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.,
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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

boiled the heated upper layer became denser. Under the microscope no blood corpuscles were found. Sediment consisted of quantities of granular material, some of it in form of casts, and great numbers of crystals of triple phosphates. No epithelial cells, no leucocytes. Numbers of long rod-like bacteria, some in couples. At 11 a.m. patient got a very violent rigor after a stage of extreme restlessness and severe abdominal pain; he became almost pulseless; blue round lips and finger-tips; shook violently; rigor lasted three-quarters of an hour. He had hot flannels applied to his heart, which as it recovered itself, began to beat with great violence. Complained much of cardiac pain and distress, and also pain in splenic region. Temperature rose to 102°. He drank freely, but vomited everything at once. At 4 p.m. he was given 10 gr. of quinine, but failed to retain it. Specimen of urine passed at 5 p.m. lighter in colour than that examined in morning—clearer, one or two red blood corpuscles, but otherwise sediment like that examined in the morning. At 8 p.m. he had another rigor, attended with severe shivering and collapse, lasting ten minutes; lips quite bloodless.

November 11th. During the night the patient passed a considerable quantity more dark, porter-like urine. First half of night was very restless and could not sleep; talkative but sensible. Was given pil. opii gr. ss at 10 p.m., repeated at 2 a.m., after which he slept. 7 a.m.: seemed better. B.O. this morning. Urine passed this morning acid, 1013, quite clear, dark red, with transmitted light, no sediment, no albumen. At 11.15 a.m. the patient got another severe rigor with great cardiac distress; relieved by hot flannels; repeated severe vomiting, at first yellow but later bright green. No urine till 1.30 p.m., when he passed a quantity of dark, porter-like urine; this specimen was dark-red in colour, but not quite so dark as that first examined; the specific gravity was 1025, and the precipitate under the microscope showed numerous granular and a few epithelial casts and triple phosphate crystals. 2.30 p.m.: The patient seemed very ill indeed, almost bloodless in appearance, the lips being as pale as face; jaundice deeper than on admission; perspiring freely; vomited yellow water, about 4 oz., and passed 6 oz. deep-red urine. 4 p.m.: Temperature 102°; he vomited at 3.30 and again at 4 (2 oz.). 4.30 p.m.: Vomited again, brownish colour; a little more urine. 5.45: Vomited again, bright green, with a small streak of blood. Temperature 99.6°; pulse 124, weak, and a little intermittent. 10 p.m.: Has vomited at intervals all the evening; passed 19 oz. of urine at 9 p.m., a rather lighter red. Examined acid: specific gravity 1020; albumen on boiling, granular casts and triple phosphates as before. Temperature 103.2°. Seems very exhausted; pulse 172, thready.

November 12th. From this he was given champagne every hour, and milk occasionally; he continued to have attacks of vomiting up to 2 a.m., when he had another slight rigor. At 3 p.m. he had general convulsions, and at 4 p.m. a severe vomiting of very bright-green material, and unilateral convulsions, involving the right arm and leg. His brother, who had been with him all night, thinking him *in extremis*, decided to take him home, and removed him in a stretcher to his house near at hand.

RESULT.

At 8.15 a.m. I was called to see him in consultation with two other doctors whom the friends had called in. He was collapsed and dying, only the faintest pulse being perceptible at the wrist. He was noticeably more jaundiced than the previous evening, and the saliva wiped from his mouth was of a deep yellow colour.

He had had no vomiting, nor had he passed any urine since he had been taken from the hospital, nor had any more convulsions. He was unconscious, and in no way responded to the energetic treatment of my colleague's hypodermics of quinine, ergotine, ether, and artificial serum. He died, after I had left, about 10 a.m. No *post-mortem* examination was permitted.

The treatment of the case was on general principles. Quinine, which was at first used, was stopped as soon as the nature of the case was clear. When vomiting became constant, nutrient enemata and champagne were given.

I examined the blood microscopically on November 11th. There was considerable leucocytosis, but the red corpuscles were not markedly altered; they readily formed rouleaux. A few microcytes were noticed. A few red corpuscles contained malarial plasmodia.

A CASE OF MEASLES IN THE PUERPERIUM.

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THE following case of rise of temperature during the puerperium is of interest for two reasons. In the first place, it was due to a definite known cause, and, secondly, it has the very unusual advantage of having a very satisfactory "control."

The patient, a young primipara, and her brother, a boy 6 years of age, were at a place of public amusement on January 28th, 1905—a time when measles was rather prevalent. The following day both showed signs of having caught cold, but as the symptoms completely disappeared in about thirty-six hours nothing further was thought of the matter. Both were out as usual on January 30th and 31st.

On February 1st the lady had a perfectly normal labour, and gave birth to a living healthy son. The infant was nursed, and the puerperium till February 9th was perfectly normal; the temperature never exceeded 98.8° F.; the mammary secretion was slightly diminished after February 5th.

On February 8th the patient's young brother showed marked catarrhal symptoms; by 8 p.m. his temperature rose to 102° F.

On the morning of February 9th the lady's temperature was almost 100° F. The lochial discharge was not offensive in character, nor was it increased in amount; there was no uterine tenderness, nor any other sign of any local cause for the rise in temperature. Having in view the typical condition of the patient's brother, it was concluded that the patient's temperature might be due to the onset of measles—a disease from which she had, so far, escaped. The infant was accordingly weaned, a second nurse was got, and she and the infant were isolated.

Regarding the patient's brother's case, which was practically a "control case," nothing need be said beyond stating that it was a severe case with some ear complications. It ran a normal course; the temperature reached 104.2° F. the day the rash appeared and then fell.

On February 9th the patient's temperature reached 101.2° F. On February 10th the temperature continued to rise, and at 11 p.m. reached 102° F.; the rash now began to appear. On February 11th the rash became general, the temperature was 103.2° in the morning, and by 8 p.m., reached its maximum, 105.2° F., in the mouth; it remained at this point till midnight, when it began to fall. On February 12th the patient had some ear symptoms, and there was a general facial oedema, causing excessive swelling round the mouth and under the eyes. In spite of this the temperature fell to