scarcely attain the fourth th rib. This border is by far the most important one to map out with accuracy, as it gives a clearer estimate of the condition of the heart. The right border coincides as a rule with the left lateral sternal line; sometimes it extends out to the sternum, but it is usually impossible to obtain a trustworthy result over that bone from the presence of its own resonant note. The lower limit cannot be distinguished from the upper margin of the liver dulness.

Such is an outline of the area of dulness which may be observed in healthy individuals in the prone or stooping posture, and it is of interest to compare the limits of the heart as ascertained in the ordinary position. For this purpose I have introduced an outline diagram (Fig. 5) from Vierordt's *Medical Diagnosis*, a standard work on the subject. It will be seen that the absolute dulness in the stooping posture approxi-

Fig. 5.

Fig. 5. Normal percussion figure of the heart from Vierordt's *Medical Diagnosis*. Light shading represents relative dulness.

Fig. 4.

Fig. 4.—Outline of percussion area from a man 26 years of age. Light shading represents increased dulness in stooping posture; x x the cardiac impulse in different postures.

mates very closely to the area of relative dulness as percussed by the ordinary method, the only difference of moment being a somewhat wider limit for the upper and left boundary in the latter. The shoulder, so to speak, of the margin does not move out so clearly in the stooping posture, a modification which is readily explained by the base of the heart having a more limited range of movement and not coming into such close contact with the chest wall as the body and apex. On the whole, however, I think it will be conceded that the resemblance is sufficiently close to enable one to draw an accurate estimate of the anatomical limits of the heart from information afforded by the absolute dulness of the prone or stooping position.

With regard to the advantages of this method of examination, I believe it will be found to be much more precise than the estimation of the relative dulness in the supine position, inasmuch as it can be carried out by a lighter stroke and the percussion note is more definite. We avoid those transitions of sound which easily lead to error and which are scarcely to be recognised even by long training and the most practised ear. The constant variation in the pulmonary borders is largely eliminated, and having to deal with an increased area giving an absolutely dull note, the accuracy of our percussion is greater and observation is less likely to err. Lastly, there is a further advantage which has been pointed out by Gumprecht in that it brings out an area of dulness which may be quite impossible to ascertain in the supine position. In tympanites of the stomach and colon I have on more than one occasion experienced difficulty in determining the cardiac area until the patient was placed in the stooping posture.

REFERENCES.

- ¹ *Jour. of Phys.*, vol. 10, Nos. 5 and 6. ² *Deut. Arch. f. klin. Med.*, Bd. 56. ³ *Journ. of Anat. and Physiol.*, vol. 9, p. 137. ⁴ Ueber die Häufigkeit eines sicht- und fühlbaren Herzpfeifens beim Menschen. By Heinrich Guleke, *Inaug. Diss.*, Dorpat, 1892. ⁵ *Loc. cit.*

MEDICAL MAGISTRATE.—Dr. John Griffith Lock, J.P. for the Borough of Tenby, has been appointed by the Lord Chancellor, upon the recommendation of the Lord-Lieutenant, to the Commission of the Peace for the county of Pembroke.

PERICARDITIS IN RHEUMATOID ARTHRITIS.

By GILBERT A. BANNATYNE, M.D. GLASG.,
M.R.C.P. Edin.

Honorary Physician to the Royal Mineral Water and Royal United Hospitals, Bath.

THE question of heart affections in rheumatoid arthritis is one which has given rise to much discussion. While endocardial lesions are found with a comparatively greater frequency than those of the pericardium some doubt has always existed as to their origin and nature. The same may be said with greater truth about pericardial lesions, and for this reason I think the two following cases may be of interest. In both we have the occurrence of an acute pericarditis during the course of rheumatoid disease, and presumably, therefore, intimately connected in origin with it.

CASE I.—R. D., aged 24, tailor's assistant, married, was admitted to the Bath Mineral Water Hospital on January 25th, 1896, suffering from rheumatoid arthritis. Her family history was unimportant, whilst her previous history had been distinctly good. She had been married two years and had one child twelve months old. Her confinement was natural and uneventful, but a month after she caught cold, and pain came suddenly in both knees, then in her ankles and feet, and spread rapidly to her hands, wrists, and shoulders, and quite recently to her elbows, jaws, and neck. Her condition had steadily been getting worse. On admission she was found to be a rather badly nourished woman, who was losing flesh fast, and whose temperature was never quite normal. Otherwise her general health was fair and her appetite good. Menstruation was scanty and irregular. On examination the heart's apex was found in the fourth interspace in the nipple line. It was heaving and diffuse. The cardiac area of dulness was increased slightly in all directions. A loud systolic and a post-diastolic murmur was heard at the apex, with accentuation of the second sound at the base. The joints most affected were those of the fingers, the wrists, the jaws, and the neck; the shoulders, elbows, knees, and ankles to a less extent. There was marked muscular wasting of the interossei and extensor muscles of the forearm and hand. There was sweating of the hands and pigmentation of the skin of the forehead, hands, and forearms. The hands and feet felt cold, and looked blue, and the axillary glands were enlarged, but there was none of the spleen. The pulse was 86 and the temperature 99.2°. The urine contained neither sugar nor albumen. Under treatment she progressed slowly, if at all. On February 16th, three weeks after admission, she complained of pain in the chest, and the temperature was found to have risen to 101.4°. Loud pericardial friction was detected over the second left cartilage, her pulse was quick, and there was some difficulty in breathing. From the 16th to the 25th the friction extended, and finally could be heard all over the cardiac area, her temperature meanwhile ranging from 99° to 103°, and her pulse from 100 to 140. During the evening of the 25th she complained of a short hacking cough, and she became slightly cyanosed. Over both apices there was slight loss of resonance, with some bronchial breathing and fine inspiratory crepitations were detached. On the 26th the expectoration, which was difficult to bring up, was rusty in character, and her temperature remained about 102°. On the 27th the heart's action was very irregular and feeble, and there was oedema of feet and legs. A trace of albumen was found in the urine. On the 28th, although the breathing was still rapid, it was not so laboured, the expectoration had become frothy, her pulse was more regular and the temperature was slightly less, as was also the oedema. On the 29th she felt better, and about midnight she became much easier, her breathing became quiet and less hurried, her pulse more regular, and her temperature fell to normal within a few hours. During March 1st she continued better, but on the 2nd she complained of some pain between the shoulders, and had diarrhoea, whilst a rash appeared on her hands. This latter was bright red in appearance, slightly raised, and seriginous in outline. Her temperature, too, rose to 101.4°. On the 3rd she was better, her breathing easier, and in no pain. The rash began to fade. On the 4th she continued better, and on examining the chest it was found that the symptoms were rapidly clearing up, and that the pericarditis was practically gone. From that date until her discharge, on the 28th, she progressed rapidly, getting stronger and better every day. The lung and heart mischief especially cleared up in a wonderful way. The murmurs, however, persisted, but they, too, were less intense in character. The joint symptoms from the onset of the pericarditis improved rapidly until all swelling and pain had nearly gone, and this continued until her discharge, when the hands were no longer asphyxiated in appearance, nor were they cold or wet. A certain amount of deformity still remained, but for all practical purposes the condition appeared to be cured.

CASE II.—G. H., aged 37, labourer, was admitted on January 3rd, 1896, suffering from rheumatoid arthritis. His family history showed phthisis on the maternal side, but his previous history was good. His present illness began thirteen months ago, after an attack of influenza and tonsillitis, with stiffness and pain in the right thumb, which soon spread to all the joints of both hands, to the knees, and later on to shoulders, elbows, wrists, jaws, and back of neck. On admission he was found to be a badly nourished man with fair appetite, bowels constive, but fast losing flesh. Within the last few weeks he had got rapidly worse. On examination, the heart's apex was found in the third interspace $\frac{1}{2}$ inch internal to the nipple line. The first sound at the apex was impure, but no distinct murmur. He had considerable swelling of the joints of the fingers, of the wrists, and knees, the other joints being affected to a less extent. Both hands and feet were wet with perspiration, and there was marked pigmentation, with wasting of the extensors of the arms. On January 7th he developed a herpetic eruption along the course of the right ilio-inguinal nerve, his general condition meanwhile remaining much the same, except that the temperature, which on admission was 99°, rose to 100°. For several days this persisted, and he suffered much from pain

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