the earliest times* have dwelt on the existence of such pains. Ricord attributed them to the difficulty with which the periosteum and medulla were distended by the inflammatory process. Fournier proposed the term *ostealgia* as a vague provisional denomination for those cases where no local lesion could be discovered in contradistinction to the other two forms of pains in the bones, namely, periostitis and periostosis, which presented palpable lesions. Lancereaux has shown that while pain is sometimes observed apart from any appreciable lesion, it is more frequently connected with a material lesion of the bones and fibrous tissues, and adduced as testimony the fact that many autopsies had revealed changes in the dura mater or an exostosis on the internal table of the bones of the skull in cases in which during life the only symptom observed had been osteocopic pains. Aching pains in the bones, most marked at night, occasionally occur after enteric fever (Cayley), but a previous history of similar pains and the fact of their yielding so readily to iodide of potassium indicated a syphilitic origin, while the absence of sweating, pyrexia, and disturbance of the general health excluded rheumatism, for which the patient herself had mistaken them.

This case further illustrates how a disease hitherto latent may be roused into fresh activity by an acute illness, though the latter is not etiologically connected with it. This fact is well known in the case of vaccination, which on several occasions has been unjustly held responsible for the phenomena of hitherto latent congenital syphilis.

In conclusion it may be said that the case is of interest as being a form of relatively benign hereditary syphilis. The osseous system alone had been affected, the brain, special senses, skin, and viscera were intact, nor was there any history or past evidence to show that they had been affected. The case thus affords a good instance of those incomplete types of disease which Trousseau denominated *formes frustes*, and so happily compared to the defaced inscriptions which from a few remaining words or letters the skilful archaeologist could completely reconstruct.

I am indebted to Dr. J. E. Beggs, Medical Superintendent at the Grove Fever Hospital, for permission to publish this case.

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SOME CLINICAL FEATURES OF MEDITERRANEAN FEVER.

WITH PARTICULAR REFERENCE TO CARDIAC COMPLICATIONS.

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FROM a considerable experience of this disease I have been forced to recognize that at the present time hardly enough value has been given, for diagnosis, prognosis, or treatment, to the important effects produced by the specific organism of Mediterranean fever on the heart.

I think these may be divided into two kinds: (1) Toxic; (2) microbic.

It has, of course, for many years been recognized that in all its stages the heart is much influenced by the disease. In the early periods, with high fever (or in any acute pyrexial relapse), the most likely cause to bring about a fatal result is heart failure, from which the patient quietly dies without warning symptoms.

* Peculiare et maximum est symptoma in morbo Gallico dolor articulum primum capitis universi, vel dimidii, deinde articulum superiorum et inferiorum, et praecipue internodiorum. Hic dolor die quiescit, noctu vel ante caenam, vel post caenam solet excruciare et insomnes reddere homines. Dolor est pungens, quasi distendens et lacerans. . . Pungunt isti dolores, quia sunt circa perihostia. Solent esse cum tumore, et sine tumore.—G. Fallopius, *De Morbo Gallico*, c. 94, Patavii, 1583.

In the later stages of the disease it is usual to find a fast pulse, irregular palpitations, excitable action of the heart on slight exertion or mental perturbation; therefore, as a routine, the character of the heart's action in a chronic Mediterranean fever case is one of the first points that I investigate.

We should bear in mind the frequency with which these symptoms are associated with general and localized attacks of neuritis, neuralgia, hyperaesthesia (so painful and distressing), or with paresis or partial paralysis of certain groups of muscles, all of which pass away after variable periods, but always tending in chronic cases to prolong the suffering, prevent sleep, and render the patient miserably depressed. Seeing these cases day by day one cannot doubt the presence of a powerful toxin in the blood acting markedly on the heart, a poison analogous in many ways to that found in diphtheria.

The point which is, however, less recognized, and to which I particularly wish to draw attention, is that not only is the heart subject to be influenced most detrimentally by the toxic product of the *Micrococcus melitensis*, but that the organism itself is able to set up endocardial changes which are most dangerous. This point seems to me to be clearly established from cases of my own, and from one occurring at the Seamen's Hospital, Albert Docks, which, through the kindness of Sir Patrick Manson, I am able to quote.

It must be remembered when treating Mediterranean fever cases that the *Micrococcus melitensis* is constantly present in the blood during the course of the illness, that it is very virulent, and that the protective leucocytes in the blood are greatly diminished both in number and in phagocytic power.

It does not seem unlikely, then, as with the streptococcus, pneumococcus, and gonococcus, that with some change in the cellular elements of the cardiac endothelium, ulcerative changes may take place and symptoms of acute or malignant endocarditis at times occur.

In looking over the notes of cases treated by me at Haslar during the last five years, I find that out of 596 there was 1 death due to cardiac failure, and out of 42 cases invalided out of the service, 11 were for organic heart disease, due to, or occurring with, the fever, besides a number who developed cardiac murmurs but were not invalided.

Of these cases, I would especially draw attention to two reported below. The second, during a typically prolonged type of the fever, suddenly developed such severe symptoms of endocarditis, that for a time the prognosis was most doubtful. In his case the diagnosis was not only proved by the clinical symptoms and agglutination reaction, but also by the isolation of the *Micrococcus melitensis* from the blood.

CASE I.—F. L., gunner, R.M.A.

History.—Gradual onset of fever and debility associated with cough and pain in the chest, thought to be bronchopneumonia. Sent to Malta Hospital, where he developed typical signs of Mediterranean fever. The blood agglutinated with a dilution of 1 in 150. The spleen was large. The fever ran high, and he was invalided home. On admission to Haslar Hospital the cardiac impulse was diffused and action excitable. He had a slight rise of temperature every evening; after a week a soft systolic mitral murmur was made out; the pulse was fast, and he had pain in the chest. The following week the bruit was noted as being long and blowing; the temperature was still of a hectic type. Four days later a presystolic as well as a systolic bruit was found. The fever slowly abated, and he regained strength, being finally invalided out of the service after two months' treatment in Haslar.

Here organic heart disease set in during the course of the disease, and by the changing character of the murmurs led one to believe that they were due to freshly-formed vegetations.

CASE II.—C. N., aged 32. Petty Officer.

History.—This man had a gradual onset of fever at Salonica which was thought to be enteric, as diarrhoea was a prominent symptom; the temperature chart and course of the disease at Malta Hospital, however, were typical of Mediterranean fever, and while there a mitral bruit developed, but was said to be very variable in character; the fever ran high and was distinctly undulant in character, there having been five waves before admission to Haslar. On admission—temperature normal, pulse 112, no pains, heart's impulse diffused, a soft mitral systolic murmur present. On December 30th, one week after, he had a rigor, his temperature rapidly shot up to 104°, pulse 130, with headache and slight cardiac distress; the next morning a loud bruit was heard, but the action was regular; the

temperature kept up and signs of pulmonary congestion were evident. On the evening of January 5th the notes state: temperature 103°, pulse 120 small, marked muscular tremors and very weak; heart showed evidence of dilatation, with a mitral systolic murmur; cough, albuminuria, and occasional attacks of shivering. On the 7th his temperature ran up to 105.4°, condition very critical, the next day the murmur was loud and blowing all over the cardiac area; after a morphine injection he had a good sleep and from that time his condition slowly improved, the heart regained tone, the lung symptoms and albuminuria cleared up and the temperature fell to normal in the mornings though still hectic in character, with wandering delirium at night, and troublesome diarrhoea. On January 8th 2 c.cm. of blood was taken from the arm in the usual way and cultivated in 50 c.cm. of broth, giving a pure growth of the *Micrococcus melitensis*. In fourteen days' time the wave had passed, and, except for extreme debility and the heart bruit, there was little to note. Two short waves, however, followed, in one of which he again had a distinct rigor, then convalescence set in and four months after admittance to Haslar (six months' total sickness) he left for his home.

Sir Patrick Manson's case seems to be essentially similar, except that the result was fatal.

In this patient the blood gave a positive agglutination reaction, found independently by Hewlett, Wright, Low, and myself. The *Micrococcus melitensis* was also obtained from the blood by Dr. Wise at the Albert Docks; a culture which he sent to me for confirmation I found reacted well with the serum of three different cases of Mediterranean fever, besides giving the cultural tests. Rigors with increased fever set in and the patient died. *Post mortem* malignant endocarditis with infarcts in the spleen and kidney were found; unfortunately the *Micrococcus melitensis* was not isolated from the spleen.

The resemblance of these cases is very great, and I have no doubt in my own mind that the endocarditis with its subsequent infarcts in the spleen and kidney were really due to a particular virulent strain of the *Micrococcus melitensis*. The importance of recognizing the possibility of this form of infection and termination cannot be over-estimated by those who have constantly to deal with cases of Mediterranean fever.

Another point which is important to remember is that in very prolonged cases with much cachexia, the agglutination reaction falls so low as often to be not demonstrable. This I am certain of, for I have followed cases through in my wards and the blood in the laboratory, and have in well-marked Mediterranean fever cachectics been unable to get any reaction with a 1 in 20 dilution; from this I draw the conclusion that the absence of a positive reaction in a chronic case does not negative the diagnosis of Mediterranean fever.

HAEMOGLOBINURIC FEVER IN SYRIA.

AND SOME NOTES ON THE OCCURRENCE OF THE DISEASE
IN PALESTINE.

By E. W. GURNEY MASTERMAN, F.R.C.S.,
JERUSALEM.

It is, I think, not generally known that haemoglobinuric, or "blackwater," fever often occurs in Palestine. Not only is the place not mentioned in the accounts I have seen of the geographical distribution of this disease, but in *Albutt's System of Medicine* it is stated "in Asia the disease is practically unknown" (vol. ii, p. 742). This is certainly incorrect as applied to Palestine, for within the last ten years I can recall some severe and fatal outbreaks which have occurred in Southern Palestine. In 1893 there was a regular epidemic of malaria in and around Jaffa, and among the fatal cases a great many presented symptoms we now recognize as being those of blackwater fever. An able native doctor described them at the time as being like "yellow fever," and, if I remember right, a correspondent wrote to an English newspaper that there had been an outbreak of yellow fever in Jaffa.

During the last two or three years there have been several outbreaks around Jaffa. For example, in the German colony of Saronia in 1901 there were 5 cases, 2 of them fatal—one a young man of 16, who died on the third recurrence of the disease, and the other a girl of 2. In 1902 three more cases occurred, but all recovered. I now have one of the recovered cases as a servant in my house, and from her description it is evident the symptoms

were the same as those detailed below—indeed, she says the disease was called *Schwarz wasser-feber*.

Similar but more fatal outbreaks have occurred at the Jewish colony of Mulebbas, and a few cases at Rishon le Zion, both near Jaffa.

In Jaffa and neighbourhood the conditions are favourable to the disease—low-lying, marshy ground and damp, semitropical heat—but that cases should arise in a place like Jerusalem must be unusual. We lie 2,500 ft. above the Mediterranean. At the time of the onset of the disease in this case (November) there had been scarcely a drop of rain since the previous April; there are no marshes; the heat has not been at all excessive (the whole summer only four times as high as 90 degrees in the shade); and on the whole the air has been dry and fresh.

At the same time, it may be noticed, we have the usual association with malaria, for Jerusalem must be called an exceedingly malarious place. Very few residents escape occasional attacks, and many of the poorer class suffer very much indeed. Among my hospital male patients I find the spleen enlarged some inches below the ribs, and easily felt in 33 per cent. of all cases; and large "ague cakes," extending to the right of the umbilicus, are common.

In respect to malaria, we are indeed worse off than even Jaffa. At the same time, blackwater fever is not common. Among the thousands of patients I see annually I have never had a case under regular treatment, though I can now recall a few who have produced for my inspection a vessel containing the characteristic urine. I have in the past looked upon such cases as simply severe malaria, with "bloody" urine as a complication.

Herr Dr. Einsler, the President of our local Medical Society, tells me he can recall seeing three other cases arising in Jerusalem, two of which were fatal. My own impression is that it is commoner all over the land than is suspected, but hitherto it has not been looked upon as a distinct disease but simply a severe complication of malaria.

In the case detailed below I think there can be no doubt about the diagnosis, but to me it was further confirmed by an experienced trained nurse, who had nursed many cases of this disease in East Africa, and who told me the appearance of the patient (on the last day) was exactly like those of fatal cases of which she had had charge:

D., a Jew of 16, whose family came from Bokhara in Central Asia, but who himself had been nine years in Jerusalem, came as an out-patient to the English Mission Hospital, Jerusalem, on November 9th, 1903. He complained of intermittent attacks of "fever," accompanied by dark urine, "like blood." As he was jaundiced, I at first suspected the presence of bile pigment as the cause of this dark urine.

The history of his illness, as obtained after his admission from his brother, was as follows. He had not been out of Jerusalem at all for two years, and had from time to time suffered from "fever." On November 1st he got an unusually severe rigor, and passed dark-reddish urine. For four days he had similar attacks daily, each one being accompanied by this dark urine, and also by severe vomiting. He got some medicine from a doctor, and the symptoms abated, and for four days he went back to his work. On November 8th he had a recurrence of the symptoms, and the next day sought admission to the hospital.

Condition on Admission.—The patient was a fairly well nourished man, markedly anaemic. His skin was slightly yellow, and conjunctivae markedly so. His temperature normal, pulse 84, regular, and fair volume. Heart-sounds rather feeble, but no murmur. Spleen enlarged, 3½ in. below line of ribs, hard, and easily felt; liver not distinctly enlarged nor tender (two days later it could be distinctly felt 1 in. below the ribs). Urine clear, light yellow colour; acid; no albumen, no sugar.

Progress.—The general course of the case is indicated by the annexed chart, on which, among other points, the occurrence of haemoglobinuria is recorded by the letter H. In detail the case progressed as follows: At 2 p.m. the patient got a severe rigor with vomiting; after shivering for half an hour, violent perspiration set in, and he began drinking great quantities of water. He passed several ounces of very dark urine, which unfortunately was upset in the bed, but the ward sister noticed that it left bloody stains on the bed clothes. Temperature rose to 103°.

November 10th, 7 a.m. Temperature this morning down to 100°; he seems comfortable but weak and at times restless. Has pain in splenic region. B.O. A specimen of urine passed last night was examined. It was dark porter-like in colour with a dirty-white sediment. It was alkaline from standing most of the night in an improperly cleansed vessel. When