

was subnormal. The cardiac lesions were exceptionally severe, the diseased aorta and the aortic valves were covered with fungating excrescences and the vegetations also extended over the endocardium; the anterior cusp of the mitral valve was perforated.

In very few patients were there rigors. Sir T. Lauder Brunton¹³ has pointed out that the access of fever in infective endocarditis is often fugitive, so that the temperature should be taken several times daily in any suspicious case. The temperature charts of many of the patients to whom I have alluded would show how important it is to exercise such a precaution and how easily the existence of a little pyrexia may be overlooked. The temperature chart of a patient who died from endocarditis under the care of Dr. J. Hill Abram emphasises the importance of the frequent use of the thermometer; for seven days the temperature was taken night and morning and there was evidence of slight pyrexia only. Subsequently the thermometer was used every four hours and then it was seen that the fever was most marked in the intermediate intervals. In some patients pyrexia was absent for days and weeks at a time; in such the transient periods of fever are readily overlooked. One woman was in the hospital three months. For days together her temperature was subnormal; after death the aortic cusps were found to be covered with vegetations, the spleen enlarged, and bacterial infarcts, causing subcapsular hæmorrhages, were found in the kidneys. Another woman was in the hospital a month. During the first fortnight her temperature was never above 100° and for the last fortnight was normal or subnormal. She was markedly anæmic and much wasted. The mitral cusps were found to be covered with vegetations and many chordæ tendinæ were ruptured and the spleen was enlarged. Another patient, a man, was in the hospital seven weeks and for the last month of his life his temperature was normal or subnormal and his heart presented the typical features of an infective endocarditis. In five patients the pyrexia was of mixed type, long periods of normal or subnormal temperatures were interrupted by short periods of fever. In six the temperature was generally little above normal or became normal or subnormal as the fatal termination was approached. In all these there was evidence of very positive renal trouble; in nearly all hæmaturia; in one hæmatinuria. It might seem likely that the disease of the kidneys afforded an explanation of the anomalous temperature in these cases were it not that the same apyretic forms occurred in subjects who presented no symptoms of nephritis. In the temperature charts of five other patients pyrexia was often absent and they were free from disease of the kidneys.

¹³ Edinburgh Medical Journal, May, 1897.

THE CHILDREN'S FRESH AIR MISSION.—The annual meeting of the Children's Fresh Air Mission will be held in Staple Inn Hall, Holborn, London, E.C., on Tuesday, April 28th, when Alderman and Sheriff Sir George Wyatt Truscott will preside. This mission sent 2430 poor and ailing children into the country last year to benefit by change of air.

SOCIETY FOR RELIEF OF WIDOWS AND ORPHANS OF MEDICAL MEN.—A quarterly court of the directors of this society was held on April 8th, Mr. Christopher Heath, the President, being in the chair. Two new members were elected; the death of a member and the resignation of two members were reported. The death of a widow, aged 87 years, who had been in receipt of grants of the annual value of £62 since April, 1887, was announced. There were no fresh applications for grants. It was resolved to distribute at the next court £1251 among the 55 widows, 13 orphans, and the four recipients from the Copeland fund who had applied for the renewal of their grants. The expenses of the quarter were £56 19s. The following gentlemen were nominated for election at the annual general meeting to fill the vacancies among the officers of the society—as vice-presidents, Mr. Couper, Dr. W. Rigden, and Mr. T. Laurence Read; as directors, Dr. Brodie, Mr. Richards, Dr. Adams, Mr. Mahoney, Mr. H. Rogers, Mr. Smale, Dr. F. H. Champneys, Dr. Younger, and Dr. Chambers. The annual general meeting of the society was fixed to be held on Wednesday, May 20th, at 5 P.M., at 11, Chandos-street, W.

AN ANALYSIS OF 220 CASES OF SUDANESE LEPROSY.

By T. J. TONKIN, L.R.C.P. & S. EDIN., &C.,
LATE MEDICAL OFFICER, HAUSA ASSOCIATION'S CENTRAL SUDAN
EXPEDITION, 1893-94-95.

As the following paper is written for those who are more or less directly interested in leprosy I have not entered into any long explanation of my results, nor have I discussed them any further than has seemed to be quite necessary for easy comprehension. For the sake of brevity and clearness I have in the main limited myself to recording my observations, arranging and tabulating them for convenience of reference. The scope of the analysis is indicated by the heading of the paper. During the time I was engaged, among other things, on the investigation of leprosy in the Central Sudan a very large number of cases of the disease passed in one way or another across my line of observation. Many of them were out of convenient range, but 220 presented themselves in circumstances which allowed me to go very fully into most of the important points relating to their disease and it is this collection that I am now bringing forward. I was able to deal with the subjects of these cases at first hand and in all but a very few instances in their own language and without the mediation of an interpreter. I need hardly point out the advantage of this. I was also in a position to examine a large number of them on several successive occasions and this has necessarily enabled me to be surer of my results than I could have otherwise been. It has also enabled me to give in some directions fuller details. I do not, however, attach extreme importance to everything I have set down, but it is always possible that others may be interested in points that seem of small moment to me, and for that reason I am reporting some things that I otherwise should have left out. To facilitate comparison I have followed the scheme of arrangement used by most previous observers.

The Sudanese leper field.—I have already published an account of the situation and size of the Sudanese leper field,¹ and geographical details at length are in any case out of place in such a paper as this, but the term Sudan, as popularly understood, is so misleading that the accompanying sketch-map is not altogether as unnecessary as might appear. A glance at it will prevent the reader from falling into the mistake of regarding the Sudan as exclusively an appanage of Egypt and will give him in the readiest manner some idea of the situation, and roughly the extent, of the field from which the instances of the disease dealt with in the following paragraphs are drawn.

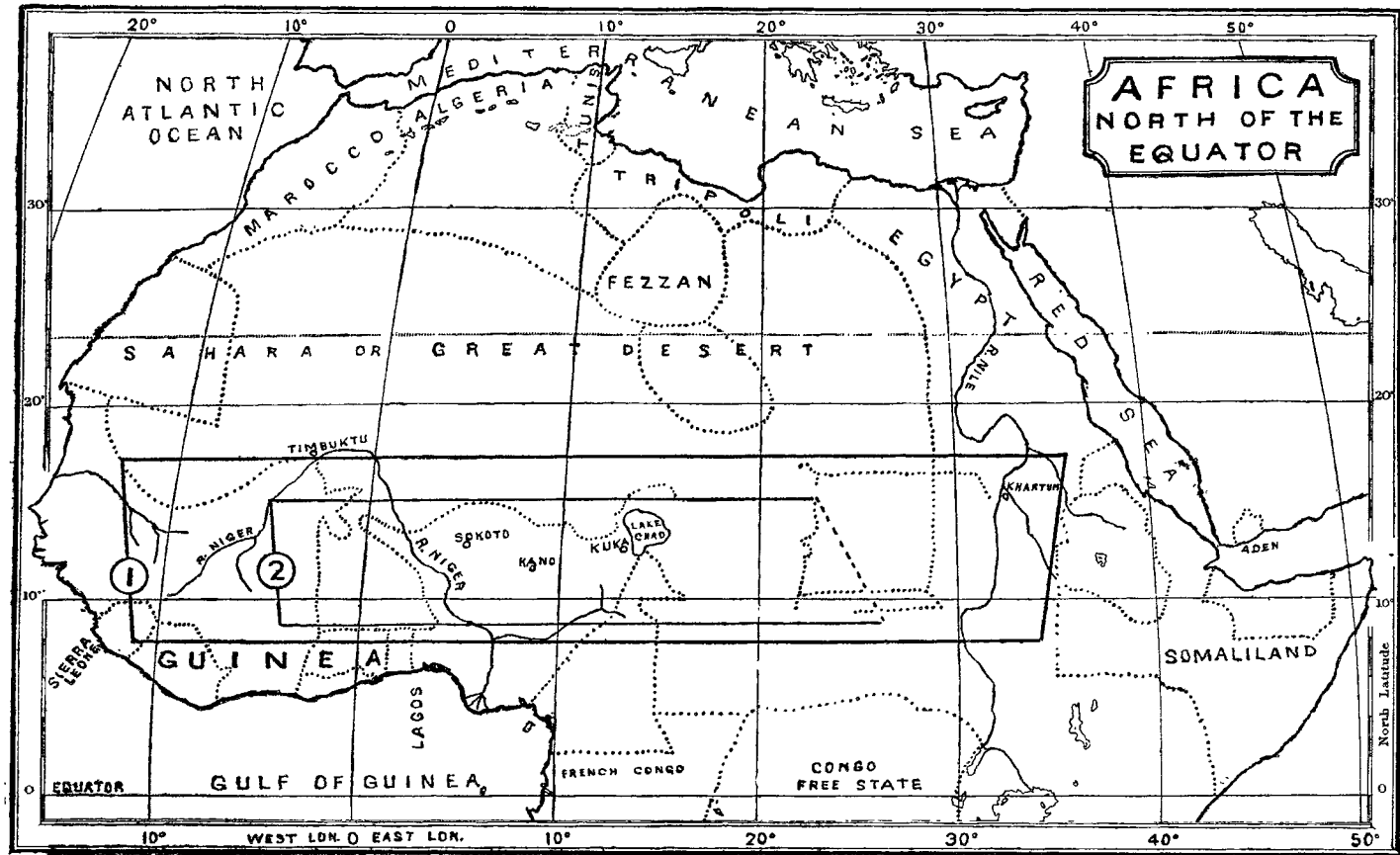
The signs of the disease.—In dealing with this part of the subject I have thought it better to report the various morbid conditions as I found them than to tie myself to any particular classification of the forms of the disease. I have simply catalogued the recorded appearances of each case and summarised the results in Table I.

TABLE I.—Cases classified according to the Signs of the Disease presented.

—	Numbers.	Percentages.
Patches only	96	43·6
Tubercles only	4	1·8
Patches and tubercles	19	8·6
Patches and mutilations	95	43·1
Tubercles and mutilations	0	—
Patches, tubercles, and mutilations... ..	2	0·9
Unclassified	4	1·8
Total	220	99·8

It will be seen that this table gives a percentage of 86·7 cases presenting patches only, against 11·3 which were characterised by tubercles. These figures would seem to suggest that the macular variety of the disease is greatly

¹ Transactions of the International Leprosy Conference, Berlin, 1897.



Outline map of Africa north of the Equator, to illustrate the leper field in the Sudan. The space marked 1 roughly defines the area of the Sudan. The space marked 2 gives some idea of the probable extent of the leper field. Kano is the centre of the districts in which leprosy is regarded as being most prevalent. Sokoto is the political capital of the Hausa States. Kuka is the capital of the kingdom of Bornu.

in excess in the Central Sudan. This is no doubt largely the case. The extreme preponderance of the macular variety which is shown is, however, subject to some modification. In the first place the expectation of life is about twice as long, if not more, in a macular as it is in a tuberculated case. The figures merely suggest the probable proportions of the two phases of the disorder among such lepers as may be alive at any given time: they do not in any sense indicate the relative number of individuals attacked, say, annually, by each. Further, the characteristic lepromata which stamp a case as tuberculated may disappear, and though by reason of the greater severity of this form of the disease such retrocession is rare, it still occurs with sufficient frequency to lead to the inclusion among the macular class of a number of cases which have not always belonged to it. In drawing up my tables no attention has been paid to statements about pre-existing conditions; the appearances set down are in every case those that were visible at the time of examination.

Relative liability of the sexes.—Of the relative liability of the sexes to leprosy, data taken in the part of the Sudan that this paper is concerned with supply definite information. Although the particular region referred to has been, and still is being, strongly influenced by Mahomedanism, some of the customs which are usually associated with that religious system have not as yet made such headway as to affect the liberty of the women, and I was in consequence able to deal with both sexes with equal freedom.

TABLE II.—Cases arranged according to Sex.

Sexes.	Numbers.	Percentages.
Males	125	56·81
Females	95	43·18
Total	220	99·99

As far as I am aware there is in the Sudan no reason why one sex should be more liable to the disease than the other.

The onset of the disease.—With regard to the period of life

at which the first definite signs of leprosy most commonly make their appearance I find my results are at variance with those of some well-known observers. The report of the Indian Commission shows that of the persons examined by it the majority became lepers between the ages of 26 and 30 years and that from 21 to 25 years was the next most favoured period. The possibility that in the Sudan they more often appear at an earlier date, while the constitution is yet unformed and the vital energies are still taxed by the strain of growth, is shown by Table III.

TABLE III.—The Age of Onset arranged in Periods of Five Years.

Years inclusive.	Number of cases.	Percentages.
1-5	11	5·00
6-10	45	20·45
11-15	41	18·63
16-20	44	20·00
21-25	31	14·09
26-30	17	7·72
31-35	9	4·09
36-40	10	4·54
41-45	4	1·81
46-50	3	1·36
51-55	1	0·45
56-60	2	0·90
Unclassified.	2	0·90
Total	220	99·94

This table gives from the sixth year onward to the twenty-fifth year as the period most frequently marked by the inception of the disease, leprosy most commonly attacking the individual during the continuance of the processes of growth and development.

Table IV. is in continuation of the same subject. It gives, divided into sexes, the cases in which the disease was developed between the first and the tenth years-inclusive.

TABLE IV.—Cases in which the Disease appeared between the First and Tenth Years arranged according to Sex.

Males.		
Years inclusive.	Numbers.	Percentages.
1-5	4	1·81
6-10	28	12·72
Females.		
1-5	7	3·18
6-10	17	7·72
Totals... ..	56 (ex 220)	25·43

That the foregoing figures, which are double those given by the results of the Indian Commission and four times more than those of Dr. Vandyke Carter, represent a general rule in the Sudan is, of course, more than I can say. All I can be reasonably sure of is that the earliest signs of leprosy appeared before the tenth year in over 20 per cent. of the cases which I examined.

Situation of the lepromata.—As I have dealt with this question more fully elsewhere² I need only mention here what I found to be the usual sites of the primary characteristic skin lesions. The face, particularly the prominences of the cheekbones and temporal ridges, the outer surfaces of the extremities, the scapular region, and the buttocks seemed to be the localities most frequently affected by the early leprosy infiltrations and possibly in the order named. I have never seen nodules on the palms of the hands or on the soles of the feet. The outer borders of the feet, however, I have often seen the seat of very dense leprosy infiltrations.

The duration of the disease.—Table V. gives in quinquennial periods the number of years which, at the date of examination, had in each of my cases elapsed since the onset of the disease. It is to my mind, and for reasons some of which I will state presently, one of the most interesting in the whole series.

TABLE V.—Years elapsed since the Onset of the Disease, arranged in Periods of Five Years.

Years inclusive.	Numbers.	Years inclusive.	Numbers.
1 to 5	100	36 to 40... ..	2
6 to 10	48	41 to 45... ..	1
11 to 15	34	46 to 50... ..	1
16 to 20	18	51 to 55... ..	1
21 to 25	8	Unclassified... ..	2
26 to 30	5		
31 to 35	0		
		Total	220

What strikes me as particularly interesting about this table is the relation which it bears to what appears to be still debated as the possibility of recovery from leprosy. That many instances of recovery from most grades of leprosy do actually occur is to me a thing beyond debate. It would be out of place to discuss the question at length here, though it is one on which in all probability many important issues will be found to hang; but in view of the idea, very widely spread even in professional circles, that the disease is a hopelessly incurable one, it is probably desirable that those who have good reasons for holding opposite opinions should take every opportunity of stating them, even though the scope of some of the opportunities, as in the present instance, precludes the possibility of putting the statement in the form of an argument. Of course, we know that from time immemorial leprosy has been regarded, not without reason, as an incurable disease. But there are two ways of reading incurable. Incurable it certainly is in the sense that at present we are not in the possession of any remedy that affects its course as dramatically as, say, mercury and the iodide of potassium affect that of syphilis.

But, personally, I cannot look upon it as incurable in any other sense. Leprosy is a disease that runs a fairly well-defined course. It is rare to hear of the actual morbid processes covering a period of more than from 15 to 20 years. I do not use these figures because they represent any rule, but because they are outside figures and are therefore safe ones. It is usually quite safe to assume that if an individual has survived the onset of his disease by anything approaching 20 years he will also have survived the disease itself. In such a case as this it will generally be found that all specific leprosy manifestations have disappeared. Their effects may remain—the fingers and toes that may have been lost will not grow again—but it is, to my mind, as illogical to regard a man on that account as suffering from leprosy when he may have enjoyed previously to the date of his examination anything from five to 15 years of unbroken health and when his capacity for labour is only limited by the actual destruction of tissue resulting from the pre-existent disease, as it would be to suggest that a person was suffering from small-pox because 10 years after he had had the disorder he happened to be still badly marked. I have seen many cases of alleged leprosy in which the individuals in question have suffered from what I should regard as a severe grade of the disorder but have nevertheless outlived all special signs of it, and entered into the enjoyment of a post-leprosy period of what appeared to me at any rate to be thoroughly good health; and I think the circumstances in which such cases as these occur have probably in the past received too little attention.

Hereditary transmission of the disease.—The next two tables (Nos. VI. and VII.) bear on the somewhat discredited subject of the hereditary transmission of the disease. They have, however, when taken with others, a sociological interest quite apart from their bearing on the mere abstract question of the possibility of hereditary transmission.

TABLE VI.—Dealing with the Condition of the Parents of the Lepers examined.

—	Numbers.	Percentages.
Number of cases in which both parents were leprosy at the date of the subject's birth	2	0·909
Number of cases in which one parent was leprosy at the date of the subject's birth	16	7·27
Number of cases in which it was doubtful whether the onset of leprosy in a parent or parents ante-dated or post-dated the subject's birth	6	2·72
Number of cases in which it was certain that the subject was born of healthy parents	196	89·09
Total	220	—

Now, if we adopt the reasonable assumption that a parent or parents cannot, by any process that may be called hereditary, pass on a disease to their offspring unless they are affected by that disease at the time of the begetting of the offspring the foregoing table makes it clear that out of my cases in only the exceedingly small minority of a fraction over 10 per cent. (10·89 per cent.) was it likely that direct transmission might be answerable for the appearance of the disease, while in 89·09 per cent. it would seem absolutely impossible that any such process could have had a hand. Again, if parents can pass on the disease to their offspring there appears no reason why they should not do so in a fair proportion of possible instances; and, if they did, it should certainly be found that a considerable percentage of the children of lepers manifest the disease in early life. My results do not bear out the supposition that any considerable percentage does, but even if it did the contact to which the children of lepers are occasionally liable and the environment to which they and their leprosy parents are in common subject are agents the operation of which is at least as probable as that of the hereditary factor. Table VII. deals with the children. 450 children had, at the date of my journey through the Central Sudan, been born to the 220 lepers I examined; and Table VII. compares the condition of such of them as were born before their parents became lepers with that of those who were born after.

² Transactions of the Royal Medical and Chirurgical Society, 1902.

TABLE VII.—Comparing the Condition of Children born before the Parents became Lepers with the Condition of those born after.

—	Totals.	Alive.	Dead.	Lepers.	Per- centages.
Number born before the onset of the disease in one or both parents	242	113	129	17	7·02
Number born after the onset of the disease in one or both parents	208	114	94	26	12·05
Total	450	Average age of survivors 16·33 years.			

These figures support all other available evidence on the subject of hereditary transmission of the disease. They suggest that if the children of lepers could be removed from contact with their parents and effectually guarded from the influence of the agencies under which the parental disease was contracted, very few, if any of them, would ever develop the disorder.

Disturbance of sexual functions by leprosy.—This branch of the subject is closely connected with the last, for an impairment of procreative power which usually culminates in sterility cannot be considered a suitable medium for the cultivation of anything that is hereditary. That advanced leprosy does interfere with the effective discharge of the sexual functions seems beyond doubt. That this interference is in proportion to the stage of the disorder is also clear; and, though lepers may and often do beget and bear children, the period during which such occurrences are usual may in persons affected by a progressive form of the disorder be limited to the first few years immediately succeeding invasion; whereas in the later stages, when the grosser alterations of structure are accomplished and the vital processes are more gravely modified—by the time, in other words, that the parents have anything to pass on to their offspring—it may be taken as a general rule that they are completely sterile. Table VIII. illustrates this state of things.

TABLE VIII.—Dealing with the Fertility of Leprous Couples.

Condition of couples.	Number of couples.	Number of persons those couples represent.	Average term of cohabita- tion in years.	Total number of children born.	Average number of children per couple.
Husband leper; } wife healthy ... }	45	80	6·85	36	0·8
Wife leper; hus- } band healthy ... }	28	54	8·5	49	1·75
Both parents } leprous }	58	100	7·5	72	1·24
Combined	131	234	7·49	157	{ 1·19 nearly.

Table VIII. needs little explanation. It is evident that if 131 couples, living together on the average nearly seven and a half years apiece, only produce as the fruit of their cohabitation during that time one child and a fraction apiece some influence capable of interfering with propagation must be at work—and leprosy is the common factor. The application of the table to hereditary transmission is also clear. 234 adults, about 130 of whom are leprous, have as a measure of their procreative capacity 157 children—all these children are, of course, born after the onset of the parental disease. Table VII. points out that of children actually born of affected parentage only about 12½ per cent. ultimately develop the disorder. Let us assume that 20 per cent. do and we shall still have less than 30 leper children with whom to fill the places of somewhere about 130 leper parents. It is needless to point out that if the diffusion of leprosy depended to any extent on such a process as hereditary transmission of the disease the disorder would,

in these circumstances, have long ago become of historical interest only.

The predisposition to leprosy.—The place of predisposition in leprology is wide and beset with many difficulties, but for the purposes of a paper which is merely an analysis of so many cases of the disease the subject is narrowed down to a single point. It is, of course, necessary to assume that some peculiar state of being, such as that denoted by the term “predisposition,” is essential to the development of the disorder. That being granted, the only question with regard to which anything like a decision can be arrived at by statistics of the kind that I have to offer is whether the origin of the predisposition is to be found in the leprosy of a parent or more remote ancestor or not. If the leprosy of a progenitor be not its source it must of course be derived elsewhere, but that is another matter and outside the scope of this analysis. The question at issue here, then, runs as follows: Is the particular condition of body which is assumed to allow the bacillus of leprosy to attack the tissues of an individual with a prospect of success due to the effect of leprosy on one or more of that individual’s ancestors, or is it not? In other words, is the tendency specific or otherwise? And in view of the results of my analysis I can hardly do other than head the few paragraphs and tables bearing on this point as the case against the specific tendency.

The case against the specific tendency.—In dealing with the lepers who came under my observation I found it possible in most instances to get a pretty full and fairly well substantiated family history. In the bulk of the cases I was able to assure myself of the condition of the grandparents as well as of the parents, and as in some instances my records reach to the great-grandparents also it may be assumed that there is, on an average, a background nearly two generations deep supporting each case. Now it is certainly possible for a given morbid tendency to remain latent through several generations in a direct line and it is conceivable, therefore, that a susceptibility having its source in the leprosy of an ancestor may possibly, acting through the intervention of many individuals, come at last to be a factor in the causation of the disease in a descendant. But a susceptibility of this kind cannot be regarded as exercising any special influence on the spread of leprosy. It is difficult to be sure of its existence. It is evident that if several individuals intervene between the leprous ancestor and the suffering descendant any or all of them, including the ultimate leper himself, may have acquired the disposition, which is assumed to be necessary, under the influence of agencies in no way connected with ancestral disease; and as it is certain, from the occurrence of leprosy in individuals whose ancestry has been quite free from taint, that the predisposition may be acquired in this way, it must be admitted that the balance of probability is against the former alternative. It may be stated, then, as a general proposition, that it is unlikely that the operating tendency in any given case is derived solely from leprosy when the family history of the subject can be ascertained to be free from taint for any considerable period, it being probable in such a case that causes of a simpler and more general nature are responsible for its occurrence. It is on this understanding that the following table divides my cases.

TABLE IX.—Dividing Cases with Regard to the Presence or Absence of Leprous Taint in the Direct Family Line.

—	Numbers.	Percentages.
Number of cases in which the direct family line was free from taint for at least two generations	159	72·27
Number of cases in which leprous taint was admitted	52	23·63
Unclassified	9	4·09
Total	220	—

It will be seen that in this table we get about 25 per cent. of a given number of cases in which it might seem possible to ascribe the predisposition to ancestral disease. Table X., however, which divides the cases in which taint was admitted according to the generation affected by the disease, shows that this percentage is liable to diminution.

TABLE X.—Cases in which Leprous Taint was admitted, arranged according to the Generation affected.

—	Numbers.	Percentages.
Number of cases in which the taint was } parental	36	16·35
Number of cases in which the taint was } grandparental or farther removed still }	16	7·27
Total	52	—

While in Table XI. the 36 cases in which parental taint was admitted are further divided with reference to the condition of the parent or parents, whether healthy or leprosy at the date of the birth of the leper examined.

TABLE XI.—Cases in which the Taint was Parental, divided according to the Condition of the Parent or Parents at the date of the Subject's Birth.

—	Numbers.	Percentages.
Number of cases in which one or both } parents were actually lepers at the } date of the subject's birth	18	8·18
Number of cases in which the parent or } parents were lepers, but only became } so after the date of the subject's birth }	12	5·45
Unclassified	6	2·72
Total	36	—

It will be seen that these tables (Nos. IX., X., and XI.) afford direct evidence as to whether the predisposition to leprosy is specific—that is, whether it is due to the influence of the disease on the person of a parent or more remote ancestor or not.

On reference to Table IX. it is evident that out of the 220 persons examined only 61 could reasonably have ascribed their tendency to the effect of the disorder on a parent or other ancestor, since for two generations at least the immediate progenitors of the remaining 159 had enjoyed good health so far as leprosy is concerned. This suggests that in at least 70 per cent. of the cases the tendency must have been of general origin. Moreover, of the remaining 61 persons whose predisposition might possibly have been derived from ancestral disease, in 16 (Table X.) a healthy parent intervened between the affected grandparent who was the possible source of the tendency and the patient, while in 12 (Table XI.) the “ancestral” disease was only developed by the parent after the birth of the person to whom he is to be supposed to have transmitted it. When we further extract the nine cases (Table IX.) which are only included as having taint in the direct family line because nothing definite could be ascertained about them, we shall find that we are left with a remainder of 18 cases (out of 220) in which, as the disease was parental and antedated the birth of the person under examination, it would not be unreasonable to consider it a possible source of the tendency. But if the leprosy of an ancestor were even an ordinary source of the tendency in a descendant, one would naturally expect to be able to demonstrate the reasonable possibility of the relationship in a larger proportion of cases than 18 out of 220.

The effect of family contact.—However the predisposition to the disease may be acquired it is evident that a large number of people must from time to time develop it and to these people at such times continued contact with leprosy means the possibility of contracting the disease themselves. Now it is likely, considering the patriarchal style of the Sudanese ménage, that a large number of those persons examined by me, in whose case it was found that either a parent, grandparent, or more had suffered from the disorder, had been subject to personal contact with leprosy at an early age—at an earlier age for example, all other things being equal, than those who having no leprosy in their own homes had to go outside for their opportunities of contact. That there is some foundation for this idea is suggested by Table XII., which shows that of the persons examined those brought up among surroundings in which

family leper contact was a possibility did actually develop the disorder at an earlier age than those from whose early associations that contact was probably excluded.

TABLE XII.—Dealing with Ages at which the Disease was developed, divided according to the Presence or Absence of Leprosy in the Family.

Condition of family.	Number seen.	Average age of onset of disease.
Cases with leprosy in the family	52	13·26 years.
Cases without any leprosy in the family	167	20·83 years.

Table XIII., which deals with the condition of over 700 individuals, the brothers and sisters of the persons whom I examined, illustrates a further probability in this direction. It compares the number of lepers among the brothers and sisters of such of my patients as admitted a taint in the direct family line and who, therefore, might have been in early contact with leprosy, with the number among the brothers and sisters of those whose direct family line was free from taint and whose early years were therefore not so likely to have been subject to home contact.

TABLE XIII.—Condition of the Brothers and Sisters of my Patients, divided with reference to the Freedom from Leprous Taint or the Reverse of the Family Line.

Condition of family.	Number of individuals.	Average age of survivors.	Number of lepers among them—alive and dead.
Condition of brothers and } sisters of persons with taint } in the direct family line... ..	166 (dead 76)	22·05 years.	13
Condition of brothers and } sisters of persons without } taint in the direct family line	560 (dead 307)	30·58 years.	17

On the showing of these two tables (Nos. XII. and XIII.) it certainly appears that persons exposed to direct contact with leprosy are more liable to contract the disease than those farther removed. They have, therefore, some bearing on the question as to how far the leper himself is responsible for the continued maintenance of his disease.

Sanitary conditions.—The general sanitary conditions of the various centres of population in the parts of the Sudan visited by me are much the same as those of semi-civilised tropical communities elsewhere. Distinctly disgusting on the surface they are probably much less offensive in reality than might at first appear, but as they have no special bearing on the endemic disease it is not desirable to enter into any consideration of them here. Of the personal habits of the people, however, it is necessary to say a word or two. With regard to cleanliness of body the average Sudanese native is influenced by considerations of appearance, comfort, and convenience rather than by any abstract longing after cleanliness for its own sake. If he lives near a pool or river he will wash his body because it looks nice to be clean and feels nice to be cool, but if it entail any effort to compass these ends he prefers to put up with the inconvenience of the dirt rather than incur the trouble of removing it. So it happens that in such a place as Kano the surfaces of the bodies of thousands are from one year's end to another undisturbed save by scratching. With the habits of the people relative to clothing and bedding I have dealt fully elsewhere;³ it is only necessary for me to sketch the barest outlines here and to repeat that I believe that they have an important influence on the diffusion of leprosy in these regions. Native cloth is woven in narrow strips which are afterwards stitched together to form the piece. Garments made from these pieces do not stand washing well. For prudential reasons, therefore, as well as from habitual indifference to dirt, clothes and bedding are rarely washed, and both are handed about freely from person to person. As I have dealt with the mechanism of this circulation of clothes before there is no need for me to go into it again. It is only necessary to state here the fact that the bulk of the cloth in personal use in the

country has never been washed and is loaded with lice and that the habits of the people make it probable that any given garment that cloaks the shoulders of a man of the people has had a past during which it has done similar service for many previous owners. For the special connexion between this state of things and the course of the disease in the Sudan I must again refer my readers to my former paper on the subject.⁴

Diet.—The natives of the part of the Sudan I am writing about are chiefly vegetarian in their habits. They have no religious or other objection to meat, but they are not hunting races and the bulk of the individuals are gathered together in towns under conditions that make meat both scarce and dear. North of 8° north latitude, the mass of the people subsist on a dish called *tuo*. This is a porridge-like mess, the usual basis of which is the flour of the guinea corn (*Sorghum vulgare*), but other flours, such as that of rice, are sometimes used. This *tuo* is washed down by a vegetable soup. South of 8° north latitude the yam (*Dioscorea sativa*) is very extensively eaten and sweet potatoes (*Convolvulus batatas*) are procurable almost everywhere. Even in the south, however, *tuo* is still the popular dish. Besides rice, guinea corn, yams, &c., the other grain and vegetable products used as food include manioc, onions, beans, a grain known as *atcha* (*Pennisetum typhoides*), and a little wheat. Where the country is populous and quiet, cows are kept, sour milk is drunk, and a small amount of butter is made. In the largest towns and where there are Arabs bread and soft native cakes (*wéna*, *massa*) are obtainable, but these have little connexion with the diet of the masses. Oils obtained from monkey-nuts and the kernel of the shea butter tree (*Bassia Parkii*) are used for culinary purposes, and, by a treatment not unlike our own malting, guinea corn is made to produce an intoxicating soup (*gia*).

Now I do not wish to pay the price of brevity by being misunderstood. I am not saying that animal food is not eaten in the parts of the Central Sudan affected by leprosy, because it is. The village must be a small one that cannot be relied upon to produce fowls and every roadside town has its flock or flocks of goats; and both goats and fowls are killed and eaten. Moreover, in the large towns cattle are slaughtered often, in some daily, and in Kano market even camel meat may occasionally be obtained, but in no case is the amount of animal food at disposal large enough, or its price low enough, to permit of these circumstances having any material influence on the diet of the masses. The only form in which, with any regularity, meat reaches the lower strata of society is that of *kilishi*. The trade in this commodity is in the hands of petty dealers comparable to those who in our own country manage the smallest and least reputable variety of fried-fish shop. The meat of the day before is bought cheaply by these dealers, cut into wafer-like strips, highly seasoned with pepper, roasted crisp, and sold hot, and it is bought by the people who eat this kind of thing in minute quantities, and used, not as a nourishment, but as a relish. With *kilishi* begins and ends many a poor-class Hausa's acquaintance with meat.

Of fish as an article of diet, and of the limitations affecting the supply of it in the regions with which I am concerned, I gave some details in my reply to questions put after the reading of a paper of my own referred to before.⁵ They were to the effect that the quantity of fish on sale in representative markets is, for sundry easily comprehensible reasons, small, and that what there is of it is, as a rule, dearer than meat. Fish, therefore, is consumed by the masses of the country even to a less extent than meat. Throughout the whole country salt is very scarce and dear.

Attitude of the people toward the disease.—Except in so far as personal tastes are concerned the attitude of the people among whom I mixed toward leprosy is one of absolute indifference. In the Central Sudan lepers are free in every way and no restraint whatever is imposed upon them. They may live where and how they like, mix with whom they will, engage in any occupation they may fancy, and marry anyone who will have them. Marriage, however, is one of the relations of life into which the element of personal taste referred to above enters largely. An advanced leper does not often marry a healthy woman, not because he is debarred by rule from doing so, but because most healthy women will have nothing to do with him. It often happens, too, that when one of a married pair develops leprosy and becomes offensive the healthy partner leaves the other when

the latter event takes place. The laxity of the institution that is looked upon by these people as marriage renders this quite an easy and ordinary affair. This separation is not due to fear of contagion but to natural physical disgust at the condition induced by the disease. Broadly speaking, the average native may be said, with regard to leprosy, to have no opinions, beliefs, or superstitions whatever. Now and then an extra intelligent man is met with who relates a chain of circumstances which has been sufficiently marked to attract his attention and which has apparently almost persuaded him to become a contagionist, but as a general thing inquiries on this subject even from educated natives elicit little more than the leisurely shrug of the shoulders and the upward movement of the eyebrows by which they may be understood to refer the matter to the inscrutable decrees of Providence.

Summary.—The facts and figures of the foregoing analysis will be found, with a few inconsiderable exceptions, to harmonise with those previously obtained. The age of onset appears to work out earlier for the Sudan than in some other exploited areas, but it is quite possible that this may be due to some local conditions that have escaped my notice and that are not represented elsewhere. It certainly appeared to me, however, that the period during which the system is subject to the constant change and strain attending growth and development might reasonably be regarded as one more likely to be marked by the invasion of such a disease as leprosy than later and more settled periods of life. Beyond this deviation there seems to be little else to give rise to discussion.

The question of recovery from the disease is interesting but it is not new. Evidence in favour of its occurrence has been given unconsciously as well as intentionally by almost every investigator whose records are available. The possibilities that exist in this direction seem to me to be large and I do not think that sufficient advantage has been taken of them.

With regard to the hereditary factor, it will be found to receive as little support from my results as from any others. The evidence afforded is against the supposition that the spread of the disorder is even remotely affected by any such process as hereditary transmission of the disease. If a leprosy person be able to pass on his disease to posterity the results of the possibility of such a transmission should be most strongly marked in his immediate descendants—that is, his children—and there should be evidence of at least the likelihood that he derived his own disorder in a similar manner. But this is the kind of evidence which my results do not supply. Very few of my lepers were born of either leprosy parentage or grand-parentage, and even with all the risks attending constant home contact with leprosy only 9.5 per cent. of their children ultimately developed the disorder. Moreover, leprosy is a disease that reacts prejudicially on propagation, and it is difficult to see how a disease that does that can reasonably be regarded as deriving support from a factor that is hereditary.

With regard to the sources of the predisposition to the disease, assuming its existence, much the same may be said. Whatever may be its source, it is most certainly not specific, not always due to the leprosy of an ancestor. Many instances are on record of leprosy having occurred in people who have had no leprosy ancestors. And further, as already shown from the effect of the disease on the reproductive faculty, persons affected with severe grades of leprosy, far from being likely to bestow morbid tendencies on posterity, are much more apt to have no posterity at all. That the leprosy of an ancestor is not the source of the tendency in descendants we are probably on safe ground in assuming, and opinions on this point would most probably be unanimous; as to where the tendency is derived, however, whether from a single source or otherwise, and how, and what is its exact place and importance among the factors that maintain and diffuse the disease, they would hardly be so unanimous. For myself I think that deficiencies of diet have much to do with it. I have already indicated the line my ideas take in the matter,⁶ so have not thought it necessary to touch on it in the body of this paper. The same consideration has withheld me from discussing the relationship between the disease and the habits of the people with regard to clothes and bedding, but I should certainly like to draw attention once more to the broad lines of resemblance that are displayed in these particular matters in

⁴ Ibid.⁵ Ibid.⁶ Ibid.

all the leper fields of the world. In other particulars there is the greatest possible diversity. Suffering from a common disease, we have men of all colours, races, religions, and habits. We have leper fields on the line and within the Arctic circle, but everywhere leprosy is we have habits with regard to clothes and bedding which are open to criticism and stereotyped national diets which I think most of us will admit leave a good deal to be desired. That in the Central Sudan the communistic way in which unwashed clothes are handed about has a direct influence on the spread of leprosy I cannot doubt; and that a diet in which the carbohydrate elements are represented to an extent which disturbs the proper proportion of the nitrogenous should handicap races reared on it in their opposition to the advances of disease appears reasonable to me. That an ill-assorted diet can cause leprosy, however, or that the disease cannot be contracted without the intervention of such a diet, are opinions that I do not hold. This one, however, I do hold, that in the Central Sudan the most frequently operating factor, not in causing the disease but in assisting to determine its incidence, is that of a badly balanced and therefore inefficient diet.

Hanley.

NERVE SUTURE AND NERVE REGENERATION.

By PAUL B. HENRIKSEN.

FORMERLY ASSISTANT SURGEON AT RIGSHOSPITALET, CHRISTIANIA.

(Concluded from p. 1022.)

[THE following paragraph sums up the points observed macroscopically after the operation described in the last section of the first portion of Dr. Henriksen's paper.]

To account for a possible presence of asymmetry, in some of the animals the nerve was sutured on the right side and interrupted on the left, while in the rest of the animals the contrary was performed. Comparison of the weight of the muscles on the side of the united and on the side of the interrupted nerve proves that the difference up to the thirty-fifth day is comparatively small and irregular and will scarcely allow of any conclusion considering the limited number of experiments and the imperfection of the methods, such as inaccuracy from the excision of the muscles and possibly some evaporation during the excising and weighing of the muscles. From the thirty-sixth day the series takes a more definite character. On the side of the united nerve a constant and rapidly increasing excess occurs. The experiment must be supposed to be a confirmation that the muscle by this time must have resumed its action—a considerable time before it can be proved by the electrical current. The microscopical examination of the samples will be treated in conjunction with the examination of the samples from the other series.

MICROSCOPICAL EXAMINATION OF NERVES.

For hardening of the nerves Müller's, Flemming's, or Marchi's fluid and formalin have been used. The nerve while hardening was extended on a piece of wood and was then imbedded in celloidin. Then there were made longitudinal sections of considerable length with the microtome. Some of the samples were teased in osmic acid, but in this way I only succeeded in getting fibres in an advanced stage, while those in the earlier stages, especially near the place of union, did not come out at all, or only as fragments in a way that allowed no conclusions as to their origin. Loosening the fibres in the sections succeeded better in getting characteristic details without tearing them totally from each other, so that the sections after the treatment had the appearance of a net with long meshes, and the fibres almost kept their mutual position. In this way it was possible to see all the components of the section in such a manner that their mutual relation might be easily understood. At the same time in the splits between the loosened fibre bundles it was possible to see single fibres and combinations of loosened fibres in different stages of their development with a clearness that I could not get in any other way. The loosening of the fibres was easily performed during the clearing up in oil or xylol.

Of great importance for the study of the regeneration of nerves is Weigert's myeline sheath stain. It furnishes

good information about the first formation of myeline sheaths, gives sharp and clear pictures of the newly formed fibres, and is indispensable for understanding the preparations stained by other methods. In some cases staining with osmic acid and safranin has been employed. The method gives beautiful and illustrative pictures concerning the division of the nuclei and the relation of the nuclei to the old and new fibres, but it does not exhibit so clearly the finer changes of structure by the formation of the myeline sheath. Van Gieson's fuchsin-picric acid and hæmatoxylin stain gives excellent information about cells and nuclei in the regenerating nerve and in combination with Weigert's myeline sheath stain about the formation of the new fibres.

For the sake of clearness the central part and the peripheral part of the united nerve will be described separately; then will be mentioned the changes in the central and peripheral part of the interrupted nerve—that is to say, where union has not occurred; and lastly will be treated the changes at the place of union.

THE CENTRAL PART OF THE UNITED NERVE.

Fifth day.—(Flemming's fluid, safranin.) About half a centimetre from the place of division are found nerve fibres of uniform colour with broad brownish-black margins on both sides. Approaching to the place of division the nerve fibres are coloured less intensely and are more of a greyish-brown with thinner margins. They are coloured unevenly and have uneven margins, just as if they were corroded, and at last they are only slightly discoloured. In a few of the fibres the myeline sheath is broken into segments. The process is thus not quite the same in all the fibres; in most of the fibres the myeline sheath evidently is absorbed towards the place of division and only in a few of the fibres there are coagulation and breaking up of the myeline sheath. In the neighbourhood of the division place there is only a striped brownish tissue with numerous nuclei.

Seventh day.—(Flemming's fluid, safranin.) The absorption of the myeline sheath begins farther from the place of division, almost one centimetre, and is more pronounced. In some of the fibres the myeline sheath is broken up in irregular places. In the section are seen long rod-shaped or oval nuclei increasing in number approaching to the place of union. Some of them are buried in the nerve fibres, that are almost normal in colour, taking up half the diameter of the fibre and being surrounded by a lighter protoplasm zone that in some places is continued somewhat forwards and backwards along the fibres. (Plate IV., Fig. 1.) Other nuclei are placed between the fibres where they show thread-like processes. In the sections these processes are seen to form long light grey threads that disappear in a nerve bundle or join together with a nerve fibre. Some of the threads are bent and twisted, and by this they may be seen to be flat, being more narrow when they are seen from the edge than from the flat. Some are only (slightly) light grey, others are darker grey, brownish, and show a distinct double contour margin and thus must be understood to be real nerve fibres. In some places the red nucleus may be seen lying by the side of an old fibre, being attached to this by processes from both ends. Near the place of division may be seen bundles of these newly formed fibres, with long red nuclei torn off from the striped tissue that forms the continuation of the old nerve fibres. The nuclei are dividing; some of them are larger and more vesicular, in others the chromatin is deposited on both sides of the long axis; in other places the nuclei are seen in pairs divided in a longitudinal or somewhat oblique plane, the one being displaced somewhat in the longitudinal direction. Besides these long nuclei there are nuclei of a round or oval appearance; they are not so intensely coloured, are larger, and seem to be flat. Some are seen on the outer side of the unaltered nerve fibres, and others are seen in membranous bands in the teased sections. Besides these two forms there are seen irregularly shaped cells entering in between the lumps of myeline in the divided myeline sheaths of some of the nerve fibres. They seem to be of no importance to the regeneration and therefore will not be further alluded to.

Twenty-fourth day and thirty-sixth day.—(Marchi, van Gieson; Flemming's safranin.) For more than one centimetre from the place of union there are found newly formed fibres. They increase in number approaching to the place of union where there is not found a single fully developed or normal fibre. They have distinct, even double-contoured,