

Th. Scott.

L E P R O S Y

WITH SPECIAL REFERENCE TO ITS OCCURRENCE

IN THE PROTECTORATE OF GAMBIA.

T H E S I S

Submitted for the

M.D. EDINBURGH

by

A. M. Wilson Rae,

M.B., Ch.B. 1921.

M.O. Gambia, West Africa.



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## I N T R O D U C T I O N .

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The choice of "Leprosy" as a subject for submission as a thesis has been made for three main reasons.

Firstly because I have been enabled to see and examine lepers showing the disease in very many of its different phases, and to realise the complexity of leprosy in its clinical manifestations.

Secondly because it has lately assumed an increased importance among tropical diseases as a result of the very great advances in treatment made in the last ten years. Previously considered a disease well-nigh incurable, it is now recognised that this terrible scourge is amenable to treatment; that to the early case not only is there hope of complete cure but almost certainty of it, and that even the advanced leper may look for amelioration of symptoms and even a termination of the active disease.

Thirdly, and most important of all, because little or nothing was known of the extent of the disease in our West African Colonies and Protectorate with the possible exception of Nigeria.

A vast organisation is at work with the avow-

ed intention of stamping out leprosy throughout the Empire. This can never be done, unless full knowledge be obtained of the extent of the disease and unless the local conditions in each area be understood.

While realising very keenly that this paper offers no real scientific increase to our knowledge of the disease or its treatment it is hoped that it will throw light on those two points mentioned above, while offering at the same time sound corroborative evidence of various facts in the disease - of which proof of communicability is probably the greatest.

This Protectorate of the Gambia even by its mere geographical position alone makes the tackling of the problem at once a very difficult thing - and one peculiarly its own.

I append in "The Annual Medical and Sanitary Report for The Colony of The Gambia" 1926, pp. 66-75, a report submitted by me to the Government on the subject of the marked incidence of the disease. The figures given then which showed definitely that the disease is extremely prevalent, are now I find grossly underestimated. The leper problem is a very serious one - a condition of affairs not previously realised here; -



nor for that matter has it been realised elsewhere among our colonies on the West Coast of Africa. In 1917 Castellani and Chalmers "Manual of Tropical Medicine" stated that leprosy "is certainly rare in West Africa"; and as late as 1925 Rogers and Muir "Leprosy", speaking of the Gold Coast Colony state "leprosy is present but little is known of its prevalency which is probably not great."

If this be considered the true state of affairs then no thorough tackling of the problem of eradicating the disease is possible. The cause is of course because no definite search and count is made.

Such a search must be very thorough or totally misleading figures will result. It must entail a search through every village and every yard in each village; because these backward people are so lethargic that only a small number will voluntarily come forward.

A short description of the country will aid in showing some of its peculiar difficulties.

The Gambia Protectorate lies on the West Coast of Africa in latitude 13° N. and is a narrow strip of country eight to ten miles broad on either side of the river of that name, and extending inland in an easterly

direction for a distance of three hundred miles. The whole country is flat and low-lying; considerable areas near the river are flooded all the year round, while in the rainy season very large tracts of country are under water; and then the villages lie closely surrounded by swamps.

The country is surrounded on all sides except to seaward by French Senegal; between which country and our own there is constant daily communication.

It is by no means a rich country; agriculture alone gives employment. The only crop grown is groundnuts of which one crop a year is obtainable. The working of this crop is done almost solely in the rainy season. To aid in this work very large numbers of "strange farmers" come every year from Senegal, working as labourers in the fields; and they very often eat and sleep with the native Gambians - a point of no small importance in the spread of the disease.

Scattered along the river are small wharf towns, scantily populated in the rainy season but at times so crowded in the dry or trade season - when possibly six ocean steamers are being loaded - that the labourers have to lie crowded together in the streets and sanitary super-

vision is extremely difficult.

Apart from the town of Bathurst on the coast, where there is a good hospital adequately staffed, the entire country is looked after by one travelling medical officer. The villages are so scattered that many areas cannot be visited more than once a year. A native dispenser under the supervision of the travelling medical officer is in charge of a small hospital at M<sup>C</sup>Carthy - one hundred and sixty miles up river - and thus there are at present only two places where continuous treatment is obtainable.

The entire country is malarious, and of recent years epidemics of relapsing fever of virulent type have swept over certain areas leaving the people in an exhausted state with small power of resistance to any infection.

These various points have been stressed because they have a direct bearing on the problem.

## H I S T O R Y.

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It is a fact generally admitted that Leprosy existed in very ancient times. Some authorities however - notably Molesworth in Australia - consider that this has not been proved and that the disease supposed to be leprosy was probably some other skin disease e.g. leucodermia syphilis etc. But leprosy in its advanced stages is a disease so startling in its manifestations that it seems impossible that the ancients would not have noted it carefully.

The earliest reference appears in the "Ebers papyrus" written in the reign of Rameses II - and relates that the disease existed among negro slaves imported into Egypt from the Sudan. This "papyrus" also contains prescriptions "for the cure of a disease characterised by nodosities and pain".

Frequent reference is made in the Bible to leprosy; but it is probable that what was called leprosy included a group of diseases of which leprosy was one, for the statements "leprous as snow" and also that it was a highly contagious though easily curable disease, give no true picture of leprosy.

In Perisa the disease existed before the days of Herodotus - and the wife of one of the kings - Artaxerxes - is stated to have suffered from it.

In India it is described under the name of "Kushba" in 1400 B.C.

It was first noted in Japan in 1250 B.C. but no reference as to it occurs in China before the second century B.C.

The general consensus of opinion is that leprosy first appeared in Africa in the Sudan and spread thence to Egypt and later to Europe and all over the world.

#### SPREAD TO EUROPE.

Hippocrates in 400 B.C. refers to the disease but his description is probably not of leprosy but of psoriasis and the first mention of true leprosy is by Aristotle in 345 B.C. The hordes of Darius and Xerxes recruited from the different races of Africa and Asia had swept over Greece; and on the retiral of these armies thousands were left behind - and from this time we get the commencement and a slowly increasing incidence of leprosy in Greece.

Spread to Italy occurred later following the return of Pompey's armies from the East: - and thence the

disease spread all over Europe - carried in all probability by the Roman Legions to Spain, France, Germany and England. The disease was extremely common in Spain in the tenth and in France in the 11th Century.

The further spread was caused largely by the invasions of the Moors and as a result of the Crusades. The return of the Crusaders not only carried the disease into parts which had previously been <sup>u</sup>inaffected but also caused an alarming general increase in the disease.

Notice of the first laws relating to the disease give us a fair indication of its spread. In 757 in France a law was made prohibiting the marriage of lepers and making it a legal ground for divorce. A very similar law was made by Charlemagne in 789. In Wales a law against lepers was made by Noel Dha, King of Wales, in 950.

The first leper hospital was established in England at Nottingham in 625 A.D. and in Ireland in 869.

The disease spread later to Scotland from England and thence north to the Orkneys and Shetlands - and it is to be noted that when the decline in incidence took place, these islands had still numerous lepers after the earlier infested parts had become almost clear of the disease.



More striking even than the spread of the disease - which we have noted as one of slow growth passing from Africa to Southern Europe and thence west and north over the whole continent - carried by invading armies - and the merchants who always follow in their tracks, - was the very rapid decline in Europe, especially during the thirteenth and fourteenth centuries. By 1550 there were very few lepers left in England - although in Scotland and Ireland it persisted for at least a century later - and in the Shetland Islands was not stamped out till 1798. In France leper houses were abolished in 1695.

In Denmark it had disappeared by the end of the sixteenth century, and a little later in Germany. In Spain it still persists to this day.

THE CAUSE OF THIS RAPID DECLINE may be attributed to two main factors; firstly the stringent laws against the disease - particularly those dealing with segregation - and secondly the improved social and hygienic conditions. Where the laws were lax and where improved conditions were slow in coming leprosy lingered longest.

Lazarettos were established in all the countries of Europe - France in the 13th century had over two thousand of them .

Among the stringent measures taken against the disease the following may be noted:

Lepers were compelled by law to wear distinctive clothes, to carry a clapper and to sound it as they passed along the roads to warn people of their presence. They were forbidden to eat with any person not a leper; to drink from any public fountain; or even to touch food when buying, but were compelled to point it out with a stick. They were forbidden to touch a child, or to enter any church or inn, but forced to live outside the towns. They were complete social outcasts; and this was openly shown as the Church read the burial service over them as soon as a diagnosis of leprosy was made.

A further cause of this rapid decline was "The Black Death" which killed off a larger proportion of lepers than of other people, because of their impoverished state of health.

#### SPREAD IN AFRICA.

Here, as in Europe, the spread of leprosy has followed on the opening up of the country - and the slave traffic must be blamed especially for this. Mungo Park states that the first lepers in West Africa were slaves introduced from the Sudan. In addition to the slave

caravans there was also the opening up of the country by huge caravans of merchandise crossing the desert and coming down to the Western Coast, especially through Nigeria.

The spread of Mohamedanism - with its accompanying lethargy and absolute want of any precautions - has contributed greatly to the increased incidence of the disease.

#### SPREAD TO AMERICA.

The original discoverers of the Continent sailing as they did from Spain and Portugal - which at that time was full of leprosy - may have introduced the disease into America; but the rapid increase was due to the transportation of African slaves of whom 1,300,000 were carried to Central America before the end of the sixteenth century. Prior to this time leprosy was non-existent among the natives of the Western Hemisphere.

#### SPREAD TO OCEANIA.

In the latter half of last century a further spread of the disease took place - to the islands of the South Seas; of which the Loyalty Group, Hawaii and New Caledonia were chiefly affected. This spread has been traced to emigrant Chinese; who probably also carried the

disease to Australia and to the Eastern Archipelago.

#### EXTENT OF THE DISEASE.

Various estimates have been made of the total number of lepers in the world. Rogers in "The Croonian Lectures on Leprosy Researches" stated the number to be three million. When it is remembered that for every visible case of leprosy there is probably another not diagnosed it will be realised that no true total is possible.

Of this three million, one and a quarter millions are in Asia, and of these half are in China. India and Japan each has well over a hundred thousand.

Europe contains about seven thousand lepers of whom the majority are to be found in the south-eastern area especially round the Balkans, although an appreciable number are in Spain and Portugal.

The number in Africa is said to be about a million. There are vast areas in Central Africa where no count at all has been made. This is so even in the partially civilised countries, and the probability is that this total is an under-estimate. At least one third of the total number of lepers in The British Empire are in British Central Africa. This is an estimate only; since

for three of the four Western colonies viz. The Gold Coast, Sierra Leone and the Gambia no figures were available. As greater interest is taken in the leprosy problem in West Africa, true figures will be produced, and I feel certain that an appalling incidence will be demonstrated.

In the French countries adjacent to our different West African colonies, where figures have been produced, the extent of the disease has been enormous. In FrenchGuinea Joyeux found roughly five per mille to be obvious lepers. In the French Ivory Coast, the figure in some parts was as high as 60 per mille.

In the Cameroons in 1902 the number was estimated generally as 20 per mille, and in one area 250 per mille, i.e. one in every four persons seen was a leper.

In the Upper Senegal Niger Area 10 per mille is the figure given.

The only part of British West Africa where figures are available is Northern Nigeria, where an estimate of 10-20 per mille is given.

The number per mille in The Gambia I have stated (vide Appendix) as 1.4. This admittedly is on insufficient data, but I am absolutely convinced that more know-



ledge would not have reduced but greatly increased this number. When we remember that the figure for India is .3 per mille, the importance of this leprosy problem in British Tropical Africa becomes apparent.

There is constant intercourse between these natives and those of the surrounding country of Senegal - and so with the other colonies and their neighbours.

When a true count is made in British West Africa we shall have a problem to face as serious as our neighbours.

#### ETIOLOGY.

Leprosy is due to the presence in the system of a specific micro-organism - the micro-bacterium leprae or Bacillus Leprae - discovered by Armaner Hansen in 1871 - although prior to this Virchow and Vandyke Carter had noted the presence of granular matter in the lepra cells and Mansen had actually attempted the cultivation of the "germ" he believed to be present in the nodules.

Prior to Hansen's discovery various theories had at different times been postulated.

We have already noted that from earliest times it was believed to be highly infectious and that strong



measures were taken against it.

The first definite theory was:-

The Hereditary Theory of Danieleesen and Boeck. They were not the originators of the theory, which has been held in Nigeria in Africa and also in the Far East for centuries, but their publications in its support brought it prominently to the fore. They regarded as evidence the appearance of two cases of the disease within four generations of a family - disregarding entirely whether the later parents were born before or after the infection of the ancestor from whom they were considered to have been infected. This theory has been completely refuted now. Rogers produces three main arguments as final proof of the falsity of this theory.

- (1) The diminished fecundity of lepers - an indisputable fact.
- (2) The spread of leprosy under conditions excluding Heredity as a causal factor. (1) The enormous spread in Oceania in twenty years - a spread so great as to entirely exclude heredity and (2) the infection of Europeans there although their ancestors had been free for many generations.
- (3) The absence of the disease when it should be present

if an hereditary disease. As proof Rogers quotes the case of one hundred and seventy Norwegians who migrated to the United States of America, when either suffering from the disease or in the incubation period; and yet when Hansen visited America not a single descendant up to the third generation had developed leprosy.

#### THE FISH THEORY.

Associated with the name of Jonathan Hutchinson - although again this theory had been believed in for many years before Hutchinson gave it prominence. This declared leprosy to be due to excessive consumption of badly preserved or decomposed fish. Hutchinson himself modified his theory later on, and it is now merely of historical interest. Probably the fact is that such a diet being deficient both in vitamins and in food content, brought about an impoverished state of health and thus provided a suitable nidus for the disease.

Now it is generally accepted that the *B. Leprae* is the cause of the disease, even although it does not meet Koch's postulates.

Morphologically *B. Leprae* bears a very close resemblance to the *B. tubercle bacillus*. It is a slender

rod, non-mobile, grain-positive and acid-fast. Muir states they may be distinguished from the tubercle bacilli by:-

- (1) The bunch-like arrangement of the leprae bacilli.
- (2) Their greater number in a lesion.
- (3) The sites where they are found.
- (4) They are more easily decolorised by dilute acids while the tubercle bacilli are more easily decolorised by alcohol.
- (5) If injected into guinea pigs they do not reproduce disease.

Kedrowski holds that microscopically they are indistinguishable, and that there is certainly no known means of distinction in the case where the material is unknown.

The appearance of the organism varies according to whether the patient be in a quiescent or reactionary phase of the disease. If from a quiescent case then we get the uniformly staining rod described above. In the reactionary phase, some may remain so or parts may not take on the basic stain and we get diptheroids and various spore-like forms, or just beaded rods.

CULTIVATION.

It is a very debated point as to whether the bacillus has ever been cultivated or not. Osler says "It has been cultivated but with difficulty - and is stated to have a pleomorphism of which the bacillus as seen in the tissues is only one stage." Rogers and Muir hold that no certain evidence has been produced to prove that the bacillus has even been cultivated outside the body. They admit that various organisms have been cultivated from leprous lesions and divided these up into four main groups.

- (1) Diptheroids.
- (2) Chromogenic acid-fast cultures.
- (3) Non pigmented acid-fast cultures.
- (4) Anaeroid bacilli.

Kedrowski states that this statement disregards the work of many research workers; and that they have no right to postulate that the finding of acid-fast and non-acid fast bacilli in a culture, is in all cases due to accidental contamination. He says that "We know very well that in a pure culture of Tubercle bacilli non-acid fast types are often met with, sometimes in considerable quantities - and no one would think of doubting the purity of such cultures. In the second place in tuber-

culous lesions we might very often find acid-fast together with non-acid-fast bacilli, and finally the postulating of such contamination of leprosy nodules that have not become ulcerated - as only from such is the material for cultivation usually taken - is a priori inadmissible, as leprosy nodules, usually situated under the deep layers of the skin, are usually separated from the epidermis by a layer of healthy papillary part, and in order to explain their contamination it would be necessary to admit the possibility of penetration of foreign organism into them from afar through the blood and lymphatic circle". Kedrowski holds that the various forms seen in culture bear a perfectly co-ordinate character, and that just as the tubercle bacillus so the leprosy bacillus may suffer changes in morphological as well as in biological conditions, which cause them on the one hand to lose their acid-fastness, and on the other to change into a form of thread fungus. He considers the bacillus leprae - according to the complexity of its development and structure should be placed in the group of actinomycoses or streptothrix-like microbes - and that the bacillary form is merely one of the forms of its state of being.

Despite this the general opinion is that the *B. leprae* has never yet been cultivated.

The same divergence of opinion exists as to the results of animal inoculation. Lesions have been produced e.g. Stanziols produced active lesions in the anterior chamber of the eyes of rabbits - but no one has so far succeeded in producing progressive disease, similar to human leprosy.

#### PATHOLOGY.

All leprous lesions are either of one of two types or of a mixture of both. These are now called skin-leprosy and nerve leprosy. There appears to be some inter-relationship between skin and nerve leprosy. The more the skin is affected, the less prominent is the nerve infection. Nerve lesions have usually fewer bacilli associated with them, and hence early lesions are more commonly of this type. At one time it was suggested that these two types were the result of infection by different bacilli; but, as we know that the one type passes into the other, this is unlikely. It has also been suggested that small quantities of toxin cause nerve leprosy and large quantities skin leprosy; but this does



not explain why, though in some cases the bacilli pass up the nerve trunks, in other cases they leave them alone.

### SKIN LESIONS.

When the corium has become infected the bacilli may lie latent for a varying period. When spread takes place we get -

- (1) A marked increase in the connective tissue cells and their conversion into fibrous tissue. The more chronic the lesion the greater is the amount of fibrous tissue - as this brings about a self eliminating process - the bacilli being eliminated by contraction. (This contraction is especially well seen among the many cases of ectropion in this country, with scars only comparable to those of very severe burns.) Along with increase in connective tissue cells we get the appearance of
- (2) rounded cells very like lymphocytes. These are readily seen in any portion of skin excised.
- (3) Giant cells are also said to be present - although Rogers considers their appearance due to cross-sections of newly formed fibrous tissue nuclei. Molesworth writing in the Medical Journal of

Australia, is emphatic that we do get giant cells, quoting a case where - even when in section no bacilli could be found - he found a typical tuberculous condition with giant cells - and was able later to definitely <sup>to</sup> prove it to be leprosy. He also states he found not only lepræ bacilli but giant cells in the substance of nerves lying between the nerve bundles.

- (4) Along with these changes we also get dilatation and thickening of the capillaries and oedema of varying amount.

A skin lesion is due either to the entrance of bacilli through an abrasion, and their passage to the corium, or to blockage of a capillary by an embolus. If the bacilli are very few we get radial spread only in the superficial layers but if abundant bacilli be present then this spread includes also the deeper layers of the corium.

Muir describes three main sub-types of skin leprosy, and each of these has a definite clinical appearance readily diagnosed. These types are:-

- (1) Papillary - where the infection is confined to the superficial layer of the corium.
- (2) Interfollicular - where it has spread to involve the

region between the follicles, and

- (3) Sub-follicular - where the deep layers of the corium are affected.

Papillary.

In this form, as a result of the fibrous tissue contraction there is a flattening out of the papillae and inter-papillary spaces: so that the epithelium is thinned out. Such a lesion may show (1) an anaesthetic centre while radial spread is still going on, owing to resolution having taken place or (2) no leprae bacilli in this layer, the infection being passed to the deeper layers.

Interfollicular.

In this case, as would be expected, the bacilli are more numerous. The natural folds of the skin are exaggerated - because the pressure from the oedema is exerted only between the hair follicles, the tension being resisted by the follicles and sweat glands pulling down the epithelium. The skin in such cases has the appearance of mosaic work - large plaques with raised edges.

Sub-follicular.

Here the pressure is not only between but also beneath the follicles, and the appearance therefore is of a smooth glossy skin surface. As this progresses we get

complete destruction of the hair follicles and the appearance of "the tissue paper" skin.

The above description applies to diffuse lesions of the skin. If there be any interference we immediately get the formation of nodules.

Whenever a bacillary embolism lodges in the corium a reaction occurs - the severity of this reaction depending on the size of the embolus. If severe then a nodule is formed, temporary or permanent. A severe reaction may cause the formation of so much fibrous tissue as to prevent any spread and a permanent nodule results. These permanent nodules may also be formed from the lodging of fresh emboli in an area which already has an existing lesion. In this case the fibrous tissue is already formed and blocks the spread from this new embolism thus causing nodule formation.

These nodules consist of fibrous tissue strands and spaces in which are found the bacilli and round cells, - the bacilli lying between the cells. Breaking down of the epithelium as a result of cutting off of nourishment by the fibrous tissue contraction frequently results in ulceration of these nodules.

Occasionally ulceration of one large nodule

without any other sign of the disease may occur and make diagnosis difficult. A case was seen here of a large ulcer of the leg in a boy - from which numerous lepræ bacilli were obtained. No other sign of leprosy could be found, and since the first appearance of the nodule there had been no possible contamination from any leper.

Finally it must be noted that one part of a skin lesion may be of one sub-type, while another part is of one of the other sub-types; and that in the same person different lesions may be of different types.

#### NERVE LEPROSY.

All such lesions are limited to the peripheral nerves. Just as in skin leprosy infection is either directly through the skin or by bacillary embolism, so in nerve leprosy it is either direct - an ascending type with passage of the bacilli up from the cutaneous nerve endings into the nerve trunk, or metastatic through the vasa vasorum from other parts of the body.

The changes which occur in the nerves are similar to those already noted in the skin i.e. proliferation of the connective tissue cells, formation of fibrous tissue and dilatation of the capillaries.



As a result of nerve infection we get marked changes in the epithelium, hairs and sweat glands - marked depigmentation especially well seen in black races, anhydrosis, curving of the nails and loss of the skin hairs which break off having become devitalised.

Reaction takes place here also - although as a rule it is not so severe as the number of bacilli is relatively less. Such a reaction is recognised both in the nerves themselves, which become swollen and tender, and in the parts supplied by them which show rapid destruction, e.g. sloughing of the soft parts and even destruction of the bone, or perforating ulcers may result. This is in contrast to the slow chronic atrophy seen in those cases where there is no reaction. Rogers in "Recent Advances in Tropical Medicine" 1928, lays stress on the fact that the tissues have much greater resisting power against invasion by the lepra bacilli in the nerve type, and that in cases where death occurs long after from some inter-current disease, no bacilli can be found in the fibrous nerve trunks. This unfortunately seldom occurs until extensive deformities have appeared.

Occasionally leprosy may affect the lungs - and in fatal nodular cases the bacilli may be found in



liver or spleen, where they may or may not form visible nodules; and in the adjacent glands they may also be detected.

Infection of other glands in the body have also been described recently.

#### CONTAGIOUSNESS of LEPROSY.

It is now a generally admitted fact that leprosy is a contagious disease, though even at the present time there are a few dissenters. The great difficulty is the long incubation period. This, extending often over years, makes it difficult to trace infection. The importance of this fact of contagiousness of leprosy cannot be over-rated; as unless this be realised leprosy can never be "entirely stamped out" - as Sir Leonard Rogers stated early this year that it would be. Prevention of infection depends on realisation of the possibilities of contagion.

In 1862 at the request of the Colonial Secretary a Committee was appointed to report on the infectiousness of leprosy. This Committee concluded that leprosy was not contagious or communicable to healthy persons by proximity or contact with the disease. They also said

that there was no evidence to justify any measures for the compulsory segregation of lepers.

As a result of this, orders were issued to repeal all laws affecting the liberty of lepers. This brought about an increase in the recorded number of lepers in various parts - especially in British Guiana, St. Kitts and the East Indies. This report stimulated leprologists all over the world to obtain evidence of the communicability of the disease.

Among other proof that produced by Dr. MacLeod, - Honorary Advisor of the St. Giles Homes for British Lepers - published accounts of three contact cases.

- (1) A boy aet 12 with nodular leprosy born in Ireland - and who had never been out of Ireland - His father had emigrated from Russia and was suffering from nodular leprosy at that time.
- (2) A family came to England from British Guiana. One son had leprosy. Another son was born two weeks after arrival in England. He occupied the same bed as his leper brother and himself developed the disease.
- (3) A women who contracted nodular leprosy from her husband in England. He had develped the disease

abroad.

A perusal of the notes in "The Medical and Sanitary Report for the Colony of the Gambia," which I made last year will furnish abundant evidence in support of the theory of the contagiousness of leprosy. Out of one hundred cases fifty-seven give a definite history in its support - and I am confident that it should have been possible to obtain it in a far greater percentage of the cases. Within the last three months I have seen twelve new cases - all of whom gave me a definite history of prolonged contact. Last year a woman with early nodular leprosy came for treatment. She had with her a child of two years. I advised that the child - who showed no signs of the disease - be sent to the grand-parents. This was not done. After two injections the mother did not return until one month ago. The child had then two definite depigmented leprosy patches on the face.

It must be remembered that leprosy is not a highly contagious disease, that intimate and as a rule prolonged contact of a healthy person with a leper is necessary before the former contracts the disease. It must also be noted that all age periods are not alike susceptible, but

that the dangerous times are childhood and adolescence. In forty-three out of the hundred cases I have quoted there is evidence of the disease having commenced before the age of twelve. The relationship between mother and child here is much closer than in Europe. They are always together. The women working in the rice fields have their babies on their backs. Breast feeding goes on much longer - up to two years in some cases. Again, intimate contact between grown-up people is also greater. They lie huddled together in airless huts - a difference of temperature of between 40° and 50° F. is responsible for this. It will often happen that an early case of leprosy lies there huddled close together with the healthy.

Of the various groups I have divided the contact cases into - the greatest is that of "Family Relationship" - which is as we would expect, as that allows the most prolonged and most intimate contact. The other groups I have noted all tend to prove the same thing. Two of these groups are especially important here.

Firstly that group - "Living in the same yard as a leper" is important because of the number of "strange farmers"

mentioned in the introduction. They come in every year - often going back to the same family year after year - with whom they remain for several months as labourers in the ground-nut fields. Often they live with the Gambia family - eating, working and sleeping with some of its members, and in several of the cases quoted they have infected those members with whom they were in close contact. The economic importance of these "strange farmers" makes another difficulty in the solution of the problem here.

Secondly there are the cases where infection has spread through the wearing of the clothing of a leper. This interchange of clothing is extremely common. A shirt of vivid hue will be seen clothing six different boys on six consecutive days. That one was a leper matters not.

The question of immediate infection following inoculation by a wound has been a debateable point. The case quoted where the first leprous patch appeared on the abdomen on the site of an abscess which had been opened - although not definite proof is of interest.

Molesworth, writing from Australia opposing segregative measures, stated that only 66 Europeans out of a population of six millions were affected with leprosy, and



149 natives out of under a hundred thousand - and said this was due to lesser susceptibility among the whites. Dr. Cook of the Australian Institute of Tropical Medicine points out, and rightly one would think, that this is fallacious. He says that if this be so it is strange that the people of Victoria should have so little leprosy compared with those of Queensland - both of whom should possess the same racial immunity. Also, as he says, little notice has been taken of the fact that in Australia there is little inter-racial intercourse - and practically none in the south. It must be noted also that that part of Australia, where the disease is endemic, is in the tropical belt where conditions are most favourable.

Leprosy is without doubt a contagious disease - and conditions where long intimate contact exist are those which most favour its spread.

#### PREDISPOSING CAUSES.

Muir maintains that in all cases the onset of leprosy is due to some predisposing cause in conjunction with infection by the B. Leprae. Some of these causes will remove themselves and of the rest most can be removed by treatment, and this must be done before carrying out

specific treatment, as otherwise there may be rather increase than diminution of the disease. Also it must be noted that the course of the disease is profoundly affected by the nature, degree and number of these predisposing causes.

Leprosy is protean in its manifestation and the diagnosis is often exceedingly difficult. The first signs may be accompanied and clouded by the appearance of some febrile disease as e.g. smallpox or influenza, and it is only with the appearance of the afebrile period that the rapid increase in leprous signs makes the diagnosis simple.

Any thing which weakens resistance over a prolonged period - be it biological, climatic or social - acts as a predisposing cause.

Of pathological conditions probably syphilis and chronic bowel disorders are the most important.

Syphilis is rampant among the natives; - the discovery and adequate treatment of it help greatly in the case of the leprous infection.

Two years ago I was able to show that 80% of the native population suffer from intestinal helminthic disease - and of these a large proportion had ankylostoma infection.

In nearly every case of leprosy, that I have been able to treat, carbon tetrachloride medication has shown the presence of ankylostoma or taeniae.

In addition many of the people suffer from obstinate constipation with consequent absorption of toxic products, the result of untreated dysenteries. Malaria - a third great predisposing cause - affects every member of the population. In the rainy season the country is one large swamp and anophelins<sup>e</sup> abound everywhere. When a splenic index has been taken it has always been found inordinately high.

#### Diet.

Rice or pap - made from corn - is the staple diet, cooked in oil or merely boiled and flavoured with pepper. There is marked absence of fresh vegetables and in many districts meat is seldom seen. A large quantity of sun-dried fish is used. There is little variety in the diet - and towards the end of the rains there is often insufficiency even.

#### Climate.

Rogers has shown conclusively the close relationship which exists between hot, humid climates and a high leprosy rate. In those dry, arid areas of the

tropics as e.g. the Western Coast of S. America, the Sahara, and German S.W.Africa, with an annual rainfall under 10", the leprosy rates are very low, while in the hot, moist areas of Tropical Central Africa, and French and Dutch Guiana the rates are very high. A striking example is Madagascar where high leprosy rates are found on the wet side of the island, while the number of lepers on the dry half are very few.

Rogers' explanation is that, as the bacillus has not been cultivated with certainty, it presumably lives only for a short time outside the body in the absence of any known animal or insect carrier - so that the absence of leprosy in hot, dry countries may be explained by those conditions being most unfavourable for extra-corporeal survival of the bacillus, while a hot, moist climate is most favourable for their existence, especially if the bacillus gets on to the skin of a neighbour from an infective leper. Again, such climate favours insect life, and the frequency of lesions on the most exposed parts of the body is explained by the fact that there the insects allow lesions through which the bacilli can get into the dermal connective tissue and nerve endings so favourable for their multiplication.

Here the season from June to November is hot and very moist - insect life superabundant and the people living in that close relationship, which permits of easy infection.

Lastly, it must be noted that the general conditions of living have a great effect as a predisposing factor. The more truly civilised a people are, the less likely is leprosy to be a danger; and that in these protectorates the problem will always be great until the people have learned the true value of hygienic conditions.

#### CLINICAL APPEARANCES.

##### Difficulties of diagnosis.

The advanced stages of leprosy of either type present a picture so characteristic of the disease that a mistake in diagnosis is well-nigh impossible; but in the early stages its appearances vary enormously and mistakes are common. Particularly is this so among the dark skinned races. Skin diseases are extremely common here, many of them resemble leprosy in some way or other, and most are extremely resistant to treatment. The early macular appearance of leprosy resembles very closely that of leucoderma but has not the sharply contrasting area of



increased pigmentation at the margin. It must be remembered that in the first stage portions of skin removed for examination may show no bacilli. The circinate appearance seen in the skin in early nerve cases is very similar to tinea - and the two diseases may co-exist. Psoriasis of which a certain number of cases crop up here at times may also mislead. Syphilis, the most protean of all diseases, may often give difficulty, and the Wassermann test is of practically no importance as a means of distinguishing.

The bacillus leprae gains entrance to the body through an abrasion of the epithelium, either to the skin or nasal mucosa. That the primary lesion was nasal was widely held but reports of long series of cases by Gomez Basa and Nicholas at Cullion, by Muir at Calcutta and by others have disproved this contention. In the very few early cases of the disease I have seen here I have in only one been able to demonstrate the bacillus in nasal smear.

The commonest early sign is the appearance of a macular lesion, varying in colour from light fawn to white, level with the skin and with a smooth non-scaly surface and irregular edge. These may appear singly or in groups, with a gradual increase both in size and number. The

lesions are at first bacteriologically negative, but in time become swollen and infiltrated and bacteriologically positive. Following this we get the first appearance of a nodular eruption. A difficulty of diagnosis in the early macular stage is that there is no loss of hair, no interference with the activity of the sweat glands and at first no change in sensibility to touch, heat or cold. In some early cases the inability to distinguish between heat and cold is a valuable aid in diagnosis.

This macular stage may however be preceded by one in which the only visible sign is a shiny appearance of the skin especially the skin of the legs.

Or again the sudden appearance of anaesthesia or of hyperaesthesia may be the first sign of the disease.

In children an infiltrated appearance of the skin on elbow or knee i.e. those parts in young children especially subject to friction and trauma - may first cause one to consider the possibility of leprosy - infection.

As to the position of the primary lesion the findings are the same as in India viz:- the primary lesion appears on those parts of the body most exposed to the

bites of insects, and those parts lain on at night - thus lesions are found in those situations when the bed of a leper is used by a non-leprous individual. These lesions therefore usually appear on cheek, shoulders, buttocks and outer aspect of arm and thigh. On one point they differ here. I have not seen a primary lesion on the feet - the soil here being mostly sandy thus reducing the possibility of finding any. In the rocky parts lesions of the feet are reported to be common.

#### Onset.

The onset of leprosy may be acute and the first symptom the appearance of a macular lesion; but as a rule there is a longer or shorter period marked by symptoms, often very indefinite and easily overlooked. Pyrexial attacks, rheumatic pains, anaemia, intestinal disturbance with vomiting and diarrhoea, attacks of local or general sweating, may persist for a long period - years in many cases - before the characteristic appearances of the disease manifest themselves. As a rule the more acute the onset the more numerous the macules and the more rapid the spread. In cases where the onset is very slow a single macule slowly extending may be the only

sign for years. These macules may be signs of either skin or nerve leprosy.

### SKIN LEPROSY

The general appearances of the skin in the various types of skin leprosy have already been described in the section on "Pathology" and need not be recapitulated here.

The macules become larger in size and in number, and may spread all over the body. There are however several sites usually attacked and others where lesions are seldom seen and then only in very advanced cases. Those parts most exposed to changes in climate - as face and arms and legs here show extensive infection. The lobes of the ears are early attacked and the disease can often be detected there by touch even before it is seen. The scalp is seldom affected (no case of scalp leprosy seen here). It is also reported that the region of the scrotum and penis is very seldom attacked, but I have seen two cases of marked leprosy of the scrotum here - both in very advanced cases - one seen at post-mortem in a case of thirty years duration. When nodular formation takes place the appearance is striking, as we get those masses of leprosy tissue so typical of the disease. As they grow they come

to stand out like plaques of a rubber-like consistency, small at first they become larger than walnuts. This appearance may remain or break down and ulceration supervene followed by cicatrisation, and result in increased deformity. The facies leonina - a mass of raised tuberos masses, with thick heavy lips, often everted eyes, and a skin devoid of hairs, is the typical picture of the face in advanced skin leprosy.

If, on the other hand, the disease remains discrete long enough we get a general thickening of the skin of the face which is then intersected by deep furrows.

The hair of the face and eyebrows - especially on the outer half falls out and there is general loss of hair all over the body, except on the scalp.

The lymph glands may become markedly swollen.

During the course of the disease new nodules will appear - their appearance being often accompanied by a febrile attack.

### EYES

Pinkerton writing from Hawaii says that every patient with leprosy will sooner or later get involvement of the eye. This may be so, but here the number of cases



of visible eye infection is very small, and where this is seen the patient is in an advanced condition, whereas Pinkerton states that the primary corneal change may occur at any stage in the disease. There appears to be more ectro<sup>F</sup>ion - the result of cicatrisation - than real affection of the eye itself.

Infection due to bacilli in the eye may result from spread from adjacent areas by the lymph stream or from some more distant focus by way of the blood. Direct inoculation through the cornea has not been recorded. A superficial involvement of the cornea gives a pannus-like appearance and may interfere with vision if it spreads over the pupil. In the deeper type there may be nodules on the cornea and spread involving the anterior chamber and the iris and tract, causing iritis and iridocyclitis.

The main causes of blindness are said to be (1) Exudative iritis and (2) Corneal changes secondary to invasion from nodules in the ciliary region and exposure keratosis. Internal and external tarsorrhaphy are recommended as a means of preserving the cornea.

#### NOSE.

Nasal infection may occur very early, probably due to inoculation by scratching. The common early site

is at the bony cartilaginous junction. A person with this is a source of great danger to others, without he himself having any knowledge of it.

Later marked ulceration going on to destruction of the septum - as in two cases recently seen - may appear. One of these cases was only able to whisper, as a result of ulceration in the neighbourhood of the vocal cords. Pharyngeal infection is not rare here.

No case of infiltration of the tongue was seen, except a case under treatment where this came on acutely, but subsided after two years.

#### LESIONS of the EXTREMITIES.

Lesions of the arm are seen especially round the elbow and are discrete in type as a rule.

The nails become thinned out and have a wavy appearance - almost as if one piece was plastered on top of the other. In some the nail stands up out of its bed - seen in cases where intercurrent disease has made it impossible for them to tend to themselves.

Lesions round the knee are common; and ulceration of the foot does occur in skin cases - as a result of cracking of the epithelium. Those ulcers lie deep

in the furrows of leprous tissue and are difficult to cure.

Under treatment certain peculiar clinical manifestations are seen in a severe reaction in skin leprosy. In one case this consisted in the rapid appearance of hard nodules all over the extensor surface of the arm - skin sections showed the presence of bacilli - swelling of the hands and feet, followed by a rapid desquamation all over the body and amelioration of the leprous signs.

Blood examination gives no characteristic picture - an eosinophilia is seen, but in a country infested with intestinal worms this is of no value.

Skin Leprosy is of shorter duration than nerve leprosy; death usually being from some intercurrent disease, of which the most important here are diseases of the chest.

#### NERVE LEPROSY.

The onset of this type is as a rule slow and the first symptoms may vary greatly in character. In some cases the first thing noted is a sensation of numbness in the fingers or elsewhere - usually at the distal end, or it

may be a feeling of shooting pains along the course of the nerve. The appearance of a macular stage usually precedes by a longer or shorter period the more distinctive features of the nerve type. The macular lesions are as a rule circinate in appearance with slightly raised edges - often hyperaesthetic and enclosing a decolorised patch which in time becomes anaesthetic. This central area becomes dry from atrophy of the sweat glands and there is also loss of hair over it. This appearance may not remain constant; it may be temporary; or may spread, neighbouring patches co-alescing. The anaesthesia comes in a definite order as a rule - to touch heat, cold and pain.

At or before this time bullae may appear - oftenest on the limbs. These as a rule rupture and leave a decolorised area of skin, or may become ulcerated and cause disfigurement.

A striking symptom mentioned under "Pathology" is the appearance of ~~Ankylosis~~ <sup>Anhydrosis</sup>. This is particularly well seen here. Patients walk in over dusty roads and present a striking picture when examined on arrival.

After a varying period signs of marked implication of the nerves become apparent - severe neu-

ralgic pain along the nerves, which may be extremely tender if compressed, and a hyperaesthesia of the parts they supply. Fusiform swellings of the ulnar nerve above the olecranon - of the radial occasionally on deep pressure into the ~~triceps~~<sup>biceps</sup>, and of the external popliteal behind the head of the fibula--are easily made out. The great auricular nerve, which is often involved, is thickened generally throughout its course - but presents no localised swelling.

As the disease advances the hyperaesthesia disappears to be replaced by anaesthesia, which becomes complete - or anaesthesia may be present from the beginning. This anaesthesia, in the extremities especially, is slowly progressive - the sensation of deep pressure often being retained long after all other sensations have disappeared.

Along with this anaesthesia trophic changes and muscle atrophy take place. In the hand the small interossei are first affected, and at the same time there is a flattening of the thenar and hyper<sup>o</sup>thenar eminences. The muscle fibres in time become replaced by fibrous tissue and contractures result, the 4th and 5th digits usually being first affected, but later on the whole hand giving the characteristic main-en-grippe <sup>ll</sup> appearance. Changes in



the face muscles give the sufferer a peculiar distorted appearance - also characteristic.

Trophic changes cause alteration in the bones. The process may be a slow absorptive one, commencing at the distal end of the phalanges; and showing slow steady shrinkage, but oftener this process is upset by the entrance of sepsis organisms, causing ulceration and necrosis. All the digits may disappear and only repulsive stumps be left. Amputation of these ulcerating fingers has been done here several times without the patient having any sensation at all, the anaesthesia being so complete. This anaesthesia is responsible for the perforating ulcers of fingers and toes so often seen.

The NAILS present a different appearance to that seen in the skin type. They become thickened, and when absorption of bone of the terminal phalanx takes place they curve over the end of the finger giving a claw-like appearance.

#### EYE.

Ectropion is found also in this type as a result of paralysis of the orbicularis palpebrarum. This paralysis also renders the cornea more liable to injury, especially as it develops loss of sensitiveness from involvement of the 5th cranial nerve. Corneal ulcers are report-

ed to be common in this type, but I have seen very very few here.

The skin in advanced nerve leprosy has as a rule a dried up and thinned out appearance, but in some cases appears absolutely normal.

A description has been given only of skin and nerve types. A mixture of these two is very common and presents characteristics of each type.

#### THE STAGES AND PHASES OF LEPROSY.

Our knowledge of these is largely due to the work of Muir - and it is essential to know them and to be able to recognise them clinically in order to treat the disease properly.

The Phases are:-

- (1) "The Quiescent, during which the bacilli may multiply and the lesions extend locally, but during which there are no general toxæmic symptoms."
- (2) "The reactionary or inflammatory, during which the lesions become inflamed and general toxæmic symptoms appear" and
- (3) "The phase of resolution following upon the subsidence of reaction."

The reactions may affect a whole lesion or merely a part of a lesion; and in certain cases a lesion may pass into the resolution phase without any noticeable local inflammation or any toxæmia.

The Stages are:-

- (1) Where the bacilli are few and the lesion limited and it may be difficult or even impossible to find bacilli in portions removed for examination.

This is the stage where treatment is most successful.

- (2) The Active stage with spread of lesions and multiplication of bacilli. This stage shows no retrogression, and is the stage where great care in treatment is necessary, otherwise a marked exacerbation may result.

- (3) The stage of Retrogression, markedly (1) a gradual arrest of reactions or (2) rapid resolution in the skin lesions or (3) rapid Disappearance of the inflammatory points caused by the bacilli set free during reactions - indicating the development of some degree of immunity.

It is in this stage that treatment should be pushed to the limit.

## TREATMENT

The first great essential in treatment is the realization that there is no specific remedy for leprosy i.e. no specific as e.g. quinine is specific for aestivo-autumnal malaria or novarsm<sup>en</sup>obilon for yans<sup>W</sup> or relapsing fever; and that treatment with chaulmoogra oil or its derivatives, causing total disappearance of all signs in early cases, has this beneficial result only when the general health of the patient is very good.

Failure to realize the importance of this treatment of the general health of the patient along with the treatment of the leprous condition must almost inevitably lead to disappointment.

Leprosy and Tuberculosis have many points of resemblance but none greater than their response to a general toning up of the whole system.

Signs of any accompanying disease must be sought for and that disease treated. This is of paramount importance in a colony such as this where syphilis is wide spread, and the greater mass of the people sufferers from intestinal parasites. As an example I cite a case treated for several months two years ago with no visible sign

of improvement or sign of any reaction. The administration of carbon tetrachloride and expulsion of several hundred ankylostome worms was almost immediately followed by signs of rapid improvement in the leprous condition.

The importance of adequate suitable diet must be constantly borne in mind. Here, unfortunately, it is a matter of great difficulty, as often towards the end of the rainy season there is a distinct food shortage. In many parts also there are large tse-tse fly belts, where no meat of any sort is procurable, and dry stinking fish mostly used. Very few vegetables are grown anywhere in the colony.

The disappearance from modern tuberculosis sanatoria of all attempts at gross over-feeding, and the insistence on graduated exercise, has given increasingly good results. This insistence on exercise is equally important in leprosy, and in any segregation camp exercise should have a very important place - otherwise a great deal of the value of modern treatment will be lost.

The general sanitary conditions of their lives and dwellings must be rigorously looked after. Much can be done as in most cases conditions are extremely bad. The huts are made of krinting - i.e. split bamboo inter-





woven - plastered with mud on one or both sides, and surmounted by a low overhanging grass roof. In the plaster of the walls as also in the wooden framework of the rough beds are countless bed bugs and other insects. A fire burns in the centre of the floor rendering the heat intense and the whole atmosphere is foul and in too many cases cleanliness is absent. Living under such conditions makes treatment difficult and recovery doubtful.

The mental outlook towards the disease of these people is again a point of great importance. In the present state of our knowledge no rapid spectacular cure of leprosy is to be expected, and these people possess no great stock of patience. The rapid cure - and visible cure - of surgical conditions appeals to them; but the time is not yet when they will rush for treatment of this disease where at the least months must elapse before a real visible, striking result can be obtained. A further difficulty along these lines lies in two great facts of the disease itself:-

- (1) Leprosy in some proven cases is a self-healing disease.
- (2) In practically all cases periods of quiescence intervene.

Leprosy is an old disease in Africa, and has been treated by bush doctors for centuries. They hail as cure any stoppage in the progress of symptoms; and their patients, unfortunately, are not then likely to seek proper scientific treatment.

#### DRUG TREATMENT.

A host of different drugs have from time to time been advocated as curative agents, only to fall into disrepute after prolonged trial. Possibly they gained a temporary reputation by an improvement in early cases as a result of general treatment or the supervening of a period of quiescence; or, more probably, because they were able to cause a temporary reaction. It is to be noted that some of these - previously given up - and again being used for this very property of causing reaction - but they are used as adjuvants to Chaulmoogra, and not as curative agents per se. It is now generally conceded that the drugs of the chaulmoogra series alone have any true curative action.

Several of the metals, especially mercury and gold, have been used at various times with conflicting results. The beneficial action of gold is again being

advocated at the present moment.

Arsenic was used but its action was in no way specific, but rather a general toning up of the system. Antimony in conjunction with chaulmoogra is still being used.

Iodine was advocated, and its effect in the form of potassium iodide as described recently by Muir will be referred to later.

X Rays were used but without any conclusive result.

Ichthyol used internally and externally also had a vogue for some time, but has largely been given up.

As would be expected much was hoped for the use of serums and vaccines.

The resemblance between the two bacilli caused tubercle vaccine to be tried against *B. leprae*, but it was found devoid of any curative action - although as a diagnostic agent it may yet prove of value.

Dycke made a vaccine called Nastin from acid fast organisms which he thought to be a culture of leprosy bacilli; and Nastin had its vogue, to pass as it gave inconclusive results.

Many vaccines made from supposed lepra cultures

and from the true lepra nodules may, by their power of producing reactions, prove of great help in treatment.

Treatment by Chaulmoogra.

3  
Chaulmoogra oil is derived from the seeds of Taraktagenos Kurgii. Its active principles are also found in certain varieties of hydnocarpus seeds - particularly hydnocarpus wightiana and hydnocarpus anthelmintica.

Its use in the treatment of leprosy is of no recent date, having been employed for many years by the natives of India. To be of any real value if taken orally huge doses are necessary, and its value was nullified in many cases because of its irritant action on the gastric mucous membrane.

Other methods of administration had to be used; and attempts were made to devise methods of giving it subcutaneously or intra-muscularly. Various ways were tried, the trouble in each case being the severe pain caused by the chaulmoogra in the tissue.

In 1914, V.C. <sup>Heiser</sup> Hassa reported good results at the Cullion Colony in the Philippines from the use of Mercado's formula. Here camphorated oil was added to lessen the pain. The formula was Chaulmoogra 6 oz.

Camphorated oil 6 oz.  
Resorcin 4 grm - used as disinfectant.

The next big step was the use by Rogers of the sodium salts of Hydnocarpus and Chaulmoogra oils in aqueous solution. This was given subcutaneously <sup>0.5?</sup> in intra-muscularly at first, but later intravenously by which route no pain at all was caused. The intravenous route had however one disadvantage that in certain cases an inflammatory obliteration of the veins resulted. At the same time Rogers was able to show that these injections produced definite reactions with febrile disturbance, and that local swelling followed by the breaking up and final disappearance of the lepra bacilli.

The next progression was the use of the esters of chaulmoogra oil, and their use gave extremely good results. Later statistics from Cullion concerning nearly 9000 cases proved the greater efficiency of the ester treatment to that formerly used i.e. Mercardo's formula. Attempts were made to fraction the fatty acids with the hope of obtaining a fraction more efficaciousness than the whole fatty acids, but these proved failures.

These esters may be used undiluted, but the addition of an equal quantity of olive oil considerably



reduced the pain caused by their injection. Various antiseptics have now been combined of which creosole (as advocated by Muir) and iodine in 1.3 per cent solution are the best.

More recently it has been shown that, efficacious as the esters undoubtedly are, their action is not so great as that of the sodium salts advocated by Rogers. A new preparation "Alepol" has been made, using the sodium salts of selected fractions of the fatty acids of the *Hydnocarpus Whightiana* oil. This has the added advantage of being perfectly painless whether given subcutaneously, intra muscularly or intravenously. The latest improvement is the preparation by Muir of sodium *Hydnocarpate* from *H. Anthelmintica*, which is non-irritating for intravenous work.

This new preparation "alepol" has the additional virtue of cheapness. This combined with the fact that seeds of *Hydnocarpus Whightiana* are available and can be grown locally, and thus an adequate supply of the esters be obtained, makes the actual supply of the drug no longer a matter of difficulty.

What the real action of chaulmoogra is has been a matter of great debate. It is probably twofold.

Firstly, benefit is derived from the local reaction due to the injection of a highly irritant substance into the tissue; but apart from this it probably has a definite specific action on the bacilli.

A certain amount of caution is necessary in the use of Chaulmoogra. Dosage in the first and third stages of the disease may be increased rapidly, but in the active second stage caution is required to avoid dangerously severe reactions with spread of the lesions. This has been seen here more than once. As regards pain on intra-muscular injection of "Moogrol" 3-4 mims of 1'1000 adrenalin chloride helps greatly to minimise this.

Here it is noted that improvement is apparent only in those cases where a definite reaction occurred. No cases have been under treatment for sufficient time to be said definitely to be cured - and, unfortunately, it has been the more advanced - and thus more difficult to treat - cases that have come forward for treatment.

The latest advance in treatment is the combination of Iodides with the Hydnocarpate. The use of Iodides is not of recent date, but because of the severe reaction it was considered that their action was definitely harmful. In 1908 it was recorded by Marchoux and

Bournet that, "during the reactions produced by iodides, both the lepra bacilli discharged from the nose, and those in nodules, which softened or suppurated as a result of the reactions, showed extensive breaking up and loss of acid fastness." Now such also happens in nodules showing similar reaction after intravenous sodium hydrocarbate.

Muir in Calcutta has given iodides extensive trial and reports marked success. He has discovered that tartar emetic given intravenously will control the prolonged febrile reaction, and thus the drug can be given with advantage. As a result of his researches he is able to postulate that when large doses of iodides can be taken repeatedly without reaction, a cure has very probably resulted.

The use of iodides however should be confined to skilled leprologists at the present time.

Before I had read of Muir's work and of the dosage he advocates, I had tried iodides in quantities of gr  $\bar{x}$ . *tdp* on a case which showed no visible improvement. Within a week he returned, with face markedly swollen, and on the extensor surfaces of both forearms were dozens of small nodules; the arms were swollen and the skin

glossy. Scraping of these nodules showed *B. Leprae*. There was severe febrile reaction, and in a few days the feet were swollen and the patient had to be confined to bed. In about a fortnight the temperature had dropped and a desquamation of the skin of arms, legs and body, in process. Following this there was marked improvement in his condition.

Whenever a smart reaction is obtained from any substance, it is upheld by some workers that that substance is producing a beneficial result. Various protein bodies have been tried. Dyce-Sharp working from Nigeria in January 1928 reports his results from injection of tinned milk - this being used as being probably the only protein easily obtainable on the West Coast of Africa (Fresh milk is seldom procurable and "fresh eggs" as a rule are undoubtedly not fresh). The immediate result of injection was a giddiness followed by respiratory dictones and precordial pain. Soon intense pain in all joints occurred attended by profuse sweating. When this subsided the patient could go home. That night a secondary reaction with rise of temperature to 103°-104° F. took place; and this might last several days. He states that the improvement in the patients so treated was very marked,

perforating ulcers of the sole of the feet clearing up in three days and nodules disappearing in ten days.

No history of any of these cases, after five months, is obtainable, - so that, although this may point out another avenue of approach for improved treatment, it appears much too dangerous for general application.

With all this knowledge of treatment at our disposal, it would appear an easy matter to efficiently tackle the leper problem. But it is not so simple. It is utterly impossible for a single doctor to adequately <sup>to</sup> treat lepers scattered over a large tract of country. Again note that treatment of the obvious cases of the disease will do little to prevent its spread. It is the early not-easily-diagnosable case that is important, those where spread of infection from nasal mucosa is taking place.

It has been noted how the decline of leprosy in the Middle Ages was largely the result of the rigorous segregation laws enacted at that time. Without some form of segregation leprosy will not be stamped out here. Before segregation be done however, thorough examination of all cases must be made by a competent leprologist, so that a separation be made between infective and non-infective



lepers.

In too many asylums the greater number of patients have been old non-infective cases, and thus has disrepute been brought on a very sane system. The value of segregation has been proved many times. Two concrete examples only need be given. In Jamaica following on the passing of a law for compulsory segregation the total number of lepers dropped by 52% in twenty-five years, while a similar decree in British Guiana brought about a reduction of 42% in thirty years.

Segregation is of course expensive for a small colony. In Oceania, Fiji has compulsory segregation, and has voluntarily offered to accommodate the lepers of Western Samoa. A similar state of affairs might exist between this colony and Sierra Leone. The time for compulsory segregation may come, or improved methods of treatment render it unnecessary, but at present it would be a hopeless failure, - as cases would undoubtedly be hidden. From Nigeria come reports that segregation "is a counsel of perfection unattainable now and for long years to come." Not only would cases be hidden here, under the circumstances, but the people being clannish, the result would be that not only lepers, but their entire families would go across

the border.

A tremendous amount of patient teaching must be done, and the responsibility put on the people themselves. I have advocated (vide Med. and Sanitary Report pp 66-75) that as a beginning each village tackle its own leper problem, segregate but not ostracise its lepers. Huts apart from the rest be built and it <sup>should</sup> be seen clearly that the lepers eat and sleep by themselves, and that no child have any communication with any leper.

Following this there would be a grouping of villages, whereby they form a small leper colony for the district - kept up absolutely by that district. This would involve little hardship on the villages concerned, and would be no drain on an exchequer none too full. These small colonies would have their own land on which the lepers could work, helping towards their own support, and also helping greatly in their own treatment. All healthy children would be rigorously prohibited from entry into these settlements. The question of a healthy woman desiring to be with her husband is different. It might, at first at any rate, be allowed - since the risk of infection is so greatly reduced with age.

These settlements could be visited regularly,

and people discharged when no longer infective. Situated close to them would be dispensaries for treatment of suitable cases, run by native dispensers, who would be taught to give hypodermic injections.

The patients would be far more interested in their treatment, as they would see improvement all round them, note it in themselves and seek to help in it. The only chance of doing any real good here as elsewhere, is by obtaining and fostering active co-operation between patient and helper.

Two final points of great difficulty must be mentioned in conclusion.

The periodic regular examination of families from whom a leper has come is a matter of great difficulty because (1) it is impossible to guarantee being in different areas at any regular time and (2) because of the constant movement of the people. The other point is even more difficult i.e. the efficient examination of all immigrants to prevent further importation of the disease, because of the extent of the boundary.

#### SUMMARY.

Central Africa presents the biggest leprosy problem which the Empire has to face and must face.

The extent of the disease is not yet realised nor will it be so until thorough and systematic search be made in the villages in all the colonies. A superficial count is so inaccurate and misleading as to be absolutely dangerous.

Vast improvement in treatment has been made in the last decade and if full advantage of this be taken the final stamping out of leprosy is only a matter of time.

Supplies of the seeds of *Hydnocarpus Wightiana* can now be readily procured and grow well in these tropical possessions. In five years after planting an adequate supply of the drug would exist locally.

The contagiousness of leprosy is not matter of conjecture, but of fact, and if this be realised, then a proper combating of the scourge is only possible if some form of segregation be used.

Compulsory segregation at present would do no good but rather infinite harm.

Segregation must be undertaken voluntarily by the natives, commencing firstly with villages and then with groups of villages. The natives would consent to do this, as they do realise the danger of the disease and understand its spread.

BIBLIOGRAPHY.

1. Allbutt's System of Medicine.
2. Castellani & Chalmers
3. Cook "The Leprosy Problem". Medical Journal of Australia, Dec. 11th. 1926.
4. Cooke "Leprosy in the British Mandated Territory of Togoland". Transactions of The Royal Society of Tropical Medicine and Hygiene, Vol. xxi. N<sup>o</sup>. 4. January 1928.
5. Dyce-Sharp "A New Treatment for The Leper" Transactions of The Royal Society of Tropical Medicine and Hygiene, Vol. xxi N<sup>o</sup> 4. January 1928.
6. Gomez, Bassa & Nicholas "Early Lesions and The Development and Incidence of Leprosy in the Children of Lepers". Philippine Journal of Science - Sept. 1922.
7. Kedrowski "The Microbiology of Leprosy Bacillus". The Journal of Tropical Medicine and Hygiene, January 16th. 1926.
8. Kerr. Isobel "The Chaulmoogra Treatment of Leprosy." Lancet August 22nd, 1925.



9. Manson-Bahr "Tropical Diseases". Chapters on Leprosy.
10. MacCromack "Jacobi's Atlas of Dermochromes" Vol.2. "Section on Leprosy" 1927.
11. MacLeod "Contact Cases of Leprosy in the "British Isles" - British Medical Journal, 1925.
12. Molesworth "The Leprosy Problem". Medical Journal of Australia. Sept.18th 1926.
13. Muir "Leprosy - A Self Healing Disease" Lancet. February 9th. 1924.
14. Muir "The Predisposing Causes of Leprosy" Lancet. January 24th. 1925.
15. Muir "Report on Visit to Leper Island of "Culion and on Anti-Leprosy Work "in Phillipine Islands". India Medical Gazette.
16. Osler "Principles and Practice of "Medicine"- Chapter on Leprosy.
17. Pinkerton "Leprosy of The Eye - An Analysis "of Records of 512 Cases of Leprosy "in Hawaiian Islands". Archives of Ophthalmology. January 1927.
18. Rogers "The Croonian Lectures on Leprosy "Researches". Annals of Tropical Medicine and Parasitology. April 30th. 1924.

19. Rogers "The Curative Action of Hydro-  
"carpates and Iodides in Leprosy".  
Lancet, January 14th. 1928.
20. Rogers "Recent Advances in Tropical  
"Medicine". Chapter on Leprosy.  
1928.
21. Rogers & Muir "Leprosy". 1925.
22. Muir, MacLeod, Sequeira, "Discussions On The Treatment of  
Pernet, Low, Henry, "Leprosy". Proceedings of The  
Kerr, Wade, Lara, Royal Society of Medicine, 1927,  
Cochrane, Travers, Vol. XX (Sections of Tropical  
Hassa, Hall and Rogers. Diseases and Parasitology, Derma-  
tology and Therapeutics and  
Pharmacology, pp. 121-149).
23. Wilson "Leprosy in Korea" - Study of 938  
cases - Journal of American  
Medical Association, 1926.
24. --- "Memorandum On the Prevalence of  
"and Prophylaxis against Leprosy in  
"The British Empire - Based on Re-  
"plies to The Questionnaire of The  
"British Empire Leprosy Relief  
"Association, with Suggestions for  
"Dealing with The Problem."
25. --- "The Various Reports of The British  
"Empire Leprosy Relief Associal.
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