combination consisting of malignant and benign tertian parasites. In 3 cases pigmented leucocytes alone were obtained, showing that the patients had recently been suffering from malarial fever.

In the malignant cases the symptoms varied greatly in severity, 8 showing pernicious complications. Two of those were moribund when brought to the dispensary, and both died within a few hours after being seen. One collapsed exhibiting the algid type of the disease, the other in a state of complete coma. Both received hypodermic injections of quinine as soon as the examination of the blood showed the nature of the case, but the disease was too far advanced for this to be of any use. The after-history of the remaining 6 is unknown, as the patients refused to go to hospital and returned to their homes in the country. No cases of hyperpyrexia were seen.

Many of those infected, especially the children, had intestinal parasites as well, the resulting anaemia being very great. In many in whose blood no malarial parasites were found, bad feeding and worms were alone responsible for the anaemia.

Wherever possible the neighbourhood of the dwelling-houses of people suffering from malarial fever was explored with a view to finding the breeding places of *Anopheles*, and in every case such search was successful. The common *Anopheles* of St. Lucia is the *Anopheles albipes* (Theobald), easily recognised by its spotted wings and its characteristic white hind tarsi. It is not a domestic mosquito, never having been found in dwelling-houses during the day; it probably hides in mango trees, bushes, or cane fields in the vicinity. Though never seen in a state of nature by day, insects imprisoned in test tubes will bite readily at any time.

The larvae are found in large numbers in all the valleys and low-lying parts of the island. As a rule one river descends through each valley, and at this season of the year, when there is little water in them, the surf beating on the shore completely blocks up their mouths. As a result a stagnant lagoon is formed behind this barrier, and in those larvae were found in tremendous numbers. The villages of the island are situated on the seashore at the mouths of those valleys—generally within a few yards of the rivers—so the prevalence of the malarial fever in them is easily explained. Round the town of Castries the conditions are different. The Castries River comes down behind the town and opens into the harbour, but, except in the rainy season, it is very sluggish, and numerous small backwaters covered with vegetation exist along its banks; in those the mosquito deposits her eggs and the larvae develop. Unfortunately native houses are crowded along its bank, and the cases of fever from Castries examined at the dispensary mostly all came from this infected area.

Anopheles larvae were also found in a small river course to the north of the town, in an open drain in the immediate vicinity of the town, and in pools of stagnant water adjacent to the Botanical Gardens. The mountainous parts of the island are more or less free from larvae, though in January, 1901, they were found at a height of nearly 800 feet at the back of Morne Fortune, one of the hills behind the town of Castries. This species of mosquito was also found in Dominica, close to the town of Roseau, breeding in an irrigated cane-field, and also in a backwater of the main river; near those areas malarial fever existed. Amongst a series of mosquitos sent by Mr. Lefroy from Antigua *A. albipes* was again present, so it is extremely probable that it is the common *Anopheles* of most of the West Indian Islands.

It is more or less evident from the close relationship existing between this species of mosquito and the malarial fevers of the place, that it is one of the insects that spreads the disease. In order to determine this point a number of experiments were conducted by feeding them on crescent-infected cases; but owing to the great difficulty experienced in keeping them alive in captivity, only a very limited number of experiments were made. Though fed on sugar and different fruits, most of the insects died two to three days after feeding. In one examined forty-eight hours after its meal of blood four zygotes were found, but in three others which had fed on an indifferent case of crescents nothing was found. Benign tertian and quartan cases were not available when

there was a stock of mosquitos, so no experiments with them were made.

The prevention of malaria in the town of Castries resolves itself into a system of better drainage and sanitary improvement. The river in its course through the town ought to have its banks walled in so as to prevent sluggish accumulation of water, as was done with such beneficial results with the Tiber in Rome, and some of the smaller infected places could quite easily be drained. For Europeans residence on the slopes of the hills overlooking the town is indicated. The officers, the troops, and other people living on the Morne very rarely suffer from fever, and though cases do occur, especially amongst the soldiers, it is probable that they were infected in the town. For people compelled to live in the town, especially in the parts near the river, the mosquito net is always indicated. The question of the valleys and other malarious parts of the island is more difficult. The mouths of the rivers could be periodically opened during the dry season to prevent as much as possible the formation of the stagnant lagoons, but as regards the swamps found in the flat parts at the north and south of the island only an extensive system of drainage would suffice.

LEPROSY AND CONGENITAL SYMMETRICAL KERATODERMIA.

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WRITERS on leprosy now and again refer in a general way to the case of a leper mentioned by Gilbert White in his *Natural History of Selborne*. For instance, Dr. George Bidie, C.I.E.,¹ Surgeon-General to the Government of Madras, touches on it in passing, stating that, in his opinion, "it is doubtful if the case was one of true leprosy." I think it can be definitely said that the case was certainly not one of leprosy. Here is White's² description:

There was in this village several years ago a miserable pauper who from his birth was afflicted with a leprosy, as far as we are aware, of a singular kind, since it affected only the palms of his hands and the soles of his feet. This scaly eruption usually broke out twice in the year—at the spring and fall, and, by peeling away, left the skin so thin and tender that neither his hands nor feet were able to perform their functions, so that the poor object was half his time on crutches, incapable of employ, and languishing in a tiresome state of indolence and inactivity. His habit was lean, lank, and cadaverous. In this sad plight he dragged on a miserable existence, a burden to himself and his parish, which was obliged to support him till he was relieved by death at more than 30 years of age. The good women, who love to account for every defect in children by the doctrine of longing, said that his mother felt a great propensity for oysters which she was unable to gratify, and that the black rough scurf on his hands and feet were the shells of that fish. We knew his parents, neither of which were lepers; his father in particular lived to be far advanced in years.

That White meant the true leprosy is shown by the subsequent remarks he makes on that disease in the course of his letter.

The case comes into the category of symmetrical congenital thickenings of the palms and soles, which have been variously designated: tylosis palmarum et plantarum, keratodermie (and acrokératodermie) symétrique des extrémités congénitale et héréditaire, keratoma palmare et plantare hereditarium, ichthyosis palmaris et plantaris, etc. The hereditary nature of the affection is a striking feature. The parents of White's case were not affected, and apparently the patient had no descendants with the complaint; the qualification *hereditarium* must therefore, in this instance, be omitted. The fact mentioned by White—that the eruption usually broke out twice a year, at the spring and fall—is interesting. Azua has recorded an intermittent case of the kind, in which every year about April the palmar epidermis became raised by turbid fluid, probably of sweat origin, leading to desquamation *en masse*, the nails being also shed; from June to November the hands were normal, but in the latter month they again became congested, with a fresh development of the hyperkeratosis.³ In a case of Radcliffe Crocker's blisters formed on the palms every autumn, the whole of the epidermis becoming loosened and peeling off, leaving the skin thin and tender.⁴

It is extremely interesting to note that a similar condition—namely, hereditary palmar and plantar keratodermia—is frequent in the Island of Meleda (Dalmatia), and that such

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.

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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