This belief in the contagious nature of leprosy still remains in many countries, and lepers are carefully isolated in Norway. But the greatest differences of opinion prevail on this question amongst medical men.

In the Report of the Royal College of Physicians in 1860, the Commission state decidedly "that the weight and value of the evidence they furnish is greatly in favour of the non-contagiousness of leprosy" (p. 7); and again: "The all but unanimous conviction of the most experienced observers in different parts of the world is quite opposed to the belief that leprosy is contagious or communicable by proximity or contact with the disease." Just as we shall see presently in the case of phthisis, they quote the experience of hospitals and the immunity of the attendants in leper asylums.

In the discussion on this question at the International Congress at Copenhagen, in 1884, the two chief speakers—Dr. Zambuco, of Constantinople, and Dr. Hansen, of Christiania—took entirely opposite views; the former maintaining that leprosy is not contagious, but is hereditary, and the latter that it is contagious, and not hereditary. Dr. Danielssen, the father of research on the subject, also takes the non-contagious view, and says: "According to my observation, heredity is sufficiently proved, whilst I have not found one single well-proved case of infection."

VII. With regard to heredity, Dr. Hansen states that of 160 Norwegian lepers who had settled in the States of Wisconsin, Minnesota, and Dakota, none of the offspring, in some cases as far as the great, great grandchildren, have shown signs of the disease. He points out that there could have been no question of heredity in the case of the great outbreak of the disease in Hawaii. A recent writer on the subject, Dr. Albert Neisser, also considers that leprosy is not an hereditary disease, though he acknowledges that the susceptibility of the individual appears to be of more importance than in any other bacterial disease.

It is not surprising that with all these points of similarity, the question should be asked, as it is by Danielssen, whether "Leprosy is not itself a tuberculous disease?" The circumstance that the bacilli of tubercle are larger and less disseminated in the tissues, ought not to be invoked as a sufficient diagnostic characteristic, nor can we draw any more conclusive argument from the mode of development of these bacteria. "The manner in which infection in leprosy takes place, the long and silent period of incubation which characterises it, recalls in the highest degree the infection of tubercle."¹⁷

Dr. Danielssen further affirms that tubercular disease is more common amongst lepers than among the general population, but even if this were true, the fact might fairly be ascribed to the general low state of health of lepers, making them more vulnerable to the attacks of the tubercle bacillus. This statement is, however, contradicted by Dr. A. Hansen, who, as I am informed by Dr. Stabell, of Bergen, in answer to a question of mine, states that "Tuberculosis, which was formerly very prevalent in the leper hospitals of Norway (and no doubt the patients were infected in the Hospitals) is now more seldom seen, because the hospitals are now not so crowded as before. Tuberculosis, indeed, attacks lepers as it does other people, but the acute form is not more frequently seen amongst phthisical lepers than amongst other persons affected with phthisis."

We cannot therefore allow ourselves to be led into affirming the identity of the two affections simply by such considerations as these. The strong tendency towards certain tissues as the favourite haunts of the two organisms, the fact that the lepra bacillus cannot be cultivated, the difference in their rate of progress, and the fact that though tubercle may attack persons affected by leprosy and *vice versa*, there is no special proclivity to this interchange, shows that though they are probably closely related, there is no identity to be discovered between them. Lastly, the ready inoculability of tubercle, and its facility of cultivation, form a marked contrast to leprosy.

Certain practical conclusions may perhaps be drawn from this review of the pathology and affinities of tubercle which may assist us when we come to deal with the question of its preventability:—

1. That the bacillus which provokes the disease comes most

August, 1889, p. 143, quoted from Grosley's *History of Troyes*. In the 14th century they were supposed to be associated with the Jews in a horrible plot to poison all the springs, wells, and rivers with their blood; and in 1321 a fearful massacre of them took place in France. At Chinon 160 were burnt in one day, and in Périgord and Languedoc, in a plague panic, fires were lighted everywhere, and lepers and Jews heaped thereon.

¹⁷ *Loc. cit.*, p. 16.

frequently from outside the body, and is conveyed by means of the atmosphere.

2. That it requires a certain length of time and certain narrow limits of temperature within which it can develop.

3. That when it is entrenched within its encapsulating tubercle it is placed at a distance both from the currents of blood in the vessels and from air entering through the lungs.

4. That its life in the body is usually a short one, and that the living tissues are antagonistic to it, or, to use Dr. Moxon's words, that "the life of the bacillar parasite is difficult—easily discouraged by unfavourable circumstances—like an aphid by an easterly wind." And

5. That the resemblances which it bears to the bacillus of leprosy may encourage us to hope that, as this latter disease has been banished from our country, so also may the different forms of tuberculosis ultimately disappear.

AN ADDRESS

ON

ENDEMIC DISEASE IN BRITISH GUIANA

AND ON

CERTAIN RACIAL SUSCEPTIBILITIES.

Delivered at the Annual Meeting of the British Guiana Branch of the British Medical Association.

By ROBERT GRIEVE, M.D.,

Surgeon-General British Guiana; President of the Branch.

Improved Condition of Coolie Immigrants.—Twenty years ago it was said authoritatively that the Indian immigrants arriving in this colony during the period of their acclimatisation were peculiarly liable to suffer from ulcers and chronic dysentery. It is most satisfactory to be able to note that with regard to the prevalence of the first of these diseases of late years there has been a marked and unmistakable improvement. Some of you may even more clearly than I do remember the number of ulcers in many estates' hospitals ready at any time to take on malignant action, and which were a continual source of trouble and anxiety to all connected with them. An appeal to figures proves the progress that has been made. In the year 1875 ulcers formed 13 per cent. of the admissions to the estates' hospitals; in 1888 the percentage of ulcer cases amongst the admissions was 4—a very large reduction. That the change may be taken as an indication of an improvement in the sanitary condition of the coolie seems to me undoubted. It shows an advance in sanitation, using the term in its most comprehensive signification as including housing, feeding, and conditions of work. It is a fact which yields much encouragement. I specially mention food, because it seems to me that better feeding has had a special and direct bearing on the question, for the ulcers partook of the scorbutic character. Whilst, as now stated, improved sanitary surroundings are entitled to perhaps the greater portion of the credit, still I think for some share of the progress we are indebted in late years to improved means of treatment by the adoption of antiseptic surgery and its attendant cleanliness. Ulcers are now lessened in number because they are checked at the outset. I am sorry to say that the other class of diseases before mentioned—namely, bowel affections—have not diminished in frequency as have ulcers. The percentage amongst the admissions to hospitals due to these diseases remains practically the same now as it was in 1875. To my mind this is an indication that improvement of water supply has not kept pace with the advance in other matters of sanitation. During the last year or two in many districts a supply of drinking water of increased purity has been available, and though the effects of the change are as yet perhaps not very marked, they may become so in a short time.

Phthisis.—I pass now to another disease, phthisis, of which the increase in this colony is undoubted, and, so far, progressive. This increase has received notice more than once, and very fully, in a paper by Dr. Ferguson in the hospital reports. I wish to direct attention specially to a phase of its local development, which is also noticed by Dr. Ferguson—the different clinical history which the disease presents as it occurs in the two races form-

ing the largest proportion of our very mixed populations. Amongst the blacks the disease, considered from the clinical side, is generally of the rapid acute form known as "galloping consumption"; or, looking at it from a pathological point of view, it presents in that race the form of tubercular caseous pneumonia. The phthisical East Indian, on the other hand, presents the clinical characteristics of the slower and more variable forms, or, speaking pathologically, suffers most commonly from tubercular interstitial pneumonia or peri-bronchitis. Now, why this difference? We have as yet no reason to doubt that the bacillus of the disease is in both instances alike, but this is a matter which it would be interesting to have settled by direct observation. The conditions under which the individuals live here are in both races practically the same. The difference, therefore, does not appear to arise from any factor acting immediately and directly on the individuals, but to be connected with something more remote. To me there seems but one solution of the matter; the peculiarity depends upon inherited differences, in fact, on racial characteristics. Now, as racial characteristics are in truth only the inherited results of influences exerted on individuals acting through an unknown number of generations, by investigation into the conditions of individual existence under which the various races have been brought to their present condition, we may arrive at some notion as to the causes of the diversity in liability of the races to various diseases, and from that obtain a clue to the varying tendency to disease in individuals of the same race which is universally recognised, if not understood—a very speculative field of inquiry it may be said. I admit this; but it is one that might, I think, be worked to advantage, at least to the extent of ascertaining the facts with regard to the differing proclivities to disease evidenced among the various races inhabiting the colony. The broad fact stands out clear and distinct, that though living under identical climatic and sanitary conditions, each of the races comprised in the population of the colony is more specially prone to peculiar diseases. To emphasise this fact is of value in this time of devotion to the study of the other element in so many complaints, the organisms, which we have a right to assume are the exciting causes. We think so much of the bacilli that we are apt to think too little of the soil in which they grow. But that the factor is not even of secondary importance I think every observer in these regions will admit. The special local condition which has contributed to the continuous spread of consumption already noticed, is, in my opinion, the great and ever-increasing overcrowding in the dwellings of the labouring classes. Overcrowding is but a synonymous term for deficient ventilation. The number of people who can be safely housed in a room, in this climate, entirely depends upon the amount of air which is allowed to pass through it. When, as is too often the case, every chink through which air might enter is closed, and the sleeping accommodation utilized is limited only by the number that can find room to lie on the floor, then the conditions which favour the multiplication of the tubercle bacillus are to be found in fullest force. In Europe the open-air treatment of phthisis is coming into vogue, and it is claimed with success. Here I have pleaded for the adoption of the open-air method of prevention, and I trust that such pleading has not been altogether in vain.

Pneumonia.—Acute inflammation of the lung tissue is far from uncommon in this colony, but it appears certain that this seldom comes from exposure to cold or climatic changes, it is either of malarial or septic origin. The history of the outbreaks of the disease in various localities points to these being dependent, in many instances, for their extension upon communication from person to person; they are local epidemics of contagious pneumonia. Many of you have no doubt seen examples of such outbreaks, mysterious sometimes as to the origin of the first case, but given one case the spread from individual to individual by direct communication is to be discerned plainly enough. Detailed accounts of such occurrences are of the greatest value, and with you it rests to put them on record as they fall under your notice.

Fever.—At least a full half of the cases treated in the estates' hospitals of the colony are classed as fever. Better and more exact information on the fevers of the colony would be of immense value both from the curative and preventive aspects. With the ever present malaria looming large in view, the natural tendency is to ascribe to its action, direct or remote, every febrile symptom, even when unmistakable organic change may be connected with it. This is the more excusable as the range of temperature bears in almost every case more or less the impress of the malarious influence. But are we to assume from this that all

the fevers of the colony are solely of malarial origin? For one, I am not quite ready to agree to any such assumption. I believe that specific fever organisms of one kind or another, apart from the malarial, live and thrive amongst our population, even though when the disease depending on their presence manifests itself, it bears an outward resemblance in one symptom at least to malarial fever. The pyrexia often varies in its progress from day to day, almost certainly to some extent following the course of the pyrexia of a fever of malarious origin. No one, I presume, questions the fact that the great bulk of the fevers of this colony are undoubtedly malarial fevers, either of the intermittent or remittent type, both equally well known and recognisable; still it is equally true that fevers which fall readily in neither class are constantly met in practice. From my own observation as far as it has gone, I have received the impression that amongst the fevers in this doubtful class there are some which are instances of diseases of a kind completely undescribed, and therefore in that sense new. Again, as hinted before, we may have amongst them examples of the action of fever poisons of kinds familiar in other regions, but when disease results from such causes in this colony, the local conditions impress it with some peculiar and so far undescribed characteristics.

Enteric Fever.—A subject so wide as this is manifestly too great to be even touched on in its full extent in an address like the present. What I have to say just now will be limited to one phase of it, and this may be perhaps most easily and clearly brought into view by putting it in the form of a question. Does true typhoid or enteric fever ever arise in the colony? We know that the range of this disease is most extensive; certainly it has now been shown to prevail at times almost in every part of the globe in which Europeans reside in numbers, and how much of the earth's surface is comprised by this definition? It has been recorded as having prevailed in countries near to this colony, and having climatic conditions almost identical with it. Yet there is not on record a history of a clear and unmistakable case of specific enteric fever of local origin. There is *prima facie* no reason in our material condition for this exemption, though there are certain facts which would go far to explain the absence of the occurrence of anything like epidemics of the disease. On my part, I believe that investigation would show that typhoid is one of the diseases in regard to which the races of men vary much in their susceptibility to its influence. It is pre-eminently a disease of white races, and here we have no white troops or large body of young Europeans who, in all tropical parts, supply the soil in which the poison operates. For some years past I have been watching for a case of undoubted typhoid, and except in one instance, in which the disease was contracted in the United States, I have not been satisfied of its existence. In the early days of my professional life, now some thirty years ago, I had plenty of opportunities of becoming acquainted intimately enough with the two forms of fever, typhus and typhoid, then differentiated only at a comparatively recent period, for in the Scotch hospitals in which in these years I was resident, there were plenty of both kinds to be seen. Since these days, happily, I have not had any opportunity of renewing practical acquaintance with typhus, but this has not been the case respecting typhoid. In the course of a rather varied professional experience before coming to the colony, I met the disease under various circumstances, but not infrequently; so coming here, I was quite ready to recognise the complaint if its well-known features had come under my notice, but this has not occurred. Here I may explain that I look upon a disease like typhoid as a well-defined and recognisable entity. I am not inclined to accept any one symptom separately as constituting the disease, nor am I more inclined to accept a single pathological fact divested of all connection with symptoms or history as constituting the disease. Ulceration of some portion of the intestine is a pathological fact, displayed very commonly on the *post-mortem* table of the Colonial Hospital. It may be at times that the ulceration is confined to Peyer's patches, and may appear like the ulceration of typhoid, but this fact, divorced from all connection with a history of the case during life which in the least resemble the history of a case of typhoid, is not to my mind conclusive evidence of the presence of the disease.

Intestinal Ulceration.—In this we come upon a fact opening out another field of observation, namely, the investigation of the causes of the extensive ulceration of the intestines, which undoubtedly are a pathological condition much more common here than in England.

Bright's Disease.—Before closing there is one other disease

which I should like to mention, namely, Bright's disease. Of its great prevalence in this country there is now no doubt; but, although this is well recognised, there has not as yet been much done in the way of describing the clinical or pathological varieties which are met with locally. Such an investigation is necessarily the foundation upon which any solid work in connection with the elucidation of its etiology or with its treatment must rest, and I trust it may be taken up in earnest by some of our members.

Intestinal Parasites.—There is only one other matter to which I may refer and then I am done, namely, the question of local intestinal parasites and their influence upon health. The identification of the ancylostomum was made some time ago by our colleague, Dr. Ozzard, and we look for further information on it from the same quarter; but there are many others which are plentifully found, and we stand much in need of these being carefully catalogued and described.

I have thus in a most discursive way covered a considerable space; but I have only scratched the surface of the soil which lies open for cultivation with the certainty of large returns to the diligent worker. Trusting that such scratchings may give some indication for the deeper ploughing to follow, I bring them before you, and I feel confident that competent labourers to undertake the task will not be wanting.

A CASE OF DILATATION OF THE STOMACH ACCOMPANIED BY THE ERUCTION OF INFLAMMABLE GAS.¹

By JAMES McNAUGHT, M.D., M.R.C.S.,
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THE case I am about to relate occurred in a factory operative 24 years of age. He is a tall and rather spare man, somewhat pale, but not anæmic. He gives a history of stomach trouble dating back four and a half years. At the beginning of the time he suffered chiefly from pain after eating, but for the last three years his greatest complaint has been of acidity and flatulency. He has occasionally but only rarely vomited, mostly after dinner, but never at any time has he brought up any blood, nor such copious quantities as would point to ectasia. During the last two years he has suffered most severely from flatulency and in connection with this the most remarkable feature of the case made itself evident. His work requires him to rise early, and on one occasion after striking a match to see the time, and when holding it near his mouth, an eruption of gas from the stomach took place. To his consternation the gas took fire, burned his face and lips considerably, and set fire to his moustache. On a subsequent occasion a similar accident happened. At the time I saw him flatulency was extreme and persistent from one meal to another, and he was also much troubled with rancid and sour eructations. His appetite was good, tongue broad and pale, and bowels regular. In examining the abdomen the subcutaneous fat was noticed to be less than normal; there was a deep depression running across the abdomen a little above the umbilicus, but below this there was evident distension and marked tympanites. The lower line of stomach dulness was about three inches below the navel on the left side, and from this point it extended in a curved line to the right, where it reached the level of the umbilicus. A semilunar area of dulness showed the presence of a considerable quantity of food in the stomach, and above this a highly tympanitic note was given extending up into the left hypochondrium. Occasionally a splashing sound could be well elicited, but on other days it was absent. There was a little, but not marked, tenderness midway between the umbilicus and the ensiform cartilage. It was evident from these signs that the stomach was dilated. The stomach tube was used on several occasions about five hours after meals, and quantities of fluid and *débris* of food varying from one to two pints or more obtained. At first it consisted of a soupy matter smelling exactly like sour yeast, and when it was allowed to stand, a layer of frothy stuff half an inch thick, like dirty yeast, formed on the top. This was full of bubbles of gas which could be seen forming and bursting as it stood in the vessel, while below there was a considerable quantity of clear fluid through which gas bubbles could be seen rising, and at the bottom there settled a sedi-

ment of grumous remains of food in a very fine state of division, resembling the sediment from pea soup. The fluid after filtration gave an acidity equal to 3.2 grammes of caustic soda per litre, and it was found to contain 0.55 gramme per litre of volatile acid, and 0.18 grammes of lactic acid per litre, the residue, equal to 2.4 grammes of caustic soda, being due to mineral acid or acid salts. A strong reaction was obtained with methyl blue and congo red.

The microscope showed enormous quantities of yeast and some sarcinae, with various remains of food. At first I was unable to detect any bacteria, but subsequent more careful examination of the clear fluid showed their presence in great numbers. The fluid extracted from the stomach a week later gave an acidity equal to 2.44 grammes of caustic soda per litre, and of this 0.26 gramme was due to volatile acid, and 0.4 gramme to lactic acid. One hundred and fifty cubic centimetres were put in a flask connected with a gas-collecting apparatus, and kept at a temperature of 100° F. In a few hours 40 cubic centimetres of gas came off, and when a light was applied it exploded with a loud report and burnt with a blue flame. Analysis of a portion of the gas subsequently collected, which I owe to the kindness of my friend Dr. Bailey, of Owens College, Manchester, gave

CO₂ — 56 per cent.

H — 28 "

CH₄ — 6.8 "

and residual air 9.2 "

That the inflammable gas existed in the stomach itself was put beyond question by pouring water into it in a thin stream through a tube and igniting the gas as it issued therefrom owing to the displacement. Only one experiment of this kind was made, as the result of the first was to produce a flame of dimensions alarming to both the patient and myself. The acidity of the stomach contents was carefully tested on many subsequent occasions, the details of which it would be tedious to give here. Suffice it to say that it varied from 2.4 grammes of caustic soda per litre to as high as 4.2 grammes. The volatile acid and lactic acid were proportionately greater when the acidity was less. Thus I find when the acidity was 4.1 grammes per litre the lactic acid was 0.3 gramme and the volatile acid 0.24, but when the total acidity was 2.55 grammes of caustic soda per litre the volatile acid was 0.72 gramme and the lactic acid 0.18 gramme. No doubt this depended partly on the food taken, but chiefly on the fact that when large quantities of HCl were present the formation of lactic acid and butyric acid by fermentation was hindered. I was at some pains to make certain the nature of the acid, because at first sight it seemed impossible that fermentation could take place in a medium containing so large a quantity of mineral acid. I found, however, that the quantity of mineral acid was never appreciably less than 2 per mille and generally much higher. The influence of the acid on the fermentation I shall return to later.

I had evidently to do with a case of stenosis of the pylorus and subsequent ectasia. The treatment was directed to removing the residuum of food from the stomach by means of the tube, once daily at least, and administering various antiseptic agents. At first his diet was limited to milk and Benger's food, fats and vegetables being excluded, and a mixture containing bismuth, carbonate of soda, and magnesium and carbolic acid in chloroform water was given him. Under this treatment he improved very much. The quantity of matter remaining in his stomach five or six hours after a meal was lessened to about half a pint, and the quantity of yeast steadily diminished till none was recognisable with the naked eye, though it was always found after cultivation. Examination of the remains of food showed that large quantities of starch grains were always left, and when he had, contrary to orders, partaken of flesh or egg, that no trace of these could be found. This diet was therefore altered; the Benger's food was stopped, lean minced meat with very little bread was given at dinner, eggs beaten up in milk or boiled were taken at breakfast or tea, and no fat or vegetables allowed. I next directed my attention to the question of preventing the fermentation by drugs. With the view of determining the agent which, while innocuous to the patient, was most effectual for that purpose, I made a number of experiments by adding definite quantities of salol, Sanitas fluid, carbolic acid, salicylate of soda, salufer and aqua chlori to the contents of the stomach placed in bottles connected with a gas-collecting apparatus and kept at a suitable temperature. A control bottle was used in every case. I found that half an ounce of Sanitas fluid to 8 ounces was useless and aq. chlori, the same (10 m to the ounce), salol in quantities of 4 grains to the ounce, and carbolic acid 3j to 3viij were only par-

¹ Presented in the Section of Medicine at the Annual Meeting of the British Medical Association held in Leeds.