cases were also at first taken for leprosy. Ehlers⁵ and Neumann,⁶ who investigated the matter, found it was not leprosy but the symmetrical affection aforementioned.

That such a confusion should have existed is another proof of the view that a variety of skin affections were in years gone by included under leprosy. But that is another story.

Leprosy—that is, leprous nodules and infiltrations of the soles and palms—is rare, and, involving the latter areas alone, unheard of. Rille⁷ has recently described leprous lesions of the soles, but the patient suffered from nodular leprosy elsewhere (face, limbs) before the soles became involved.

BIBLIOGRAPHY.

¹ Papers relating to the Treatment of Leprosy in India from 1887-95. Calcutta. 1896. P. 27. ² *Natural History of Selborne*. By Gilbert White. Letter xxxvii, dated Selborne, January 8th, 1778. (Barrington Series). ³ Azua, Un Cas de Kératodermie Symétrique Congénitale et Héritaire, *Journ. des Mal. Cut. et Syph.* 1892. P. 101. Cited by Dubreuilh in *La Pratique Dermatologique*. Vol. ii. 1901. P. 934. ⁴ H. Radcliffe Crocker, *Diseases of Skin*. Second edition. 1893. P. 365. ⁵ Ehlers, Mal de Meleda. *Ann de Derm.* 1897. P. 657. Cited by Dubreuilh in *op. cit. supra*. P. 936. ⁶ Neumann, Ueber Keratoma hereditarium (Mal de Meleda), *Arch. für Derm. u. Syph.* xlii, 1898. P. 163. Cited by Dubreuilh, *loc. cit. supra*. ⁷ Rille, Ein Fall von Lepra tuberosa mit Lokalisation an den Fusssohlen. *Lepra* (Bibliotheca Internat.) Vol. ii. Fasc. i. P. 7, with plate and fig.

BLACKWATER FEVER.

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IN the BRITISH MEDICAL JOURNAL of September 14th, 1901, Surgeon-Major Kleine publishes, on behalf of Dr. Koch, an interesting series of cases of blackwater fever. He considers that the history of these cases favours Dr. Koch's theory that the haemoglobinuria which at times complicates malaria is generally if not always due to the action of quinine on corpuscles damaged by malaria. From my point of view the detailed history of these patients is defective, for there is not sufficient information given as to how they spent the time immediately preceding the haemoglobinuric attack. In 8 out of 15 cases (namely, Nos. 2, 3, 7, 8, 9, 10, 12, 14) this point is incidentally mentioned—it is either stated definitely or it can be assumed from the context that the patients had not been in bed until the onset of the haemoglobinuria. In the remaining 7 cases nothing is said definitely on the subject, though the context might lead us to suppose that they were similarly circumstanced. Surgeon-Major Kleine further quotes the fact that in the 19 cases reported in German East Africa during 1899 and 1900, "almost always it was expressly remarked that the temperature was not febrile when the remedy was taken." In these cases, therefore, we can safely assume that the patients were probably up and about, although the administration of quinine shows that at the time some prodromal symptoms or feelings of malaria must have existed. I would point out in passing that this only illustrates a very common occurrence, and one which frequently leads to error in diagnosis. This is that the temperature very often remains normal, or even subnormal, at a time when the patient's symptoms and the state of his blood would point to the fact that a malarial attack has already begun.

It may be observed that every one of these cases of both series, in which the previous history is given, favours the view which I expressed in the *Journal of Tropical Medicine*, April 15th, 1901, namely, that blackwater fever is due to a chill acting at a certain stage of the development of the parasite, in a patient predisposed by acute or chronic malarial infection. I offered a conjecture—which may or may not be right—as to the manner in which the chill acted.

In my opinion the important point is the practical one, that with malarial patients, especially those who have been much exposed to infection, the most scrupulous care should be taken to avoid chill during the apyretic stages. Acting on this assumption, an old malarial patient should get to bed directly he feels the first symptoms of a malarial attack, even though the temperature be yet normal, or only very little above normal. Similarly, after an attack he should be kept in bed until the temperature has been normal for at least twenty-four hours. This simple rule would, I am firmly convinced, prevent many, if not all, cases of haemoglobinuria, and yet, how many patients in the tropics would submit to such discipline? Personally, in dealing with myself and my

patients, I make this a standing rule to be broken only in circumstances of extreme urgency, and it has never been my lot to see a case of blackwater fever develop when a malarial attack was treated in this manner. This in spite of the fact that during the past ten years I have, I suppose, administered as much quinine as Dr. Koch has ever done in his life. How does Dr. Koch explain an experience of this kind?

The whole theory of the influence of quinine in causing haemoglobinuria rests on the fact that in the majority of cases there is a history of quinine having been taken; but this fact proves nothing if we remember that in tropical countries practically everybody takes quinine. To him who dwells in the tropics the slightest feeling of *malaise*, or being out of sorts, at once suggests the idea of fever, and he takes quinine. It is not, therefore, a matter for surprise that in so many cases of blackwater fever there is a history of quinine. It might be urged just as logically that quinine is the cause of every other disease occurring in the tropics, in which the prodromata in any way resemble those of malaria. Take influenza, for instance. If this occurred much in tropical countries the doctor would invariably find, when he was called in, that the patient had taken quinine under the idea that he had fever. If pneumonia followed it could be stated just as logically that it was due to the fact that quinine had been taken.

Dr. Koch apparently doubts the authenticity of all cases of blackwater fever in which there is no history of previous quinine administration. I may observe that I myself once suffered from the disease when I had had no quinine for two months, and among Indian sepoys I have had similar cases in which quinine could be absolutely excluded. Further, I know of 2 cases which occurred among aborigines under a skilled observer, and in these there was no possibility of a quinine history. The suggestion which Surgeon-Major Kleine quotes from Stephens to the effect that these aboriginal cases are not haemoglobinuria at all, but are probably due to bilharzia, does not say much for the latter's opinion of the intelligence of the ordinary medical observer. Personally, I have seen both diseases, and I fail to see how any practical man could, with the exercise of care, confuse the two.

The fact that blackwater fever as a rule only occurs in those who have suffered a good deal from malaria, or in those who have been exposed to chronic infection, is readily explainable by my hypothesis. It is well known that in early attacks the system reacts much more strongly than in later ones. The temperature, headache, and general disturbance are much greater, and in consequence the patient is forced to give in. He is new to the disease, and treats himself more rationally as an invalid. Later on, when he has suffered often, his system appears to acquire a certain toleration, and the symptoms and onset are more insidious. For hours before he may be conscious of nothing but a feeling of *malaise* and slight chilliness, although the attack has really begun. The result is that he continues to expose himself to chill at the very stage when it is most dangerous for him to do so. Added to this, his blood is weakened by previous attacks, and this factor supplies the predisposition which I, like Professor Koch, consider essential.

Another interesting point is brought out by Surgeon-Major Kleine's series of cases. Whatever may be the exciting cause of the haemoglobinuria, in my experience the cases which are more apt to prove fatal are those in which no quinine has been given before its onset. In examining Surgeon-Major Kleine's cases we find that only one out of the fifteen proved fatal. In this one there had been four previous attacks of blackwater fever, said to have been brought on by quinine. In consequence, the patient when under treatment for the last time was dosed with methylene blue. As he did not improve he was given 0.3 g. of quinine. Haemoglobinuria supervened and the patient died. It should be observed that though his previous haemoglobinuric attacks were said to have been caused by previous quinine treatment, yet the patient recovered. In my opinion his chances of recovery in his last attack would have been greater, whether haemoglobinuria had supervened or not, if he had been treated with large doses of quinine instead of methylene blue.

Everyone, even Professor Koch, admits that in quinine we have the only reliable and potent antidote to malarial fever. This being the case, its administration is a matter of vital

importance. Professor Koch's theory is apt to frighten the inexperienced, especially among the laity, and they will be inclined to use the drug too sparingly. It is only right that they should know that in other hands quinine has not produced such alarming results as Professor Koch attributes to it. Supposing that it is true that quinine does cause haemoglobinuria, there is no doubt that it cures thousands of patients of malaria for every one that it kills by producing haemoglobinuria. Anything, then, that tends to make people shy of taking it in sufficient doses will result in severer attacks of malaria accompanied by a heavier death-rate. I speak of what I know, and I can affirm from personal experience that Professor Koch's theory has done incalculable harm in those parts of tropical Africa known to me.

NOTES ON FILARIA DEMARQUAIL.

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THE history of *filaria Demarquail* dates back to 1895, when Dr. Manson discovered its embryonic forms in slides of blood sent from St. Vincent by Dr. Newsam, and again later in slides sent from St. Lucia by Dr. Galgey.

Two years after this, out of a number of blood slides of aboriginal Indians sent from British Guiana by Dr. Ozzard, the same authority found two apparently new species of embryonic filariae, one with a blunt tail, the other with a sharp, and provisionally named them *filaria Ozzardi*. Daniels in 1897² found that those latter parasites were very common amongst the Indians in the interior of British Guiana, and in 1898 at a necropsy of an Indian found the parental forms of the blunt-tailed filaria.³ In the following year he discovered the mature forms of the sharp-tailed filaria, finding that it was longer and broader than the former, and resembled *filaria Bancrofti* in some respects.⁴

Galgey in St. Lucia in 1899 found two parental forms of *filaria Demarquail* at a necropsy of a native of that island, describing them as being found in the upper part of the mesentery.⁵ He sent one to Ozzard in British Guiana for identification, who found that the worm was a female measuring $2\frac{1}{2}$ inches long, but owing to imperfections on the specimen, nothing of a definite nature could be made out.

Manson, on comparing the parental forms of the blunt-tailed British Guiana filaria with adult forms of filaria perstans from Africa, found that they were identical, as were also the embryos, and there is very little doubt that after a comparative study of living and stained embryonic specimens of *filaria Demarquail* from St. Lucia, Dominica, and St. Vincent, the sharp-tailed British Guiana embryos are identical with that latter parasite. Specimens of infected blood from those different places when prepared in the same way, namely by washing out the haemoglobin of thick films with water, and then staining with haemalum without fixing, show the similarity very well, and after seeing living specimens of each the identity in measurement and other details appears undoubted.

THE EMBRYOS OF FILARIA DEMARQUAIL.

The embryos of *filaria Demarquail* are very small, but they may be detected, even with low powers of the microscope, in the peripheral blood by day as well as by night. At first their movements are exceedingly active, and they do not remain fixed in one place, as in the case of *filaria nocturna*, but freely wriggle about over the slide.

After their movements have ceased, either naturally or after poisoning them by various vapours, they become stretched out, and can be examined in detail. They measure on an average 0.205 to 0.208 mm. long by 0.005 mm. broad; stained specimens being shorter owing to shrinkage, and also showing more variability in length.

With high magnification in fresh specimens no sheath is visible: this fact probably accounts for their free movement in the blood film. The cephalic armature consists of a rather poorly developed prepuce, not serrated as in the case of *filaria nocturna*, and much more difficult to see, and of a minute

spine which is alternately pushed out and retracted. A V spot exists 0.052 mm. from the head, the angle being more obtuse than in *nocturna*; on account of its small size it is rather difficult to make out. In some specimens there seems to be an indication of a tail spot, but of this I am not certain. The centre of the body is occupied by a core of cells, which become granular as death takes place, and those in dried specimens take on the various stains readily.

In stained specimens no trace of V or tail spot is to be seen, but some haemalum prepared specimens show a break in the continuity of the cells a little anterior to the position of the V spot, similar to that seen in the case of *filaria nocturna*.

PREVALENCE.

St. Lucia.—Out of a total of 472 persons examined from all parts of the island, 23, or 4.87 per cent., were found to be infected with embryos of *filaria Demarquail*, but a detailed examination of the special districts infected brought out the interesting fact that the parasite was very limited in its distribution; 16 of the 23 cases came from Gros Islet, a native village in the north part of the island, situated on sandy soil on the sea coast, with swampy ground and scrub bush around it. Of the other 7 infected cases, 2 had lived in Gros Islet, 1 near it, another at Monchy, also a village on the north coast, while the remaining 3 had never been in that part of the island, having lived in or about Castries, the main town. Soufriere, the second town of the island, and Vieux Fort, a village at the south of the island, always gave negative results. This sharp limitation of the parasite in St. Lucia is very interesting, showing that the infection is chiefly, if not always, got in the country; and, further, that one village may have it while another has not, though they somewhat closely resemble each other, as is the case with Gros Islet and Vieux Fort.

Dominica.—Only 160 cases were examined, and of those 2 alone exhibited embryos of *filaria Demarquail*; one a man from Batalie, a small village near the sea coast at the mouth of a valley, and the other a policeman who had lived all over the island; the latter had been a soldier in Sierra Leone in his youth, but the former had never been out of the island. No cases were found in Roseau, the chief town of Dominica. This, again, points to a localised sphere of infection, and, had time permitted the examination of further cases from Batalie, more from that area would probably have been found infected.

Barbadoes.—*Filaria Demarquail* does not exist in this island. 600 examinations from all parts of the island giving negative results, a fact already mentioned in a paper read at the British Medical Association meeting at Cheltenham last summer on the malarial and filarial diseases of that island.

British Guiana.—Ozzard and Daniels⁶ have shown that the small sharp-tailed and blunt-tailed filariae of British Guiana are not got on the coast of that colony, but only in aboriginal Indians and others living in the interior, and the latter also pointed out that as many as 58.3 per cent. of those people were infected with one or other of those two parasites, the blunt-tailed predominating over the sharp-tailed. In British Guiana 163 pure-blooded Indians were examined by me in different parts of the interior, and out of this number 105, or 64.4 per cent., a slightly higher figure than that of Daniels, were found to be infected with *filaria perstans* or *Demarquail*. Double infections were found in 38, *perstans* alone in 56 instances, and *Demarquail* alone in 11, giving 49 cases of the latter out of 163 Indians examined, or 30.01 per cent. This gives relatively a much higher percentage than St. Lucia and Dominica, but the general population of the whole island, including the towns, was considered in those cases, whereas in British Guiana only the specially-infected area was taken into account, the statistics of Georgetown and other parts of the coast cleared of bush, which do not contain this parasite being omitted. The predominant physical feature for the occurrence of this parasite in British Guiana would seem to be the presence of forest and bush—that is, uncleared land. Georgetown and the cultivated strip of land along the coast is free, whereas on the Waini river, in the bush close to the sea, 9 out of 15 Warau Indians living there were found to be infected. Conversely at Wismar, in the interior sixty miles up the Demerara river, where the forest has been cleared for a con-