and sleeplessness. On the 24th his temperature rose to 101° , and he had some pain in the chest on deep inspiration. On examination, pericardial friction was heard along sternum, and this on the 25th had become general over the cardiac area. Temperature now rose to 102.6° . On the 27th there were signs of fluid in the pericardium and pleuritic friction at the left base was also detected. On the 29th his temperature began to fall, and he felt better in himself, and from now onwards his pericarditis and pleurisy rapidly cleared up, and he was discharged nearly well on February 20th. At this time not only had the chest symptoms disappeared, but the joint conditions had much improved, and his general health was better than it had been since the onset of his illness.

With what were we dealing in those two cases, which are typical of a class of cases by no means extremely rare? Were we dealing with a mixed infection, or were those cases due to the same infection as had given rise to the arthritis? That they must have been due to an infective organism is, I think, clear, as how else can we explain the sequence of events? In one we had pericarditis followed by pneumonia, diarrhoea, and a skin eruption; in the other herpes followed by pericarditis and pleurisy; while both occurred in the course of acute rheumatoid changes. They thus strongly point to some blood infection. Another point is the rapidity with which the symptoms cleared up in the joints—a rare occurrence in uncomplicated rheumatoid arthritis. Did the organism, if such were the cause of the pericarditis, during its life cycle elaborate in the blood a substance curative to the joint changes? If so, the cause of both conditions must surely have been identical. If rheumatoid arthritis were due to a reflex nerve change, or to one having a central nerve origin, one would expect the arthritis to be made worse by the occurrence of such conditions as the above cases illustrate. In fact, one is driven to the conclusion that they must have been due to an infective organism, and that in all probability the cause of the rheumatoid disease was the cause of the pericarditis, and that these organisms gave rise to some metabolic change inimical to their further existence both in the joints and other special sites. In pure uncomplicated rheumatoid arthritis such production must occur more slowly. If this theory is not correct, the solution of the mystery must rest in the supposition that the infection was a mixed one, and that the products of one were inimical to the life of the other. Unfortunately, in neither case did I feel justified in aspirating the pericardial fluid, but in both from the joints I obtained specimens of the organism I believe to be specific to the disease.¹ I much regret not having been able to obtain proof as to the cause of the pericarditis. In both cases endocardial lesions had arisen before coming under observation, but in neither could reliable histories of their onset be obtained. In neither, however, was there any history of antecedent illness likely to cause such lesions, and I think, therefore, we may justifiably presume that they arose during the course of the illness, and that they were caused by the same agent as gave rise to the pericarditis. I am much indebted to Mr. L. H. Walsh for the careful notes of these cases.

REFERENCE.

¹ *Lancet*, April 25th, 1896.

THE PARASITE OF MALARIA IN THE FEVERS OF WEST AFRICA.

By SURGEON-CAPTAIN C. W. DUGGAN, M.B.,
Army Medical Staff.

DR. MARCHOUX in a paper entitled *Le Paludisme au Senegal*, published in the *Annales de l'Institut Pasteur*, describes the symptoms and treatment of malaria in Senegal, West Africa, and gives a detailed account of the appearances presented by the parasite in the peripheral blood and internal organs. His observations extend over a considerable period of time and include the examination of a large number of cases.

I propose to refer to Dr. Marchoux's paper with a view to indicate some points wherein his description differs from what I observed in cases of malaria at Sierra Leone. According to Dr. Marchoux the symptoms of malarial fever in Senegal are as follows. There is a feeling of *malaise* for two or three days, followed by fever of a continued or remittent type, with vomiting. Icterus is frequently present. The patient is extremely weak, often delirious, and threatened with a pernicious attack. After three or four days' treatment

the patient is well, but from the twelfth to the fourteenth day there is a return of the fever. Another relapse occurs twelve to fourteen days later. In some patients the attacks continue till the malaria has become chronic. Pulmonary congestion is the most frequent complication.

Dr. Marchoux made a microscopic examination of the blood in 347 cases, and found parasites on every occasion. They varied in number according to the gravity of the attack and the time at which the observation was made. Five to six and even more parasites were seen in the same field, and one to four parasites in each affected red cell. The author makes special reference to a process of the parasite by means of which it penetrates the red cell. He gives a drawing of two stained specimens in which this process is visible. He has seen a parasite partly within and partly without a red cell. As a rule the parasite develops without pigment. Crescents were seen on the twelfth day after a first infection, and were the first signs of a relapse. At this period large pigmented parasites in their external appearance resembling tertian parasites were found, but instead of forming rosettes they became spheres, and the pigment collected itself in the form of a central halo, and then the spheres became oval and finally crescentic; these two last stages may be wanting as in tertian and quartan types. Dr. Marchoux considers that the constant appearance of the crescents at the time when there is a recurrence of the fever points to an encysted stage capable of further development. He has never seen a free crescent. He believes that the form of the malarial parasite seen by him in Senegal is identical with the summer-autumn parasite of Marchiafava and Bignami. He states that it has already been described by Ziemann in the Cameroons and by myself at Sierra Leone.

In the Roman parasite the formation of pigment is the rule and on the West Coast it is the exception. With reference to the size of the parasite Dr. Marchoux affirms that it is smaller than the Roman parasite (during the winter not more than 1μ) and towards the month of November, when the fever is milder, it is of the same size as the Roman parasite, and later on even exceeds it. He is of opinion that the resistance offered by the individual attacked is the most important factor in influencing the size of the parasite; the greater the resistance the parasite encounters the larger it becomes. When convalescents return to Europe with chronic malaria, it is the large form of parasite that is found in their blood, and in support of this statement Dr. Marchoux affirms that M. Laveran, who has examined many such cases, has always found this form of parasite.

From Dr. Marchoux's conclusions I quote the following: "In Senegal malaria is only contracted during the rainy season. A daily dose of 25 cg. of quinine sulphate is strongly recommended as a prophylactic. The parasite of malaria is polymorphic." From a comparison of the symptoms of malarial fever in Senegal (Dr. Marchoux's observations were made in the part which lies to the north of the Gambia) with what I found at Sierra Leone, it appears that there is no essential difference between the fevers of the two districts.

My observations on the fevers and parasite of malaria of Sierra Leone are recorded in a paper read before the Royal Medical and Chirurgical Society and published in the *Transactions*, vol. lxxx, p. 213. As will be seen by a reference to this paper the onset was sudden in a number of my cases. In some, relapses occurred every few days, in others less frequently. Pulmonary congestion was a rare complication. I was unable to detect a process connected with the parasite in any of my stained specimens. Before the death of the parasite the pseudopodia were always retracted, and the parasite assumed a ring shape. So far as I was able to observe no parasite was seen to possess a pseudopodium specially designed to enable it to penetrate a red cell. The parasite adhered firmly to a red cell, and while developing appeared gradually to sink into it. I did not observe any appearance which might be interpreted as that of a parasite only partly within a red cell. In all my cases the development of the parasite was accompanied by the formation of pigment. Crescents generally appeared a few days after the commencement of fever. I have seen them as early as the end of the second day in a patient who had never previously suffered from malarial fever. In a number of hospital patients

the appearance of crescents in the blood was not followed by fever. They persisted for several weeks, and the patients were discharged from hospital apparently in good health.

In some of my cases deeply pigmented spherical bodies accompanied the crescent form; in them the pigment was generally arranged in a dense mass occupying the interior of the parasite. These pigmented parasites were never larger than a normal red cell, and could readily be distinguished from the large pigmented parasites of tertian fever. In one instance I saw a pigmented sphere slowly change into a crescentic form. Among several hundreds of crescents that I examined with care, I did not observe any appearance suggestive of an encysted stage of the parasite, and in this connection I may mention that I saw in one oval form of the crescent series three large, well-marked spores, each possessing a distinct nucleolus, while a fourth spore, which had evidently just escaped, lay close to this oval-shaped parasite.

In many cases crescents were free in the blood, no fine line connecting the ends of the crescent being visible, and the periphery of these crescents did not stain with eosin.

At Sierra Leone the season of the year did not influence the size of the parasite. Frequent observations were made during both the dry and rainy seasons, and the parasite was always found to present the same appearances.

Some patients were frequently admitted to hospital with attacks of malarial fever, while others suffered to a very small extent; but in all cases the same size of parasite was evident.

Since my return to this country I have had an opportunity of examining several sailors invalided after the Benin expedition. They were sent to the Royal Naval Hospital, Plymouth, where I made a careful examination of their blood, some months after they had left the African coast. In these cases the appearances presented by the parasite were in every respect identical, both as regards its form and size, with what I observed at Sierra Leone—the minute unpigmented forms, the slightly pigmented, and crescents. The activity of the parasite did not appear to be at all diminished by the change of climate, and I detected no difference in its size. In one of the cases the temperature had been normal for two days. All the men had been treated with daily doses of 10 to 15 grs. of quinine sulphate.

From this brief account it will be evident that the conditions referred to by Dr. Marchoux as affecting the size of the parasite of malaria caused no alteration in the appearance of the parasite seen by me at Sierra Leone and in this country. I am of opinion that the parasite of malaria observed by me at Sierra Leone is identical with the parasite of the summer-autumn fevers of Southern Italy, described by Marchiafava and Bignami, and that there is no evidence of polymorphism in its development. The only case suggestive of polymorphism was admitted to hospital shortly after the beginning of the rainy season. The appearance of the parasite agreed in every detail with the description of the tertian parasite given by Golgi, and with specimens of this parasite kindly sent me by Dr. Thin.

Malarial fever may be contracted during the dry season. I have employed quinine as a prophylactic in daily doses of 5 grs., but only succeeded in inducing a few persons to continue this treatment. The results were sufficiently encouraging to warrant the advisability of all who have to live in a malarious district adopting this precautionary measure. The hydrochlorate is preferable, being stronger, more soluble and palatable than the sulphate.

TRANSIENT AND RECURRING PARESIS IN ACUTE CEREBRAL SOFTENING.

By S. A. ORD-MACKENZIE, M.R.C.S.ENG., L.R.C.P.LOND.
Woolhampton.

THE somewhat indefinite symptoms of acute softening due to thrombosis of the cerebral arteries may make those observed in this case of special interest.

At the commencement it was one of the usual type. Severe and persistent neuralgic headache above the left orbit had existed in a woman aged 66 for ten days or so before I saw

her. Except that her power of memory seemed to fail from day to day, the symptoms remained practically unchanged for a fortnight, till one afternoon she became suddenly drowsy, cerebation was slow, and the speech blurred. Two hours after this there was slight paresis of the right side of the face, shown only by a faint droop of the angle of the mouth, and by the ala of the right nostril being flattened. The pupils were equal, sluggish, and somewhat contracted. The conjunctivæ had a yellowish tint. The following morning the paralysis had disappeared, the conjunctivæ were normal in appearance, but the patient remained semi-comatose.

She passed a restless night, and the next day the nose and mouth were drawn far over to the left side, the conjunctivæ were again yellow, and the skin of the face had a distinctly jaundiced look. The extremities were cold as far as the knees and elbows, and also the left side of the neck. The knee-jerks were gone. She was sensible to pin-pricks on the soles, but sensation seemed dulled, and the feet were drawn slowly away, and only after a distinct interval. The right hand was hyper-extended, the fingers and elbow flexed. The hand and forearm would respond slightly to stimulation, but power in the shoulder seemed to be lost.

Six hours later no traces of paralysis or pigmentation were to be found, the patient spoke quite rationally, but the same evening it all returned, and she was as drowsy as ever.

Two days later she began to pass her water under her, and the pulse, which still continued full, dropped from 54 to 42. (No drugs had been given since the coma first set in.) Next morning there was marked external strabismus of the right eye, which had gone by the following day. The pupils of both eyes were generally strongly contracted when the lids were suddenly raised, and then slowly dilated. No optic neuritis was present in either eye, but the choroid of the left had a blotched and mottled appearance as though from old capillary hæmorrhages. The surface of the cornea of the right eye was hazy and as though studded with minute boiled sago grains, which had not been noticed when the eyes were examined the previous day. The media had become cloudy, and only one or two of the retinal vessels could with difficulty be indistinctly seen.

There had been no history of any impairment of vision. When the examination of the eyes was commenced there was no paralysis of the face, but it was very marked before the examination was concluded, and then it appeared that local irritation, such as raising the lids or touching the tongue, would bring it on. At those times when these focal symptoms were most marked the conjunctivæ would become deeply stained and the face also, though not when the palsy was induced by irritation. The urine shortly after would have a dark "portery" appearance, though this grew less marked as the case went on. It was faintly acid, contained no albumen or sugar, but immense quantities of oxalate of lime and minute traces of bile pigments.

There had been great frequency of micturition for some days, almost half-hourly calls, and a considerable quantity at a time. Two days later the pulse rose to 76 and the respirations to 34, though examination of the chest revealed nothing. Rhythmical movements of an athetotic character were present in the right arm.

The forearm would be flexed to a right angle, then rotated in; the thumb was then strongly flexed, and lastly the limb would be slowly extended again. This would occur about six times a minute, and lasted several hours. Four days later the patient spoke quite sensibly, smiled at and knew everyone around her, remarked that she felt better and would like something to eat, objected to milk, but made signs for something solid. Half an hour later the coma was as deep as ever.

Towards evening she showed signs of Cheyne-Stokes breathing, pulse 88, strong, and incompressible. Next day the temperature, which had throughout the illness been normal, rose to 100° F., pulse 80, respirations 48; pupils unequally contracted, the right the smaller of the two; all reflexes gone, both superficial and deep. No urine had been passed for twenty-six hours, the bladder appeared empty, and on passing a catheter only a few drachms were drawn off. The bowels had been constipated all through. Slight hypostatic pneumonia at both bases.