energies to the elucidation of this problem. Moreover, as the mentality of the native precludes the possibility of accumulating the necessary material at a permanent base, the team would be essentially a roving body, well equipped in every way and furnished with a really efficient travelling laboratory; whilst its activities, including periodical examination of treated cases and the collection of statistics, would extend over a period of about three years.

The personnel would include probably two clinicians and two or three trained protozoologists, assisted by an adequate staff of assistants, and also one or more veterinary surgeons—since it is obvious that, if the method we have put forward fulfils our anticipations, it opens up the possibility that the treatment of the future will resolve itself into the simultaneous intravenous administration of arsenical compounds and the intrathecal injection of salvarsanized serum derived from the horse, ox, or other large animal already experimentally infected and treated.

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GLYCOSURIA OF MALARIAL ORIGIN.

BY

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MALARIA may simulate very many internal diseases, but we are not aware of cases of malarial glycosuria and malarial diabetes having been placed on record; the following two cases may therefore be of some interest.

CASE I.

J. F. S., a demobilized soldier aged 32, was admitted to the Tropical Section of Orpington Hospital on April 13th, 1921, as a case of malaria. He had his first attack in August, 1916, when malignant tertian parasites were found; since then he had had numerous relapses. On admission the temperature was 98.8°, and the pulse 120; the patient was pale and tremulous; he stated that he had lost flesh and complained of great weakness. Physical examination of the chest revealed nothing worthy of note. The spleen was not palpable. No malarial parasites were present in the blood. Differential count: Polymorphonuclears 63, large mononuclears 9, lymphocytes 33, eosinophiles 1. In the faeces no cysts were found. The chemical analysis, kindly carried out by Dr. Lynch at the Central Chemical Laboratory, Ministry of Pensions, Chelsea, gave the following results:

Total solids, 22.1 per cent.			
Fat	...	...	8.6 per cent.
Fatty acid	...	...	2.8 "
Soaps (as fatty acid)	...	...	2.8 "
Total	...	...	14.2 per cent.

The urine, specific gravity 1031, was slightly acid, the amount passed during twenty-four hours was only very slightly increased. It contained sugar 8.5 grams per litre, but no albumin. Acetone and diacetic acid were absent.

On April 14th the patient was placed on a fairly intensive quinine treatment (10 grains three times daily). The sugar present in the urine decreased rapidly after twelve days' treatment, but there was still a quantity sufficient to reduce Fehling's solution and to give phenyl-glucosazone crystals, though not enough to estimate. The patient was then given, in addition, a course of six injections of quinine hydrochloride intramuscularly of 15 grains each. On May 10th the quinine hydrochloride injections were discontinued, and quinidine was given in their place (30 grains daily).

The urine was examined again on May 13th, 16th, 17th, and 18th, and did not give any reduction of Fehling's solution, nor did it give phenyl-glucosazone crystals.

At no time was the patient put on antidiabetic diet, and there was no restriction of his carbohydrate intake. There can be little doubt, therefore, that the disappearance of the sugar was due to the administration of quinine.

When the patient was admitted to hospital he was feeling very weak, and complained of having lost a great deal of flesh. There was, however, no polyuria, no boulimia, no thirst. On

May 20th, 1921, the general appearance of the patient had much improved, and he expressed himself as feeling better than he had done for years.

CASE II.

Mr. E. N., aged 44, married, with no family history of diabetes, consulted one of us in March this year. During the war he served in one of the allied armies, and in 1916 he contracted, while in the Balkans, a severe malarial infection; he had several relapses, the last being on December 19th, 1920. In January, 1921, he noticed that he was feeling more hungry and thirsty than usual, was passing much more urine than normal, and was losing flesh. He consulted a medical man, who found a fairly large amount of glucose in the urine (2 per cent.), and placed him on a very strict diet, which induced only a slight decrease in the amount of sugar present in the urine. When the patient consulted one of us in February the urine contained 1.5 per cent. of glucose; it was acid, specific gravity 1032, no albumin, and acetone and diacetic acid were absent. The amount of urine passed during the twenty-four hours averaged 6 pints.

The patient looked rather emaciated and very anaemic; the skin was of a pale, earthy colour, with patches of hyperpigmentation resembling chloasma, so often seen in cases of chronic malaria. His spleen was very slightly palpable and very hard. The examination of the blood did not show any malarial parasites, but there could not be any doubt clinically that he had chronic malaria, and the diagnosis was made of "diabetes in a malarial subject." He was advised to continue the strict diet he had been having for the glycosuria, and in addition to take 10 grains of quinine three times daily for his malaria. He came back three weeks later feeling much better; the enlargement of the spleen had disappeared, and—a most interesting feature—the amount of sugar had decreased enormously, being less than 0.1 per cent. We came to the conclusion that it might be a diabetes syndrome of malarial origin, and suggested to the patient that he should go back to ordinary diet but continue the quinine. He came to see us regularly once a week and the sugar did not increase; only a trace was present. During March the patient went to the South of Europe on business, and during all the time he was away (four weeks) did not take any quinine. He came back to this country in April, and three days after arrival, after playing golf in the rain, had a shivering fit, followed by very high fever, which ended in profuse sweating. The spleen again became palpable and hard, and examination of the blood showed the presence of a few rings of malignant tertian. The urine was examined after the temperature had come down to normal; it contained 1.2 per cent. of sugar. The patient was placed on an intensive quinine treatment by the mouth and intramuscular injections for six weeks without any dieting; not only did the symptoms of the chronic malarial infection disappear, but the urine became completely free from glucose, when examined by the usual methods of analysis (Fehling's, Nylander, phenyl-hydrazine, fermentation test).

CONCLUSION.

Our observations tend to show that there is a form of glycosuria of malarial origin, and that this condition may at times (Case No. 2) become so severe as to simulate true diabetes, the patient losing flesh, becoming very weak, complaining of thirst and hunger, and passing a large amount of saccharine urine. The glycosuria in our two cases was cured by the administration of quinine in full doses without any dietetic treatment.

SLOUGHING OF THE LOWER UTERINE
SEGMENT FOLLOWING PLACENTA PRAEVIA:
LAPAROTOMY: RECOVERY.

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A MARRIED woman, aged 27, was admitted to Addenbrooke's Hospital on January 22nd, 1921. She had been delivered, on January 1st, of her second living child, but not without complications. Twenty-four hours previous to delivery she had a sudden severe haemorrhage and had called in her doctor, who, although he was unable definitely to diagnose placenta praevia, had strongly suspected it. He immediately ordered her to bed with instructions to remain there. She failed to carry out these orders, but remained up and did her work for the twenty-four hours previous to the birth of the child, suffering from no further haemorrhage. The delivery of the child was uncomplicated and occurred rapidly after two or three pains and before the arrival of her doctor. The placenta however was retained, and was removed digitally and without anaesthesia one hour later. It was found to be very adherent to the uterine wall.

All apparently went well during the first eight days of the puerperium, after which an irregular pyrexia developed. On January 16th (the fifteenth day of the puerperium) the patient had a severe haemorrhage, and this persisted on and off until she was urgently admitted to hospital. She had no vomiting, and complained of little or no pain. Constipation was present, but was corrected by purges and enemata. On the day preceding the haemorrhage she had had diarrhoea, which had been checked with paregoric, and ergot and nuxvomica had previously been given her by mouth.

I saw her on January 22nd, at 10 p.m., when the history suggested the retention of some products of conception which had become septic. She was exceedingly anaemic, with a small running pulse of 120 per minute, and respirations 24 per minute. She was wasted, and stated her inability to take much nourishment. Although complaining of little or no pain, she had the "abdominal type" of facies. The abdomen was soft and moved well, but there was slight tenderness over the brim of the pelvis. On vaginal examination it was found that the uterus was not particularly enlarged or tender, but there was an offensive blood-stained discharge.

Operation.

Under anaesthesia I explored the uterus. The os readily admitted the examining finger, which, when passed within the uterine cavity, came upon a firm transverse ridge of tissue, which at first I thought was a fragment of placenta. (It afterwards proved to be the lower edge of the normal posterior uterine wall.) Immediately above and posterior to the internal os I was struck by the softness and lack of resistance of the tissues. With a pair of blunt ring forceps portions of soft and blackened tissue were removed, which were thought to be detritus from the placental site.

In the same situation some tissue was grasped in the forceps which did not come away. Suspecting the possibility of its being a loop of bowel, I drew it down as far and as carefully as possible, and by means of special illumination demonstrated that my suspicion was only too well founded. Fearing that I had perforated the uterus, and in spite of the patient's desperate condition, I decided that the only course was to explore from above and, if possible, to remove the uterus. I divided the parietes by the ordinary mesial incision, but found the peritoneum adherent to underlying structures. The adherent coils of intestine were carefully separated and a condition of firm plastic peritonitis was found gluing all structures to one another including uterus, appendages and bladder. On further separating the coils covering Douglas's pouch, I came down on a large collection of foul-smelling pus. This was sponged out and the coil of small intestine which had been seen *per vaginam* was drawn up. It was found to be firmly adherent at both ends deep down in the pelvis, and over an area measuring 3 in. by 2 in. on the side opposite to the mesenteric attachment the serous coat was eroded away, leaving the submucous coat exposed. Owing to adhesions it was found impossible to resect this. The hole in the back of the lower uterine segment was explored and cleaned as much as possible, and the appendix, which was very oedematous and lying in the abscess cavity, was removed. Owing to the generalized adhesions in the pelvis (general peritonitis was absent) and the grave state of the patient nothing further was attempted. A large tube was passed down to the bottom of Douglas's pouch and the wound closed. A piece of thin-walled colotomy tubing was inserted *per vaginam*, through the os uteri. This was removed twelve hours later, having apparently not assisted drainage to any great extent.

After-History.

During the next twenty-four hours the patient held her own and complained of little pain. The temperature fell to normal and the pulse remained about 136 per minute. She was kept on brandy by the mouth and camphor subcutaneously, and managed to take slop food in the absence of vomiting.

Forty-eight hours after operation the temperature rose to 101°, and this nocturnal rise persisted till the fifth day, when the temperature rose to 103°. Douches of Dakin's solution were given night and morning, and the abdominal wound continued to drain freely, at first emitting foul material indistinguishable from faecal content.

On the third day, by means of calomel gr. 1/3 given four-hourly, the bowels acted well and continued to do so regularly during her recovery. By the end of the second week the temperature had fallen almost to normal, the patient was taking her food well, and the abdominal wound was cleaning and tending to close. Vaginal discharge had almost ceased. At the end of the fifth week she was sent home, the abdominal wound having closed and the patient's condition being most satisfactory. Her doctor has now been to see her, two and a half months after operation, and reports that she is fairly well and has had no need to call him in.

The case would appear to be one of infection of the placental site, aided possibly by its low position in the uterus, followed by a gradual sloughing of the uterine wall at the placental site, and secondary haemorrhage therefrom. A coil of small intestine lying in Douglas's pouch became adherent and eroded, and the infection spreading produced pelvic peritonitis and pelvic abscess.

Although the bacteriology of the case was not worked out, the strong presumption is that it was an infection by *B. coli communis*, and it is surprising that the patient withstood and survived a condition so desperate.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL

ENCEPHALITIS LETHARGICA WITH SEVERE HICCOUGH AT THE ONSET.

A SINGLE man, aged 27, had, in the course of his business, to visit ships at the London Docks. On the morning of December 11th, 1920, he felt quite well; at mid-day, on his way home from the docks, hiccough began, and was so persistent that at the end of two hours he consulted a doctor. Dr. Frazer, who saw him at this time, writes that he had "severe hiccoughs and general malaise." He then began to vomit about every quarter of an hour. Hiccough and vomiting persisted till 4 the next morning (a period of sixteen hours) when he went to sleep. Both symptoms disappeared and have not recurred.

For the next twelve days he remained in bed; he had headache and felt feverish, but did not feel sleepy. He travelled to his home at Cardiff on December 23rd; seeing everything double. On that evening he was seen by Dr. Piddian (to whose kindness I am indebted for the notes of his condition). He then had a temperature of 102°F., was very tremulous, and delirious at times. A few days later he imagined he was being kept confined in a cabin on board ship, and became so violent that he had to be placed under restraint. Three days later he was quiet and became very sleepy. I saw him on January 17th, 1921. He was then stuporous and complained of diplopia. His face showed the typical Parkinson's mask. There was no squint or nystagmus, the pupils were equal and reacted to light, and the fundi were normal. The limbs were rigid and coarse regular tremor of the left arm was present. The reflexes were normal, except that both abdominal reflexes were absent, and that left-sided extensor toe response was obtained by the methods of Oppenheim and Gordon, but not by stroking the sole of the foot, the latter producing flexion.

He gradually got better. At the beginning of April (three months after the onset) he noticed twitchings of the left shoulder which later spread to the right. These have persisted. The muscles involved are the trapezius, sternomastoid, latissimus dorsi and pectorals. The contractions are coarse, of regular rhythm (often synchronous with the pulse beats) about 70 to the minute. They are most liable to come on in the evening and keep him awake. They disappear during sleep, but are always present when he awakes in the morning. The left side is more affected than the right.

For some months he has had to use a magnifying glass for reading, but now (six months after onset) he can read Jaeger 2 and Jaeger 4, though only for a short time; the pupils respond only slightly to a bright light. Nystagmus is present. The reflexes have become normal, and the rigidity of face and limbs has disappeared. While quite clear mentally, there remains a certain intellectual stiffness.

Concurrent epidemics of hiccough and encephalitis lethargica have been reported during the last two years, in all parts of the world, and more especially in France. Sicard and Dufour¹ have published cases, connecting "epidemic hiccough" with encephalitis of the myoclonic type. The course of an attack of epidemic hiccough is usually as follows: After a prodromal period lasting three or four days, during which lassitude, headache, general pains and slight fever are present, severe hiccough appears. It may occur in "rhythmic crises," lasting fifteen to ninety minutes, with similar intervals of rest, or may be continuous. It usually lasts for two to four days, but may persist for ten. It may be complicated by bouts of vomiting. Spasms occur at the rate of six or eight to the minute. The onset and termination are usually sudden. It is reported as being common in males and rare in children.

Hiccough occurring as a prominent symptom has been described in connexion with outbreaks of influenza in this country; isolated cases of "spasmodic" hiccough have also been published,² but I do not know of any recorded case of severe hiccough of the epidemic type directly followed by undoubted encephalitis lethargica, as in the case here recorded. French authors have regarded these epidemics of hiccough as a mild form of the graver disease.

It may be objected that the small number of cases in which epidemics of hiccough and encephalitis lethargica have been clinically connected is no greater than can be