with a minute quantity of fluid from a blister over a tubercle, and also with seven or eight times the quantity of fluid from a blister over the normal skin of the same leper. They were then sealed. Each tube also contained air for about one-third of its length. The dates of these experiments ranged from March 31st to May 13th. In all cases samples of the blisters, both over the tubercles and on the back, were examined for the bacillus. Being at this time without an incubator, the tubes were packed with a small thermometer in a cylindrical box and carried in the axilla, a proceeding adopted by Spallanzani in the last century when studying the action of the digestive juices on food. A month later these tubes were examined and resealed, and in several cultures were seen. A tube containing a culture was now sterilised as far as was possible without injury to the contents, broken at both ends, and dropped into fluid glycerine-bouillon and shaken carefully. In this bouillon a culture of bacilli, which we believed to be Leprosy Bacilli for the reasons given below, developed, a slight turbidity occurred about the third day and a pellicle subsequently formed on the surface, so that a good tenacious scum was produced by the twelfth day. In the mode of formation of this pellicle, minute particles appeared on the surface of the bouillon, which daily increasing in number, joined together, so that the whole growth could be seen steadily advancing from the centre to the periphery. In some cases, a fortnight later this pellicle sank partly in the fluid. From such original bouillon cultures, gelatine and glycerine-agar media were inoculated. The growth liquefied the gelatine, and grew also well on agar. Generations up to the sixth were carried on at Simla from April 26th to August 20th, 1891. Out of about twenty-five experiments (some of which failed to give any culture in the capillary tube), the following may be given as examples:—

1. Blister fluid over the tubercle of Puniya, and fluid taken from a blister over normal skin of the back of same leper, mixed in a sealed capillary tube with an air space. Taken on April 14th, 1891, kept at body temperature until May 2nd, when part of the contents was examined. Leprosy Bacilli brightly stained were found. The tube was resealed, and on May 14th, this was broken and dropped into bouillon. The tubes were kept at a temperature of 26°C. On May 25th a minute white speck was seen floating on the surface of the liquid. Two days later, a thin film, most dense in the centre, almost covered the surface of the liquid. A small amount of this film subsided to the bottom of the test-tube. Microscopic examination showed rods which were very feebly motile; they did not change position, but slowly bent and returned to the straight condition. Most of the bacilli were in clusters. In measurement and shape these were undistinguishable from Leprosy Bacilli. Specimens stained in warm Ziehl's solution for twenty minutes and decolorised in 25 per cent. nitric acid, were stained pink. No beaded appearance was seen. On May 28th two specimens of this growth were stained for twenty-four hours in Ziehl's solution, decolorised and found to show quantities of brightly stained bacilli, separate and in clusters. On May 29th the bouillon was covered with a coherent pellicle. By June 1st the pellicle had in great part subsided *en masse* to the bottom of the tube.

Cultures in glycerine-bouillon, glycerine-gelatine, and glycerine-agar were made from this original growth and carried on for several generations. The growth in gelatine shows on the second day a minute white speck on the surface, and subsequently a liquefaction takes place along the needle track. This appearance is represented in Fig. 2, Plate II. Subsequently two strata of growth form, separated by a clear liquid space. The microscopical appearance of these strata is identical as far as staining with the Ziehl method is concerned; both consist of bright red rods, but those from the upper stratum are slightly thicker than those from the lower one.

The growth on agar presents an irregular outline at the edge of the needle stroke, and this extends rather rapidly, so that a great part of the surface is covered. Its subsequent spread is, however, exceedingly slow.

All these growths in bouillon, gelatine and agar were pure and undoubtedly identical, and the bacilli stained excellently, and resisted decolorisation with 10 per cent., 15 per cent. and 20 per cent. nitric acid. This resistance varied at different periods of growth, being greatest in the early stages. Subsequent generations, though retaining the shape of the original, did not resist the action of the acid so well, but still stained to some extent. The individual bacilli were shorter and slightly thicker than those obtained in alcohol hardened leprous tubercles. They also had a distinct tendency to aggregate into clusters and lines.

This series of experiments was repeated and the details were as follows:—

2. Expressed tubercle juice of Bishan Singh, and fluid from blister over sound skin of the same leper, was collected in a sealed capillary tube on April 14th; examined on May 6th and 7th, stained bacilli were found in strings and in groups. No admixture of other bacilli found. A bouillon tube was inoculated and a growth commenced. This culture however fouled a week later.

3. Expressed tubercle juice of Bishan Singh, and fluid from a blister over the sound skin of the back of the same leper, was taken on April 14th; examined on May 8th, numbers of stained bacilli, which had the appearance of a culture, were found. No other bacteria

present. The tube was resealed and again examined on May 14th; no bacilli were found. A bouillon tube, inoculated on May 14th, was the commencement of a series similar to Series I.

Cultivation experiments with Excised Tubercle in Bouillon.

Rahim Khan, a Muhammadan, formerly resident in Thibet, has very marked tubercular leprosy. The disease commenced two and a half years ago, and the patient is at present in the full height of the disease; the lobes of both ears are enormously pendulous and thickened. The whole aspect of the man indicates that he is extensively diseased. The uvula is destroyed and there is extensive ulceration of the fauces and the raucous voice indicative of laryngeal trouble. His saliva and sputum abound in nests of Leprosy Bacilli. It was determined in this case (and the results of the treatment were a great improvement in the health of the patient) to excise the tuberculated masses in the ears. Accordingly, on May 22nd, 1891, the whole lobe of the left ear was excised with all antiseptic precaution. The mass was placed in a sterile plate, and with sterilised knives, one cut being made with only one knife, pieces from the centre were placed in bouillon, gelatine and on glycerine-agar. Nine tubes were prepared in this way, seven being bouillon. The pieces of tissue abounded in bacilli. From none of these experiments were Leprosy Bacilli obtained. The growth on agar about a week later showed streptococcus chains, and the gelatine presented the same growth; the other tubes, with one exception, became turbid and foul, but in this isolated instance a bacillus shown in Fig. 7, Plate III, was obtained, and successive pure cultivations raised upon gelatine-agar and bouillon. The bacillus, which was certainly obtained from the tissues, and was never seen in numerous accidental foulings, we believe is probably the third variety figured by Neisser. It stains exactly like the bacillus of the tissues with Ziehl's solution, and resists powerful decolorisation with acids. Gram's method makes its structure very evident, and it stains also by the method employed by Giacomi for the Syphilis Bacillus (?) of Lustgarten. Aqueous methyl-blue and most of the common aniline dyes stain this vacuolated bacillus perfectly.

This vacuolated bacillus is absolutely immotile even in the early stage. At this period a rod-shaped organism, in length and breadth exceeding the typical Bacillus Lepræ, but smaller than Bacillus Subtilis, is seen aggregated into clusters and now shows no trace of vacuolation. This subsequently

occurs when the culture is about one week old, and one to four clear unstained spaces are to be seen in the rod, which is slightly increased in length. Bacilli, shorter than Bacillus Lepræ, with only one ovoid space, are also to be seen, and occasionally a club-shaped form with a terminal space. At all periods of growth this bacillus stains admirably by the methods employed as diagnostic of the Bacillus Lepræ of the tissues. We do not believe these clear spaces are spores. In no instance were isolated spores seen. The study of the bacillus in hollow ground glass slides did not throw any light on this question.

The growth of this vacuolated bacillus in bouillon is very like that formerly described for the bacillus obtained by the blister method. In gelatine the appearance is, however, so characteristic that it was easily possible to at once detect the culture. A speck growing to a white disc appears after twenty-four hours, the needle track at the same time commencing to form a stocking-like liquefaction in the gelatine. At the end of sixty hours a tenacious pellicle forms on the surface of the gelatine, and a flocculent deposit settles down to the lower part of the liquefied stocking-like tract. Subsequently the pellicle becomes still denser, and by the end of ten days the growth has almost ceased, all the gelatine is fluid, and a flocculent loose deposit occurs at the bottom of the tube. The growth on glycerine-agar is much slower, and there is nothing very typical about this.

On June 20th the lobe of the right ear of the same leper was excised, and with all precautions, pieces of tuberculated tissue were placed in six tubes of bouillon and two of gelatine. The cultures were kept at the room temperature, which averaged 25°C. On the eleventh day in two of the bouillon tubes distinct turbidity was noticed, and both tubes showed bacilli closely resembling the Leprosy Bacilli of the tissues in shape and staining reactions. No other bacilli were present. The vacuolated form was not met with. A pellicle formed on the surface three days later and the culture was still found to be pure. Generations from these original growths were carried on in bouillon and gelatine and agar, and these remained quite pure for some weeks, when great fouling occurred in many tubes.

This bacillus, as far as we could judge, exactly resembled the one obtained in our earliest experiments. It stained excellently with Ziehl's solution and resisted decolorisation with 26 per cent. nitric acid. As is figured in Fig. 2, Plate III, a hull becomes very apparent around the bacilli, and this is capable of being well shown with watery methyl-violet and to a less

extent by the method employed for showing the capsule of Friedländer's Pneumococcus.[50] The aggregation of these bacilli into groups is most peculiar, and the appearance figured in Plate III, Fig. 4, recalls very much the arrangement within the tissues, masses of bacilli being collected together into clusters, due possibly to the nature of the hull of the bacillus.

Cultivation Experiments with Juice from Leprous Tubercle.

On July 8th an unbroken tubercle on the elbow of the same leper was scraped with a knife, washed with carbolic soap and perchloride solution, and after puncture with every precaution, leper juice was collected in a sterile capillary tube and dropped into glycerine-bouillon. Six tubes were prepared in this way, of which four rapidly became fouled with growth of Bacillus Subtilis and staphylococci; the other two remained pure and were kept in the incubator at 37°C. In both these tubes free bacilli, undistinguishable from Leprosy Bacilli, were found about the tenth day. We did not have the opportunity of continuing this culture on agar.

As will be seen from the next section, all attempts to establish the pathogenic character of the three bacilli described above were without success, and consequently it is impossible to affirm with certainty that the cultivation of the Bacillus Lepræ has been accomplished by us. We do, however, claim to have obtained the same bacillus from the tissues of lepers by three different methods. Allowing that the form of this cultivated bacillus is slightly broader than that met with in the tissues, this alone would not constitute a serious distinction, for, as is well known, bacilli, which are specific pathogenic parasites, vary greatly in size, according to the nature of the medium[51] on which they grow, and further the same specific bacillus varies in size according to the animal in which it occurs.[52] Lastly, the aspect of the cultivated Bacillus Lepræ is unknown to us. Also, as far as staining reaction and measurements can serve as criteria, the bacillus we have figured does correspond with that of the tissues. In the course of numerous foulings of cultures neither this form nor that of the vacuolated bacillus were ever found as a contamination, and we are convinced that both these forms were obtained

(50) Friedländer, op. cit.

(51) See Buchner's figures of Bac. Anthracis in Zopf's Die Spaltpilze.

(52) K. Huber, Deutsche Med. Wochen. 1881, No. 8. Bac. Anthracis is largest in mice, smallest in the cow, and of a mean between these in rabbits and guinea-pigs.

from tubercular masses. No skin was adherent to the pieces used as a source of culture. It is on the staining properties that we rely, and we believe that we have cultivated two bacilli of different morphological form which respond to the staining methods used as diagnostic of the Bacillus Lepræ. However, the rapidity of the growth of the subcultures greatly exceeds that of the original. This has surprised us, for we should have expected that the rate of multiplication of the specific bacillus of a disease such as leprosy, which is so essentially chronic, would have been slower.

Quite recently Campana[53] found in cultivations of tissue from tuberculous leprosy a bacillus which morphologically entirely resembled Bacillus Lepræ. Treated by Ehrlich's method this did not take on a double staining. In two cases this bacillus grew in agar-agar and in meat peptone to which 3 per cent. of grape sugar was added. The bacillus did not grow in fluid media. Inoculation experiments on rats were negative. Campana does not infer that this bacillus is identical with Bacillus Lepræ, since he only found a resemblance between the two. It is perhaps possible, though we do not incline to this view, that the bacillus described by Campana may resemble that of our cultures.

We refrain from definitely asserting that our culture is the Leprosy Bacillus, since it apparently possesses no pathogenic power. Possibly leprosy, like syphilis and cancer,[54] is an essentially human disease. We propose to continue these cultivation experiments as opportunity offers.

VI.—TRANSMISSIBILITY OF THE DISEASE BY EXPERIMENT.

Keanu, a Hawaiian man in Oahu Gaol, in consideration of the commutation of his sentence of capital punishment, voluntarily submitted to inoculation of a leprous tubercle under the skin of the left fore-arm. This was performed by Dr. Arning on September 30th, 1884,[55] and at this time no symptom of leprosy was present, and no family history of the disease could be obtained. In four weeks' time the patient suffered from rheumatic pains in the left shoulder, and subsequently also in the elbow and wrist joints.

(53) Campana (Reforma Med. No. 14, 1891), quoted from British Medical Journal of June 20th, 1891.

(54) Hanau's rats became affected with cancer, not in consequence of the introduction of cancer from the human subject, but of a cancerous growth taken from another rat.

(55) Journal of the Leprosy Investigation Committee, No. 2, 1891, page 132, where Arning's paper in Archiv. für Dermat. und Syph. January 1891, is translated.

Accompanying these symptoms there was a painful swelling of the ulnar and median nerves; fever was absent. By the end of six months the neuritis had gradually decreased, and a small leprous tubercle had formed on the keloid growth at the site of inoculation. Leprosy Bacilli could be detected in the tissues of this scar sixteen months after inoculation. Definite symptoms of leprosy existed in September 1887, and in another year the patient was at the full height of the disease.

In estimating the value of this experiment it must be remembered that Keanu's son, nephew, and maternal first cousin were lepers either before or after the experiment.[56] But apart from this, which we are not inclined to use as a strong argument, Arning's experiment, in order to be scientifically free from objection, needs to be performed with reference to the question of race and habitat. Hawaiians become lepers with great frequency, and the disease may be regarded as endemic at the present time in the Sandwich Islands. The possession of a criterion of the *living* Bacillus Lepræ is also of the first importance in experiments of this nature.

Attempts to transmit the disease to man were made more than thirty years ago by a Norwegian physician, who endeavoured to inoculate himself and other individuals with leprosy by the introduction under the skin, of leprous tubercles, blood, and pus from lepers. These efforts to transmit the disease uniformly failed.

Profeta[57] has similarly failed to inoculate himself or others with leprosy, and Cagnina[58] met with the same result. Numberless physicians and surgeons during the performance of autopsies, or operations on lepers, have accidentally wounded themselves, and there is no case recorded of the subsequent appearance of leprosy.

Experimental inoculations upon animals have never succeeded in establishing leprosy as it is clinically seen in man. A visceral leprosy, if such a term may be employed, has however been obtained by some experimenters, though a far greater number of observers have failed.

In Neisser's observations,[59] a freshly extirpated leprous tubercle was introduced into the peritoneal cavity, subcutaneous tissue, and anterior chamber of the eye of rabbits and dogs. The value of these experiments, in some

(56) Report on Leprosy in Molakai, by Dr. Swift, who attended Keanu's relations.
(57) Profeta "Sur l'Elephantiasis des Grecs"—Giornale intern. dell. sc. med., 1884, quoted by Leloir.
(58) Quoted by Leloir, p. 238.
(59) Virch. Archiv. Vol. 4, 1881, p. 534.

of which there was perhaps a local lepra, as well as some subsequent negative observations in which "cultures" and leprous tubercles were used, is thus summarized after a criticism of the work of others in this direction. "Leprosy in animals has not yet been established by certain convincing experiments." [60]

Köbner [61] and Hansen, [62] (both of whom inoculated monkeys) obtained negative results. Damsch [63] after implantation of leprous tubercle in the anterior chamber of the eye of rabbits, described an infiltration of the iris, ciliary body, and Descemet's membrane, with cells containing Leprosy Bacilli. In consequence of the enormous number of bacilli, he believed that a definite multiplication of these had occurred within the body. But it is the case that pieces of tubercle left in ordinary water for some weeks similarly shows the water to be full of Leprosy Bacilli, which no doubt are washed out of the mass, and it is quite conceivable that the results obtained by Damsch may be a simple mechanical process, especially since a distribution of the bacillus within the body did not occur. The implantation experiments beneath the skin and into the peritoneal cavity of cats do not appear to have led to more than the results obtained by Neisser, that is, a possible local lepra. Campana's [64] experiments consisted in numerous inoculations of leprous tubercle on the vascular comb and beard of fowls. A local tumour frequently formed at the seat of inoculation, and large cells produced in the inflammatory process took up the Leprosy Bacilli in the same manner as inorganic particulate foreign bodies would be ingested by white blood corpuscles. Campana believes that the results obtained by Damsch are capable of the same interpretation Vossius,[65] by inoculation in the anterior chamber of the eyes of rabbits, found an extensive accumulation of Leprosy Bacilli in the cornea, iris and ciliary body, but a distribution of these bacilli throughout the body, and the establishment of visceral or cutaneous lesions, is necessary before these can be considered other than implantation experiments.

Hillairet and Gaucher [66] inoculated a pig with leprous tubercle; E. Vidal

(60) Virch. Archiv. 1886, Vol. 103, p. 384.
(61) Virch. Archiv. 1882, Vol. 88, p. 282.
(62) Congress de Copenhague, 1884, quoted from Leloir.
(63) Virch. Archiv. 1883, Vol. 90, p. 20.
(64) Campana, Archiv. suela. Scienza Med. 1883, and Clinica Dermatopatica, 1883, quoted from Neisser.
(65) Bericht der XVI Vers. der Opthal. Gesell. Heidelberg, 1884, quoted from Neisser.
(66) Société de Biologie, 1881, quoted from Les Bacteries, p. 760, by Cornil and Babes.

repeated the experiment, and the implanted mass a year later showed numbers of Leprosy Bacilli, though none were to be found in the surrounding tissue; these observations may therefore be regarded as negative. Thin, Kaurin,[67] Arning, Rake,[68] Bordoni-Uffreduzzi (who failed to inoculate with his cultivated Bacillus Lepræ) have hitherto met with failure to establish leprosy in animals.

Hitherto the transmissibility of the disease to animals has succeeded only in the experiments of Melchor and Ortmann.[69] In rabbits they describe an intestinal and a lymphatic gland lepra as developed four months after the introduction of freshly extirpated leprous tubercle into the anterior chamber of the rabbit's eye. After inoculation a subacute general infective disease is established, and the autopsy showed almost all the viscera (cæcum, lymphatic glands, spleen, lungs) to be infiltrated with nodules varying in size from a pin's head to a pea. In these nodules were quantities of typical Leprosy Bacilli. Numbers of observers,[70] who have seen the specimens obtained from these disseminated nodules, are satisfied that a genuine visceral leprosy was established by the experiments of these observers, and the conclusion that under certain circumstances leprosy is an infective disease transmitted by the agency of the Bacillus Lepræ, may fairly be made from these experiments. At the same time it must be stated that, although a few animals became acutely infected, a far greater number escaped infection.

With the exception of the last-mentioned experiments, it may be considered that the proof of the inoculability of leprosy has not yet been given. Had Melchor and Ortmann worked with a pure culture of Bacillus Lepræ and again recultivated the bacillus found in the organs, the proof would have been complete, and Koch's postulates for the specific pathogenic nature of a bacillus would have been satisfied. As mentioned above, Bordoni-Uffreduzzi failed to inoculate with his cultivated Bacillus Lepræ.

Our experiments were commenced at Almora on April 3rd, 1891. Pieces

(67) "In spite of several experiments I have not been able to prove Dr. Ortmann's inoculation of the Bacillus Lepræ on rabbits." Journal of the Leprosy Investigation Committee, No. 2, 1891, p. 68.

(68) Reports of Trinidad Leper Asylum, 1884, 1885, 1886, 1887, 1888, 1889.

(69) Berl. Klin. Wochen. 1885, No. 13, and Ibidem, 1886, No. 9.

(70) Among others Arning (p. 117, Journal of the Leprosy Investigation Committee No. 2), who writes "there is no doubt to my mind that Dr. Ortmann of Königsberg has succeeded in inoculating animals with leprosy. I have seen his specimens and am fully convinced of the accuracy of his results. Campana, Wesener Munch. Med. Wochen. 1887, No. 18) and others have adversely criticised the above experiments.

of freshly extirpated tubercle from the wrist were removed with all antiseptic precautions, and a piece of the jejunum of a dog, about 4 inches in length, was resected, washed out with sterilized warm water, then closed at one end, the piece of tubercle introduced, and the other end closed. The mesentery was left attached. The gut was now joined by double rows of Lembert's suture and the abdomen closed. Two dogs were operated on in this way; the first died on the sixth day. Considerable peritonitis was found. The sutures were in place, and there was no obstruction of the bowel at this point. The resected loop contained a yellowish fæcal liquid. In this liquid the piece of tubercle lay unchanged. All the viscera appeared healthy. The fluid in the resected bowel and the mucous membrane were examined microscopically. Plenty of Leprosy Bacilli were found in half the specimens of the former; the latter were negative. The second dog died on the sixth day. Resected ends of the bowel had almost separated. In the resected loop, which was gangrenous, the piece of tubercle lay unchanged. Cover-glass specimens of the tubercle, the contents of the loop, and the submucous layer of the bowel wall were taken. In the first, were quantities of Bacillus Lepræ, in the second, three specimens contained Bacillus Lepræ, and one showed none, in the third, no bacilli were found. These two experiments were performed chiefly with a view to estimate the possibility of success in the operation. The piece of tubercle would have remained in the loop, and had distribution of the bacilli occurred, these would in all probability have been found either in the lymphatic glands or in the fluid of the thoracic duct. The admixture with digested food and mixed intestinal secretions, such as the bile, which might affect the activity of Bacillus Lepræ, would also have been obviated. The bacilli found in the contents of the resected loop were probably only mechanically washed out of the introduced tubercle.

Further experiments were carried on in the laboratory built by the Indian Government for the Commission at Simla. Cultures obtained from blister fluid and leprous tubercle were mixed with about twenty times their volume of ·65 per cent. saline solution, and the mixture so obtained was used for all the inoculations at Simla.

On June 5th, 1891, a minute quantity, about one-fourth of a minim, of a culture of the first generation on agar of the bacillus which was vacuolated (see Cultivation Experiments) was introduced with antiseptic precautions into a pocket in the left ear of a pariah dog, and at the same

time about 1 minim of the original culture of the bacillus (which morphologically appears like Bacillus Lepræ) in bouillon, into a pocket of the right ear. Both these cultures at the time of inoculation were examined and found to be pure. On June 9th a small subcutaneous lump was felt on the left ear, but none on the right. On June 15th both ears at the points of inoculation presented distinct local thickening, and about 1 minim of the second generation of the vacuolated bacillus on agar was mixed with twenty times its volume of ·65 saline solution, and 1 minim of this subcutaneously injected into the right ear about one inch from the thickened nodule. One minim of the culture (fourth generation on agar) was introduced into the left ear. On July 2nd this dog was injected at the same place in both ears with 1 minim of the original culture from tubercle in bouillon. This was fourteen days old, and the solution injected was 1 : 20 saline. This dog died on July 15th, with symptoms of salivation, wasting, and multiple subcutaneous abscesses on limbs and abdomen. On the left ear, but only at the site of the last inoculation, there was distinct thickening of the cartilage shown by section; on the left ear nothing was evident. The viscera were healthy, but the axillary glands were somewhat enlarged. The lungs, liver, spleen, mucous coat of stomach, testis, kidney, pus from the abscesses, and tissue from the local thickening of the ear were examined microscopically by means of cover-glass preparations. In no instance were any specimens of the bacilli described by us present.

On June 3rd another dog was chloroformed, the peritoneum was opened, and about one minim of the culture on gelatine from the culture on agar of the bacillus resembling Bacillus Lepræ, was introduced. The culture was examined and was found to contain plenty of these morphologically similar bacilli.

The dog was killed and examined on August 14th. There were adhesions of the peritoneum in the neighbourhood of the cæcum. There were no enlarged glands or growths. The liver, spleen, kidneys, stomach, intestines, heart, lungs, parotid and submaxillary glands were all normal, and microscopic examination showed no bacilli.

On July 8th, and subsequently at intervals of about a week, another dog was injected in the right axilla with about five minims of a solution of a culture on gelatine from the culture in bouillon of the bacillus obtained from a leprous tubercle by the second method described above, *viz.*, by introducing a piece of leprous tubercle, removed with all possible precautions, into

bouillon. This dog was killed and examined on August 15th. All the viscera were normal, but the mediastinal and mesenteric glands were enormously enlarged. Microscopic examination showed no bacilli in the viscera or glands.

On June 11th, and subsequently at intervals, a rabbit was inoculated beneath the skin of the abdomen with a solution of the fourth generation on agar from the culture obtained by the first method, *viz.*, cultivation in blister fluid. The rabbit was killed and examined on August 11th. The liver contained two white growths which showed psorospermiæ under the microscope. The other viscera were healthy, and examined microscopically no Leprosy Bacilli were found.

On June 11th, and subsequently at intervals, another rabbit was injected on the right side of the abdomen with the same solution as the last. It was believed that the liquid entered the peritoneal cavity. The rabbit was killed and examined on August 12th. Nothing was found at the point of inoculation. The suprarenal capsules were slightly enlarged. All the other viscera were healthy. No Leprosy Bacilli could be found in the suprarenal capsules or in the other viscera.

On July 2nd the anterior chamber of the left eye of another rabbit was injected with two minims of a solution of culture obtained by the second method. On July 8th the anterior chamber of the right eye of the same rabbit was inoculated in the same way. The iris was wounded during the inoculation. Both eyes healed perfectly and no growth was visible in the anterior chambers. The rabbit was killed and examined on August 13th. The liver showed a semi-purulent cyst, which was found to contain psorospermiæ. The other viscera were normal, and there were no enlarged glands. Four specimens of the aqueous humour of the right eye were examined microscopically. One bacillus comparable with the culture was found. This was probably a portion of the original injection. No growth of bacilli was found. The lens and cicatrix on the same side were examined with negative result.

On June 13th, and subsequently at intervals, another rabbit was inoculated in both ears and in the left axilla with one minim of a solution of the fourth generation on agar of the culture obtained by the first method. On June 24th a slight thickening was noticed at the site of inoculation, and a drop of blood and fluid taken from this place was stained and examined. It showed four or five deeply stained pink bacilli,

and in one part of the field a group of faintly stained bacilli, matted together, and strongly suggestive of the culture introduced. The rabbit was killed and examined on August 10th. There was some thickening at the points of inoculation on the ears, but no bacilli were to be found in them. The liver contained two semi-purulent cysts showing psorospermiæ. The other viscera were healthy. There were no enlarged glands. No Leprosy Bacilli were found in any part of the body.

On June 24th, a monkey (*Macacus rhesus*) was injected on the inner side of the right thigh with about four minims of solution of the fourth generation on agar of a culture obtained by the first method. On July 1st it was injected in the left groin with about two minims of a solution of the original culture from a leprous tubercle obtained by the second method. The monkey never showed any local thickening or evidence of infection, and when last seen, on August 24th, was, to all appearance, healthy.

From the above series of experiments it will be seen that in the two dogs operated on at Almora, Leprosy Bacilli were found in the resected portions of bowel, but that these were in all probability mechanically washed out of the introduced pieces of tubercle. In the Simla experiments a few bacilli were found in one ear of a rabbit at the point of inoculation, and a single bacillus was found in the anterior chamber of another rabbit, also close to the site of inoculation. In view, however, of the very small number of these bacilli, and of the absence of any general diffusion through the bodies of the rabbits, we can only regard the bacilli as portions of the cultures actually introduced. Indeed, these cultures did not appear to be pathogenic. There was nothing to point to a multiplication even at the point of inoculation. We therefore consider all our inoculation experiments absolutely without result.

Interesting negative evidence was also obtained at Ahmedabad, where we examined twenty-one fowls which had lived for periods of four or five years in the compound of the Leper Asylum, and had been in the habit of pecking the ulcerated feet of the lepers to such an extent as to necessitate their removal. They had been kept apart for our inspection. All these fowls, when we examined them on August 29th, were healthy and in good condition. Surgeon Quicke informed us that a fowl had recently died in the Leper Asylum compound which had a large tumour under one wing. Unfortunately the specimen was not kept as he had ordered, and the opportunity of examining it was lost. From

Dr. Quicke's description, however, the tumour appeared to be too large for a leprous nodule. From the numerous recorded experiments of observers in various parts of the world, and from our own attempts at inoculation, we consider it extremely doubtful whether a true leprosy, such as we recognise clinically, can be produced in animals. In the absence of this step, Koch's postulates remain unfulfilled, and it is impossible to say whether the cultures we obtained from leprous tissues and fluids are growths of Leprosy Bacilli or not. Further experiments which we are determined to prosecute with reference to cultivation and transmissibility to animals, will, we hope, tend to elucidate the ætiology of a disease which we believe to be due to a specific bacillus.

APPENDIX II.

Laboratory Work at Simla.

BY

A. A. KANTHACK.

Most of the work described in this short appendix was done conjointly with the late Surgeon-Major A. Barclay. As, however, his untimely and deplorable death occurred before these lines were even begun, the sole responsibility of all statements contained therein must rest with me. The results of this portion of the practical work will be stated as briefly as possible, as they convey nothing new, and are mostly negative, or old established facts.

I.—EXAMINATION OF TISSUES AND DISCHARGES.

The method of staining for Leprosy Bacilli in each instance was that generally known by the name of Koch-Ehrlich, or Ziehl's modification thereof.

(*a*) Tubercles from the ear of a tubercular leper, an inmate of the Tarn Taran Asylum.

Typical masses of bacilli were found in the corium of the skin, the epithelium in all specimens being free. The bacilli were arranged in irregular clusters, but at times in small chains of two or three rods. Most of them are pointed, a few however having blunted ends. The other physical characters are so well known as not to deserve special mention. It may, however, be noticed that in some specimens isolated fuchsin bodies, similar to those found in Rhinoscleroma and the fungoid disease (Mycetoma) were found.

(*b*) Discharge from sores of anæsthetic lepers, inmates of the Subathu Asylum.

Five patients of a purely anæsthetic type were chosen and ten cover-glasses prepared from each. Of fifty cover-glasses ten only showed positive results, and these were all taken from one patient. Here Leprosy Bacilli, in masses or scattered about separately, could be found in almost every field.

(*c*) Discharge from sores of mixed lepers belonging to the same Asylum.

Two patients were selected and again ten cover-glasses prepared from either. The result was positive in one instance.

(*d*) Nerve from a purely anæsthetic case.

Surgeon-Major E. Lawrie, while performing the operation of nerve-stretching, removed small pieces of the greatly enlarged ulnar nerve.

Cover-glass preparations gave no results. Sections of the nerve, hardened in alcohol and stained in the usual manner, revealed a few bacilli between the fibrillæ, and also numerous giant cells similar to those observed in a tuberculosis, but smaller. Some of the giant cells contained from one to three Leprosy Bacilli.

(*e*) Nerve from a purely anæsthetic case.

This specimen was also obtained through the kindness of Surgeon-

Major E. Lawrie. The result of the microscopic examination was negative, although the piece was excised from a highly diseased part of the nerve. Round cells resembling leucocytes were found in large numbers; giant cells were absent.

The nerve in each case, as exposed by Dr. Lawrie, was much thickened, transparent and greyish-yellow, and hard to the touch. The microscope revealed besides the above-mentioned cells an atrophy of most of the primitive fibrillæ and also a well marked fatty metamorphosis of the round cells, especially visible in unstained specimens.

(*f*) A discoloured anæsthetic patch.

Surgeon-Major Lewtas kindly removed a small piece of skin from an anæsthetic discoloured patch of a leper. No bacilli were found in any of the forty-three sections prepared.

(*g*) Itch pustules of—

(1) two anæsthetic cases and

(2) two mixed cases from the Madras Asylum.

The pustules were situated on apparently healthy portions of the skin. Six cover-glasses were prepared from each case, but the result was negative, no leprosy bacilli being found.

(*h*) Bulla on the dorsum of the hand of an anæsthetic case (Tanjore). Ten cover-glasses were prepared and stained, but all with negative result.

The patient was afflicted with pure anæsthetic leprosy, and had paræsthesia of both hands. The ulnar nerves were both thickened. The bulla was spontaneous in origin, and not due to any injury or burn. The patient spoke with confidence on this point, and considering that there was only a slight loss of sensation, there is no reason to doubt him, and the bulla must be regarded as a sequela of the nervous lesion.

II.—EXAMINATION OF BLOOD AND SERUM.

(*a*) Blood and lymph obtained by pricking a tubercle.

Two patients from the Subathu Asylum were employed, and after pricking the tubercle pressure was applied, so that with the blood the juice of the tissues was squeezed out. Twelve cover-glasses were prepared, and all the specimens showed the characteristic bacilli, mostly in cells and masses.

It was impossible to obtain pure blood from a vessel.

(*b*) Blood obtained by pricking an apparently healthy but anæsthetic part of a mixed case.

Two patients from the same Asylum were again chosen, and after pricking the skin pressure was applied to the part. Twenty cover-glasses were prepared, and in two Leprosy Bacilli enclosed in cells were obtained. As, however, these men presented extensive cutaneous lesions, it is difficult to say whether the bacilli had not been shed from other parts of the body, and thus reached the cover-glass as impurities, so to speak, especially when it is remembered that only two out of twenty specimens showed bacilli.

(*c*) Blood obtained by pricking the dorsum of the foot of purely anæsthetic cases.

Six patients, three from Subathu and three from Hyderabad, were employed, and though sixty specimens were prepared and examined in no case was a positive result obtained.

(*d*) Lymph or juice pressed out of tubercle after previous ligature.

Every specimen (four in number) was swarming with Leprosy Bacilli.

(*e*) Blisters raised over tubercles.

Four patients were employed and forty specimens of the exuded fluid examined. The clear serum was free from bacilli in all instances. In one case the blister subsequently became sero-purulent, and of six specimens made of this fluid one contained typical bacilli enclosed in cells. The pustule was punctured, and the collapsed pellicle removed, and next day the soft crust examined. Of six specimens four showed numerous clusters of bacilli. It seems, therefore, an uncommon occurrence to find bacilli in absolutely clear serous blister fluid. This does not seem surprising, considering that the greater part of this fluid comes from the vessels, and excepting epidermal *débris* only contains leucocytes. Blistering is, however, different from vaccination, where the irritant is inoculated more deeply into the tissues, and it may be possible that in this manner cells in the corium containing Leprosy Bacilli are set free with the exuded fluid.

(*f*) Blisters raised over apparently healthy parts of skin of tubercular lepers.

The same patients were employed, and the same number of specimens prepared, however with uniformly negative result.

(*g*) Blisters raised over apparently healthy skin of anæsthetic lepers.

Three patients were blistered, and thirty cover-glasses stained, but bacilli were not found in a single instance.

III.—EXAMINATION OF SECRETIONS.

(*a*) Saliva—

The saliva of three tubercular cases with extensive facial lesions was examined at the Subathu Asylum.

Thirty specimens were prepared, and Leprosy Bacilli, mostly in clusters and embedded in cells, were found in every specimen.

(*b*) Nasal secretion—

Two patients suffering from extensive facial tubercular leprosy were selected. Except that minute tubercles were visible on the alæ nasi, the nose was quite healthy. Yet all the twenty specimens stained and examined, contained Leprosy Bacilli in fair number.

(*c*) Vaginal secretion of a tubercular leper—

Fifty specimens were examined, but no bacilli found.

(*d*) Menstrual discharge of an anæsthetic leper.

Thirty specimens were examined, but no bacilli found.

The chief results will now be given in a tabular form:—

TABLE I.

Source of material.	Number of patients examined.	Number of specimens prepared.	RESULT.
(1) Nerve of anæsthetic leper	2	...	*Positive in one case.*
(2) Anæsthetic discoloured patch	1	43 sections	Negative in all specimens.
(3) Discharge from sore of anæsthetic leper	5	50	*Positive in 10.* Negative in 40.

Table I—*continued.*

Source of material.	Number of patients examined.	Number of specimens prepared.	RESULT.
(4) Discharge from sore of mixed leper	2	20	*Positive in 1.* Negative in 19.
(5) Itch pustules of anæsthetic leper	2	12	Negative in all cases.
(6) Itch pustule of mixed leper	2	12	Ditto.
(7) Bulla over dorsum of hand of an anæsthetic leper	1	10	Ditto.
(8) Blood and lymph from a tubercle	2	12	*Positive in all cases.*
(9) Blood and lymph from healthy but anæsthetic part of skin from a mixed leper	2	20	*Positive in 2.* Negative in 18.
(10) Blood obtained by pricking dorsum of foot of a purely anæsthetic leper	6	60	Negative in all cases.
(11) Juice obtained from tissues of a tubercle	1	4	*Positive in all cases.*
(12) Blister over a tubercle	4	40	Negative in all cases.
(13) Pustule over a tubercle	1	6	*Positive in 1.* Negative in 5.
(14) Crust from (13)	1	6	*Positive in 4.* Negative in 2.
(15) Blister over healthy skin of tubercular leper	4	40	Negative in all cases.
(16) Blister over healthy skin of anæsthetic case	3	30	Ditto.
(17) Saliva of tubercular leper	3	30	*Positive in all cases.*
(18) Nasal secretion of tubercular leper	2	20	*Positive in 14.* Negative in 6.
(19) Vaginal discharge of tubercular leper	1	50	Negative in all cases.
(20) Menstrual discharge of anæsthetic leper	1	30	Ditto.

IV.—EXAMINATION OF WATER FROM TANKS FREQUENTED BY LEPERS.

(*a*) Water from a tank at Bombay, much frequented by lepers.

Only ten specimens were prepared and these were all negative.

(*b*) Water from a tank at Tarn Taran, much frequented by lepers.

Hundred specimens were prepared: fifty were stained according to Ziehl's modification of Koch-Ehrlich's method and another fifty according to Baumgarten's method. In no specimen could any Leprosy Bacilli be found.

V.—DUST AND EARTH FROM LEPER HUTS OF THE TARN TARAN ASYLUM.

(*a*) From hut inhabited by a tubercular leper with ulceration on the right foot.

(*b*) From hut inhabited by a tubercular leper with ulcers on both feet.

(*c*) From hut inhabited by a tubercular leper with ulcers on both feet.

In each case one hundred specimens were prepared: fifty were stained according to Koch-Ehrlich's method, fifty according to Baumgarten's method. In all three hundred specimens the result was negative.

(*d*) From hut inhabited by an anæsthetic leper with ulcerations on all extremities.

(*e*) From hut inhabited by an anæsthetic leper with ulcerations on both feet.

(*f*) From hut inhabited by an anæsthetic leper with ulcerations on both feet.

In each case fifty specimens were prepared: twenty-five were stained according to Ziehl's method and twenty-five according to Baumgarten's method.

In all hundred and fifty specimens the result was negative.

These results will be given in tabular form:—

TABLE II.

Source of material.	Number of specimens examined.	Method.	Result.
(1) Water from tank at Bombay . . .	10	Ziehl's . . .	Negative.
(2) Water from tank at Tarn Taran . .	100	50 by Ziehl's . . / 50 by Baumgarten's	Ditto.
(3) Dust from hut of tubercular leper with ulcers on one foot	100	50 by Koch-Ehrlich's / 50 by Baumgarten's	Ditto.
(4) Dust from hut of tubercular leper with ulcers on both feet	100	Ditto . . .	Ditto.
(5) Dust from hut of tubercular leper with ulcers on both feet	100	Ditto . . .	Ditto.

Table II—*continued.*

Source of material.	Number of specimens examined.	Method.	Result.
(6) Dust from hut of anæsthetic leper with ulcers on all extremities	50	25 by Ziehl's . . / 25 by Baumgarten's	Negative.
(7) Dust from hut of anæsthetic leper with ulcers on both feet	50	Ditto . . .	Ditto.
(8) Dust from hut of anaesthetic leper with ulcers on both feet	50	Ditto . . .	Ditto.

VI.—IMPLANTATION OF LEPER TISSUE INTO ANIMALS.

On January 22nd five animals—one monkey, two cats, and two rabbits—were operated on in Dr. D. D. Cunningham's laboratory at Calcutta. The tissue was removed from the ear of a tubercular leper, and minute pieces removed from the centre of the excised mass, used for implantation. These particles were inserted either into the anterior chamber of the eye, or dropped into the peritoneal cavity. The operation in each instance was performed with sterilized instruments and all antiseptic lotions carefully avoided, sterilized water alone being used. The eye operations were performed on the monkey (one eye), one cat (both eyes), one rabbit (one eye), and the abdominal implantation practised on the other cat and rabbit. Unfortunately both the rabbits died before they came into my possession. The animals were left at the Zoological Gardens of Calcutta, and after the rains had set in, sent up to Simla, where they arrived by the beginning of August in rather emaciated condition, due, no doubt, to the fatiguing journey.

I propose now to state briefly the result of these implantations

(*a*) Cat, operated on January 22nd, 1891—

A particle from a leprous tubercle of the size of a hemp-seed was deposited into the peritoneal cavity.

August 11th, 1891.—Excepting the emaciated appearance of the animal nothing abnormal could be detected. On killing the cat

and making an autopsy, the original nodule was easily found in the omentum. It appeared larger, but this was evidently due to a new formation of fibrous tissue around it, and a delicate thread of fibrous tissue was traced from the nodule to the parietal peritoneum.

No dissemination whatever had occurred, the peritoneum, intestines, and other viscera being quite free.

On microscopic examination bacilli were found in the nodule, not different in shape and arrangement from those in any tissue removed from a tubercular leper. *The result is therefore absolutely negative.*

(*b*) Cat, operated on January 22nd, 1891—

An iridectomy was performed on both eyes and a particle from a leprous tubercle of the size of a hemp-seed inserted into either anterior chamber. No reaction whatever occurred, no iritis or conjunctivitis, and the corneal wound healed well.

August 11th, 1891.—The animal was somewhat emaciated, but otherwise perfectly healthy, and excepting in the eyes no changes could be detected anywhere. The media of both eyes were clear to naked-eye examination and to inspection by focal illumination and the ophthalmoscope. No iritis could be observed in either eye. The nodule was plainly seen in the aqueous humour and was apparently undiminished in size.

(1) *Left eye.*—The nodule was visible on the temporal side, being fixed by a small thread of fibrous tissue to the scar in the cornea on the one hand, and to the adjoining part of the iris on the other, so that it could perform only very limited movement. The corneal scar was partly vascularised, but otherwise the cornea was perfectly clear. The iris was normal to all appearance and only slightly swollen at the insertion of the fibrous thread which connected it with the nodule. The aqueous humour was clear.

A dissection confirmed the observations of this examination.

Microscopic Examination.

Many cover-glass preparations were made of the aqueous humour, and all contained Leprosy Bacilli in fair number. Two or three masses, evidently shed from the nodule, were found in each specimen, but loose and independent bacilli could not be detected. It is thus highly improbable that the bacteria had grown or multiplied in the aqueous humour.

The eye was carefully washed with sterilized water, and cover-glass preparations made of the iris. Near the site of the implanted nodule, where the thickening in the iris existed, many Leprosy Bacilli were found, but the same, though much more scantily, were observed in all parts of the iris examined. The bacilli occurred mostly in cells, but a number of loose ones were also seen amongst the tissue elements of the iris. Sections of the iris were not made through want of apparatus, but little pieces were squeezed between cover-glasses and then treated in the ordinary manner.

In view of the absence of all morbid appearances in the iris and the comparative scarcity of the bacilli in more remote parts of the iris, I am inclined to think that the organisms have either been deposited on, or taken up into, the substance of the iris from the aqueous humour into which they had been cast off from the original nodule through maceration. The original nodule presented no differences microscopically from tissue freshly removed from a tubercular leper, and was full of bacilli.

The retina and vitreous humour were quite free from leprosy, or other, bacilli.

(2) *Right eye.*—The nodule was floating free in the aqueous humour. The cornea was clear and the linear incision made at the time of operation had healed, almost without leaving a scar. The iris and other parts of the eye-ball, both on ophthalmoscopic examination and oblique focal illumination, appeared unaffected. Microscopic examination was made of the original nodule and the aqueous humour only, the result being that the nodule was apparently unchanged, but the aqueous humour was quite free from bacilli. What remained of the two eyes has been reserved for examination by serial sections at a future time.*

* June 4,892: Such examination revealed an entire absence of bacilli withir the tissues of the iris choroid, etc.

(3) A careful autopsy and search did not reveal any dissemination or secondary growths in any part of the body.

Therefore the result of these two animal experiments must be considered on the whole as highly unsatisfactory, as there is an absence of infection or, so far as can be judged by cover-glass preparations, of even a local proliferation. The want of inflammatory, or other general and defined changes in the iris, the clearness of the aqueous humour and of the cornea are all against a local contagion, to borrow a term from Virchow. The result of the experiment on the next animal was equally unsatisfactory.

(*c*) Monkey, full grown, operated on January 22nd, 1891—

An iridectomy was performed on the left eye and a piece of tissue from the same leper who supplied material for the other animals, was inserted into the anterior chamber. As it was impossible to prevent the animal from scratching, or interfering with its eye, some reaction, amounting to slight suppuration and hypopyon, took place, and the cornea also became slightly inflamed.

August 22nd, 1891.—The monkey was somewhat emaciated, but otherwise no morbid changes were visible on the surface. After death a close examination of the eye revealed intense inflammation of the conjunctiva amounting to chemosis, inflammatory changes of the cornea, and suppuration in the anterior chamber. The wound in the cornea had not healed and matter from the anterior chamber was bulging out through the same. Subsequent dissection showed that the iris was almost completely destroyed, at least irrecognisable in the mass of thick pus which had taken the place of the aqueous humour. The anterior surface of the lens was white and softened. The rest of the media were perfectly normal to the naked eye.

Microscopic Examination.

(1) In the conjunctiva no bacilli were found by means of cover-glass specimens, except at the unhealed wound, whither most probably they had been discharged from the anterior chamber.

(2) Matter from the anterior chamber contained bacilli, but only in small quantity, two out of six cover-glasses preparations showing the typical masses.

(3) The other media of the eye showed no bacilli.

All the other organs of the animal, excepting a few enlarged mesenteric glands, were apparently normal. The glands will be left for future examination, but it is highly improbable that they contain any bacilli, as the whole peritoneum appeared to be healthy. No reference has been made to the experiments of Damsch and others, because in the absence of sections it is quite impossible to speak with any confidence on such matters as local proliferation and dissemination. This portion of the work has been reserved for another time.*

VII.—CULTIVATION EXPERIMENTS WITH THE BACILLUS LEPRÆ.

The results of these experiments—premature and short-lived—have been published in the British Medical Journal (June 6th and 20th, 1891). As after a careful review of all the facts, benefiting by the kind criticism of Professors C. Fränkel and Baumgarten, I feel convinced that the artificially grown bacillus was not the Leprosy Bacillus; it will suffice simply to mention that attempts were made by me, in conjunction with the late Surgeon-Major A. Barclay, to cultivate the bacillus, but with no result. An explanation of this matter has already appeared in the above-mentioned journal.

ADDENDUM.

VIII.—EXAMINATION OF MOSQUITOES FOR LEPROSY BACILLI.

The following observations were made in conjunction with Surgeon-Major S. J. Thomson. The insects were obtained through the kindness

* *Cf.* preceding footnote.

of Surgeon-Major B. O'Brien, Civil Surgeon and Principal of the Medical College, Agra.

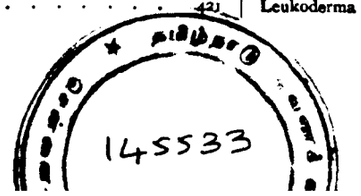
Three sets of mosquitoes were enclosed in muslin bags which were tied round the legs of three lepers. Of these one suffered from ulcerative tubercular leprosy, another from the tubercular form, and the third from anæsthetic leprosy. The bags were applied for a couple of hours in the morning, and again in the evening. At the end of four or five days many of the enclosed insects had died. These, with the few surviving ones, were put into separate bottles filled with absolute alcohol and sent up to the laboratory of the Commission at Simla for microscopic examination.

Ten mosquitoes were selected from each set. Some were gorged, especially those from the two tubercular cases, but most of the insects were of a natural size and undistended. They were placed in water to remove the alcohol, and then each set separately ground up in a small mortar with the least possible quantity of ·75 per cent. salt solution, and ten cover-glass preparations made, using up the whole material. In this manner thirty specimens were obtained and stained for sixteen hours in anilin-fuchsin and treated in the usual manner. On microscopic examination no Leprosy Bacilli were found in any of the cover-glasses; a few cocci in some of the specimens were the only organisms seen.

Mosquitoes and Leprosy.

NATURE OF DISEASE.	Number of mosquitoes examined.	Number of specimens prepared.	RESULT.
(1) Tubercular leper without ulcers . . .	10	10	Negative.
(2) „ „ with ulcers	10	10	Ditto.
(3) Anæsthetic leper	10	10	Ditto.

INDEX, AND NAMES OF AUTHORITIES QUOTED.



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