experience, and one occurring in my own experience and produced by haemorrhage from a fracture of the base of the skull running through the posterior fossa has already been mentioned.

The cause of the syndrome would appear to be the relative vulnerability to pressure of the respiratory as compared with the cardio-inhibitory centre. The compression may be produced by haemorrhage into the posterior fossa or by an intracranial tumour or abscess, and in the latter instances may result from oedema in association with the lesion. This was apparently the mechanism in the case which follows. Recognition of the syndrome enabled one to relieve the condition and subsequently deal with its cause, a cerebellar cystic glioma.

### HISTORY OF CASE

The patient, a single woman aged 30, was transferred to the surgical unit from another hospital with a diagnosis of cerebellar tumour. The signs and symptoms were typical of such a lesion, and there was a high degree of bilateral papilloedema. A ventricular estimation had been carried out, and she was in one of the unit wards awaiting operation when one morning I had an urgent message from the house-surgeon (Mr. J. F. Meredith), asking me if I would come and see her as she had suddenly collapsed. On reaching the ward I found him carrying out artificial respiration. The patient was unconscious, and as soon as the efforts at artificial respiration were relaxed she would become cyanosed, the pulse would slow and eventually stop, and she would be to all appearances dead ; but with renewed artificial respiration the pulse would come back, the cyanosis pass off, and her colour improve.

With a knowledge of the outlook—namely, indefinitely prolonged artificial respiration and then death when exhaustion of the workers led to cessation of artificial respiration—it was realized that radical action was imperative. An oxygen cylinder and a gum-elastic catheter were sent for. By manipulation the catheter was placed through the glottis ; on connecting it to the oxygen supply the colour of the patient was kept pink and the pulse remained good. She was taken at once to a theatre, and without any anaesthetic and with the assistance of my colleague Mr. R. V. Cooke and of Mr. Meredith a suboccipital decompression consisting of the removal of the greater part of the post-occipital bone back to the foramen magnum so as to remove its posterior margin, and a laminectomy of the atlas, were carried out. When the dura mater was opened the cerebellar tonsils bulged into the wound, and the medullary compression was evidently relieved. At this stage respiratory movements commenced ; shortly afterwards the oxygen supply was turned off, and it was found that the catheter could be withdrawn from the trachea. The wound was closed and the patient returned to bed. She regained consciousness and recovered from her alarming experience with no untoward effects except for a small cerebrospinal fluid fistula, which, however, soon closed. The decompression relieved her symptoms to a great extent, and she was discharged to her home and was comparatively well for eight months. She was then readmitted and the cyst removed from the cerebellum, but unfortunately she contracted bronchopneumonia, from which sixteen days after operation she died. The interest in the case here, however, lies in the appearance of the posterior fossa compression syndrome and its treatment by emergency posterior fossa decompression.

### COMMENT

By prompt radical treatment it may be possible to tide the sufferer from its effects through an otherwise fatal syndrome, and it would appear that, when in a case of raised intracranial tension—whether from haemorrhage, new growth, or abscess—respiratory failure occurs, prolonged efforts at artificial respiration should not be carried out, but with all possible dispatch relief of medullary compression should be secured by an emergency posterior fossa decompression. Oxygen administered through an intratracheal catheter, as in this case, may enable the operation to be undertaken ; but, failing introduction of a catheter in this way, an emergency tracheotomy or laryngotomy would appear to be advisable and the oxygen should be supplied by this route. It might be objected that, rather than the performance of such a radical procedure, a reduction of intracranial tension with relief of medullary compression might be brought about by ventricular tap or by intravenously administered hypertonic salt solution or glucose ; but such means would appear less certain, and in any case would produce only transitory effects. In the posterior fossa compression syndrome, therefore, the indication would appear to be an extensive mechanical relief of pressure, as in the case to which reference has been made.

## A CASE OF MALARIA DUE TO PLASMODIUM OVALE STEPHENS 1922

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Stephens (1922) reported a new malaria parasite in a man coming from East Africa, and named it *Plasmodium ovale* on the basis of its morphological features in the asexual stage, pointing out that it resembled rather closely the quartan parasite, but that infected corpuscles were frequently oval in shape and showed Schüffner's dots. Later, Stephens and Owen (1927) described another instance of infection with *P. ovale* in a case from Nigeria ; male and female gametocytes were on this occasion described which were indistinguishable from those of quartan except for the stippled, decolorized margin of the red cell ; they concluded that the relationship, if any, of *P. ovale* to *P. malariae* remained to be demonstrated.

Warrington Yorke and Owen (1930) met with an infection with *P. ovale* in a hospital patient from Nigeria, and by means of direct blood inoculation passed it through a series of five general paralytics or tabetics. The parasites were morphologically indistinguishable from *P. malariae,* but the cycle of schizogony was completed in forty-eight hours, and the temperature charts were tertian in type. The infected red cells were moderately enlarged, and occasionally enormously so ; they were pale, fragile, frequently oval in shape, having usually an irregular, raggy outline, and being heavily stippled, as described by Stephens.

James, Nicol, and Shute (1932) obtained blood containing *P. ovale* from a case of Professor Warrington Yorke's, passaged it through fourteen general paralytics, and studied the sexual form in the mosquito ; the arrangement of pigment in young oocysts was found distinctive. The disease was also transmitted by the bite of experimentally infected mosquitos, *Anopheles maculipennis,* as well as by sporozoites obtained from the salivary glands and injected intravenously. Later the same authors (1933) reviewed the whole subject, and concluded that the nomenclature of *P. ovale* was correct.

Since 1922 only four naturally infected cases appear to have been diagnosed in this country, and for this reason the following case, which presents several features of interest, seems worth recording.

### CASE RECORD

The patient, a male aged 28 years, was sent by his firm on August 8th, 1932, for examination regarding his suitability for the Tropics, and following a favourable report proceeded to West Africa, arriving at Lagos on September 19th, 1932, and leaving Freetown on February 9th, 1933, for England. During this period of 143 days he visited Nigeria, the Gold Coast, Gambia, and Sierra Leone in this order, and was quite well except at the end of October in Lagos, when he was severely bitten by sand-flies, and suffered from an attack of

fever lasting two days. Throughout the tour he used mosquito boots, a mosquito net at night, and took quinine (five grains daily) as a prophylactic measure. At no time was he conscious of being bitten by mosquitos.

The patient arrived in England on February 20th, 1933, and was quite fit until March 22nd, when at about 7.30 p.m. he developed a rigor lasting half an hour; this he attributed to a chill. The second attack came three days later—that is, March 25th—when a rigor occurred at 7.20 p.m. and lasted one and a half hours. The third attack occurred on March 27th, about 6.30 p.m., and by 8 p.m. the temperature had risen to 103.8° F.; on this occasion the rigor lasted two hours, and the patient sweated freely after taking hot drinks. The fourth attack, on March 29th, came on at 6.30 p.m., with a rigor lasting three-quarters of an hour; by 7 p.m. the temperature was 101°, and again profuse sweating occurred. On each occasion the patient said the rigors were of a mild character, and that the shivering could to some extent be voluntarily controlled.

On March 30th the patient was referred to me by Dr. R. H. Dixon with suspected malaria. Examination then revealed a palpable spleen, a temperature of 98.8°, a yellow skin, and a dark-brown urine, which showed urobilin (+) and a trace of bile salts and albumin. The leucocyte count was 7,200 per c.mm.; the differential count showed neutrophils 58 per cent., lymphocytes 30 per cent., monocytes 11 per cent., eosinophils 1 per cent. In blood films prepared with Leishman's stain the laboratory reported that malarial parasites, atypical in type and resembling quartan malaria, were present. Owing to the fact that the temperature, though originally quartan, now showed a tertian type of periodicity, infection with *P. ovale* was suspected on clinical grounds, and this was confirmed by Dr. C. M. Wenyon, who re-examined the blood films. Dr. Wenyon reported that the morphological appearances of the parasite were typical of *P. ovale*, that the characteristic distortion of the red cells was present, and that the infected corpuscles were heavily stippled with Schüffner's dots in films stained overnight with Giemsa, though they were not evident in Leishman-stained films.

Next day (March 31st) the patient was admitted to the Hospital for Tropical Diseases. The temperature reached 99.4° at 6 p.m., and blood films again revealed *P. ovale*. The red blood cells were 4,720,000 per c.mm.; haemoglobin 90 per cent.; colour index 0.96; diameter 7.2 $\mu$. The urine showed bile salts (+), urobilin (+), and a trace of bile pigment, while the van den Bergh reaction gave a negative direct but a strongly positive indirect reaction of 2.5 units. Oxalated plasma collected under paraffin showed no trace of oxyhaemoglobin bands when examined spectroscopically through a thickness of 1.3 cm. The plasma urea was 38 mg. per cent., and the plasma cholesterol 109 mg. per cent. Treatment was commenced with atebrin, 0.1 gram being given per os. Next day (April 1st) there was malaise, the temperature reached 100°, but there was no rigor. Atebrin was continued, 0.1 gram three times daily, and quinine bihydrochloride, 7½ grains in 10 c.cm. of saline, was injected intravenously.

The subsequent clinical course was afebrile and uneventful, and parasites permanently disappeared from the blood. Intravenous injections of quinine (7½ grains) were continued daily until April 4th, and atebrin (0.1 gram three times daily) until April 5th. The urine had by then lost its dark-brown appearance, and though bile salts persisted only a trace of urobilin was detected; the van den Bergh reaction was also found to be normal—indirect reaction 0.2 unit. The reticulocytes equalled 3.6 per cent., and from this date until April 8th the daily reticulocyte counts were 5.7, 6.3, and 3.4 per cent. respectively. On April 10th the reticulocytes had fallen to 2.5 per cent.; red blood cells, 5,120,000 per c.mm.; haemoglobin, 102 per cent.; colour index, 1.0; average corpuscular diameter, 7.2 $\mu$; leucocytes, 8,300 per c.mm.; the differential count was normal, the monocytes having fallen from 11 to 3 per cent. By April 11th the reticulocytes had reached a normal level—that is, 1 per cent.; the urine was also normal, the spleen was no longer palpable, and the patient was discharged from hospital.

When seen again, on April 22nd, clinical examination revealed no abnormality, the blood showed no evidence of parasites, the van den Bergh reaction was normal, and the urine contained neither urobilin nor bile salts.

### COMMENTARY

This case is an instance of infection with the fourth species of malaria parasite—that is, *P. ovale* Stephens 1922—contracted during a short tour of 143 days in West Africa. Throughout this period the patient had consistently taken prophylactic quinine, and this is the probable reason for the long incubation period, unless the two days' fever in Lagos be interpreted as representing the primary malarial attack.

The species of parasite was suspected on clinical grounds when the laboratory reported atypical quartan malaria in a febrile patient who, though at onset manifesting quartan, later showed a tertian periodicity. Another peculiar feature was the late time of onset of the rigors, which occurred between 6.30 and 7.30 p.m.; generally malaria fever commences before or about midday, as emphasized by Manson.

The biochemical and haematological observations were of interest. Haemoglobinaemia was not evident at the time the plasma was examined, but the urobilinuria and hyperbilirubinaemia indicated that the blood destruction usually associated with malarial fever was going on. Bile pigments in the urine are unusual in malarial fever, and probably originated from a mild toxic hepatitis in this instance. The reticulocytosis reached a maximum of 6.3 per cent. some eight days after the initiation of specific treatment. This is usual in malaria, for once the parasites are controlled or destroyed regenerative activity of the bone marrow results in reticulocytosis and new blood formation.

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# Memoranda

## MEDICAL, SURGICAL, OBSTETRICAL

### RECURRENT RENAL CALCULI

#### NEPHROLITHOTOMY TWICE ON EACH KIDNEY IN THE SAME PATIENT

Mrs. X, then aged 37 years, was sent to me in February, 1911, by the late Dr. Sortain Hancock, with definite signs and symptoms of stone in her *right* kidney. Radiographic examination proved the presence of a large branched calculus occupying the pelvis and some of the calyces of that organ.

I performed nephrolithotomy on February 15th, and removed, as I thought whole, a large stone with four "legs." The wound was drained for the first forty-eight hours, and then healed rapidly, and she was discharged on February 25th, ten days after the operation. There was no evidence then of any calculus in her left kidney.

She was readmitted on April 25th, 1914, now with definite evidence of a stone again in her *right* kidney, and one also in her *left* kidney. On May 1st, 1914, I explored her *left* kidney, the one which had not previously been exposed, and removed a stone. The wound healed rapidly. On May 19th, 1914, I explored her *right* kidney, the one from which I had removed the large stone in 1911, and found and removed a small stone from the pelvis. This may have formed upon some debris which I had failed to extract at the first operation.

Two years later, in March, 1916, she again showed herself with evidence of stone, once more in her *left* kidney. By this time she had become an "expert" in the diagnosis of renal calculi, and she ventured to bring her brother, in whom she was sure there was a stone in his *left* kidney! And so it turned out to be. I admitted them both to the hospital on the same day, I operated on