patency of the airway is maintained by positioning of the patient's head or by the insertion of a soft rubber oral airway (Guedel type); or a short nasopharyngeal tube is passed through the nose. We have found it beneficial to employ supplementary nitrous-oxide-oxygen anaesthesia (see below).

The patient is now transferred to any desired position on the operating table, and one arm is firmly bound to a splint, the unpadded end of which is inserted beneath his shoulder. The tourniquet is applied and the needle inserted into a suitable vein. With the drip turned off, the venous congestion produced by the tourniquet causes blood to diffuse back into the indicator—proof of the intravenous position of the needle. The drip is now started (40 to 50 drops a minute), and the needle is fixed in position by adhesive tape. We have found that the needle is held more securely and lateral movement prevented if a small piece (1 in. by $1\frac{1}{2}$ in.) of tape is placed beneath the needle with the adhesive side uppermost. Two longer pieces are then applied over both needle and tape, securing them to the surrounding skin (Fig. 4).

FIG. 4

Further injections of 5% solution of pentothal are made, as required, by syringe and needle into the pressure tubing, which should be pinched on the drip-bulb side during the injection to prevent "backwash" and consequent delay in the passage of the drug to the patient.

Advantages

Positive-pressure drip apparatus eliminates the cumbersome unstable stand required by gravity drips, and as the saline reservoir can be placed quite near the vein, long lengths of tubing are avoided.

The glass aspiration indicator facilitates venepuncture.

Should the needle become dislodged between the injections of pentothal, saline only will be introduced into the subcutaneous tissues. In other methods (Rivett and Quayle, 1940; Macintosh and Pask, 1941) irritant pentothal may so escape.

Accurate doses of pentothal can easily be injected and their full effect quickly assessed. The same concentration of the drug (5%) is used throughout, thus avoiding error due to different solutions.

The absence of Y-tubes joining separate containers of saline and pentothal (Rivett and Quayle, 1940; Thomas, 1942), syringes, multi-directional taps (Rivett and Quayle, 1940; Semans, 1941), and numerous lengths of tubing lessens the weight attached to the arm and hence there is less likelihood of the needle becoming dislodged. More important is the lack of "dead space" (Adams, 1941) in the apparatus we describe; hence there is minimal delay in action of the drug due to "backwash" and uncontrollable dilution.

Supplementary Anaesthesia or Oxygen

Organe and Broad (1938) reported a series of cases in which supplementary nitrous-oxide-oxygen anaesthesia was used. Various other writers have advocated supplementary oxygen (Carraway, 1939; Carraway and Davison, 1941; Davison, 1943). In endorsement of these views we have found that supplementary nitrous-oxide-oxygen anaesthesia is of value because: (1) The total dosage of pentothal required is considerably smaller, hence the degree and duration of post-operative respiratory depression are reduced to a minimum. (2) A continuous visual indication of the patency of the airway and the volume of respiratory exchange is provided by the movements of the rebreathing-bag. (3) The percentage of oxygen can be increased greatly in excess of that found in atmospheric air, thus overcoming anoxia due to shallow respiration. (4) Rhythmic pressure on a rebreathing-bag filled with oxygen provides the most efficient form of artificial respiration should apnoea occur.

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A FATAL CASE OF CEREBRAL MALARIA

BY

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Cerebral malaria is a rare cause of coma in this country, but with the return of large bodies of our troops from areas where subtertian malaria is endemic this complication may be seen more frequently. It is a medical emergency which often responds well to early treatment, but its gravity can be judged by the mortality of 40% recorded by Sandford, Crawford, and Warr (1940) in a series of 48 cases. The following description of a fatal case is presented as a reminder that malaria should be considered in the differential diagnosis of coma and that, unless treatment is started early, gross irreversible pathological changes occur in the brain.

Case History

On Sept. 2, 1942, a 28-year-old stoker was admitted to a naval hospital in a semi-comatose state, having been sent from a destroyer whose previous travels were not divulged. He had been suffering from a severe headache for three days, but had not reported this as he had not wanted to miss going on leave. On the morning of admission he had reported sick, complaining of a severe headache, frequent vomiting, and fever. No abnormal physical signs had been found by the medical officer of the destroyer. The patient had lapsed into a semi-comatose state during his journey from the ship to hospital.

On examination he was found to be semi-comatose, but was easily roused by a loud voice or a pin-prick; he was not able to speak, and was incontinent of urine. T. 103, P. 120, R. 28. The skin was dry and muddy-coloured but burning hot. The teeth were tightly clenched. The pupils were contracted and slightly irregular, and reacted sluggishly to light; the fundi showed no abnormality. There was no paralysis of the limbs, and the muscle tone was normal. The deep reflexes were all present, but were sluggish. Abdominal reflexes were present. Plantar responses were flexor. There was no neck rigidity or positive Kernig's sign. A few moist sounds were heard at the base of the right lung. Heart sounds were normal. B.P. 110/70. The abdomen showed no rigidity and the liver and spleen were not palpable.

In view of the possibility that he might have a meningococcal septicaemia he was given 1 g. of sulphapyridine intramuscularly while the following investigations were being carried out:

Urine: Specific gravity 1014, acid; a heavy cloud of albumin, blood-positive chemically, numerous red blood corpuscles and granular casts seen. *Blood:* Urea, 42.1 mg. per 100 c.cm.; sugar, 85 mg. per 100 c.cm.; leucocyte count, 14,200 per c.mm. (polymorphonuclears 77%, lymphocytes 12%, large mononuclears 11%). *C.S.F.:* Lumbar puncture revealed clear fluid under normal pressure, containing 3 cells per c.mm., no excess of globulin, 50 mg. of protein and 92.8 mg. of sugar per 100 c.cm.

In view of the negative findings it was thought that the diagnosis lay between septicaemia and cerebral malaria. Blood films were therefore taken; numerous ring forms of *Plasmodium falciparum* were found, but no crescents. An intravenous 5% glucose-saline drip was started immediately. 10 gr. of quinine dihydrochloride together with 10 min. of 1/1000 adrenaline hydrochloride was dissolved in 20 c.cm. of water and given slowly by a hypodermic syringe into the rubber tubing of the drip apparatus. The intravenous glucose-saline was continued throughout the night, and on the morning of Sept. 3 the patient was fully conscious and able to recognize his relatives. His general condition seemed much improved (T. 99.2°, P. 100), but persistent vomiting prevented the administration of fluids by mouth. A further dose of 10 gr. of quinine dihydrochloride was given in the same manner as before. His condition continued to improve during the day. A blood count showed 4,400,000 red cells per c.mm.; haemoglobin 100% (Haldane); and 18,000 leucocytes per c.mm. On Sept. 4 his temperature rose to 104°; his appearance was more icteric, and he again lapsed into coma. Lumbar puncture was repeated and 10 c.cm. of clear fluid was removed. 10 gr. of quinine was given intravenously and a slow-drip blood transfusion was started. No improvement resulted from these measures, and by the morning of Sept. 5 his condition had greatly deteriorated. The blood urea was 118 mg. per 100 c.cm. His temperature had risen to 105°, and despite frequent tepid sponging it remained between 104° and 105° until death occurred on Sept. 6.

At necropsy the salient features were as follows. Brain: Congestion of the vessels on the surface of the cerebral hemispheres; numerous petechial haemorrhages present throughout the brain, but more so in the cerebral hemispheres than in the brain-stem. Spleen: Enlarged to twice normal size, dark, soft, and flabby; pulp purplish

grey in colour, with a semi-fluid consistency and general loss of structure. The liver was also enlarged, and showed a purplish-grey friable cut surface. Hypostatic congestion was present at the bases of both lungs.

Dr. W. W. Woods, Consultant Pathologist to the Navy, reported on sections of the brain, spleen, liver, and kidney as follows:

"In the two pieces of brain many of the capillaries contain numerous minute nodules of pigment which is negative to Perles's test for iron; this is in keeping with its being malarial pigment. Further, the general configuration of the numerous well-defined dots of pigment is strongly suggestive of malaria. Some of the pigment appears to be in red blood corpuscles, where it ought to be, but much of it seems to lie free in the lumen of the capillaries, perhaps because the corpuscles containing it are very indistinct. Also, I found it impossible to stain the bodies of the parasites themselves, in which the pigment is presumably contained; in a very few instances I thought I could see the faintly stained body of the parasite around the dot of pigment. I can only presume that this failure to demonstrate the parasites is due to post-mortem degeneration or poor fixation, or both. In the white matter of the piece of cerebrum there are numerous petechial haemorrhages, some of them obviously perivascular and some of them ring-haemorrhages; there are very few in the grey matter and a considerable number in the more heavily myelinated part of the other piece of brain, whose site I fail to recognize. In spite of the failure to show the bodies of the parasites, I think there can be no doubt but that this is a specimen of cerebral malaria.

"There is abundant pigment in the spleen and—which I think is more significant—it is abundant in the Kupffer cells in the liver and free in the sinusoids, and is negative to the test for iron. There is very little pigment in the glomeruli of the kidney. I could not see the actual parasites in these three tissues."

Commentary

Lack of knowledge of the movements of the man's ship added to the difficulty of diagnosis in this case. It was afterwards ascertained that he had been in Sierra Leone two weeks before he was taken ill. The diagnosis was reached largely by exclusion, but on reviewing the presenting symptoms and signs they do fit in well with those of other cases which have been described. The comatose state with no abnormal signs in the central nervous system, the muddy sallow complexion, the moderate leucocytosis with a raised monocyte count, are all commented on by Sandford, Crawford, and Warr (1940). In the present case one striking feature was the pin-point pupils.

Differential Diagnosis.—The differential diagnosis includes, of course, all the causes of coma, but those particularly apt to cause difficulty are:

1. *Alcoholism.* The drowsy irrational state with slurred speech found in the early stage of cerebral malaria is very suggestive of alcoholism, as has been noted by Minett and Bernfield (1937).

2. *Meningitis,* in particular the fulminating meningococcal infections, may also be simulated. Wright (1941) stresses the fact that lumbar puncture should be carried out in every case, as cerebral malaria may cause neck rigidity, and the finding of *P. falciparum* in the blood does not exclude a coincident meningitis.

3. *Cerebral haemorrhage, thrombosis, or embolism* may also be mimicked by cerebral malaria. Embolic phenomena may be produced by malarial parasites.

The conclusive proof of the diagnosis in this country is the finding of *P. falciparum* in the blood. Usually an infection severe enough to cause coma is a heavy one, and a blood slide will show numerous parasites. A few cases, in particular those with an embolic onset, may have only a light infection, and parasites in the peripheral blood may be difficult to find.

Treatment.—Treatment should not be delayed while further diagnostic procedures such as sternal puncture are carried out. If the history and physical signs are suggestive quinine should be given at once. This may be a life-saving measure, and is helpful in diagnosis if a favourable response is obtained.

The fatal outcome in this case was disappointing, particularly after the dramatic response to quinine in the first 24 hours. However, it serves to impress the fact that if treatment is to be successful it must be prompt. The delay in treating the malarial infection before the complication of coma supervened was due to the patient's own foolishness in concealing his illness: in most cases malaria should be diagnosed and treated before pernicious complications occur. In this case quinine dihydrochloride was administered solely by the intravenous route. The dosage used—i.e., 10 gr. daily—was higher than

the 5 to 7 gr. advocated by Manson-Bahr (1942). No toxic reactions were seen that could be attributed to the intravenous quinine, but care was taken to give it slowly through a hypodermic needle into the intravenous drip apparatus. Adrenaline hydrochloride, 10 min. of 1/1000 solution, was always given at the same time. Intramuscular quinine is advocated in these cases by Wright (1941), but he does not give any reason for avoiding intravenous injection. Fawcitt and Walters (1941) recommend a transfusion of reconstituted blood serum as a detoxicating and stimulant measure, and Manson-Bahr (1940) recommends blood transfusion if there has been great blood destruction. Lumbar puncture, with removal of 10 c.cm. of cerebrospinal fluid, had no effect in relieving the coma in the late stages of this case, but in view of the marked pathological lesions of the brain found at necropsy this is not surprising.

I wish to express my thanks to the Senior Medical Officer of this hospital for permission to publish the case, and to Dr. W. W. Woods, Consultant Pathologist to the Navy, for his report on the pathological sections.

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Medical Memoranda

Suppurative Mastoiditis in an Infant

I have been much impressed by Mr. P. W. Leathart's paper entitled "A Common Cause of Diarrhoea, Vomiting, and Dehydration in Infants" (Aug. 7, p. 168). It struck me as having been based on sound reasoning and wide experience in a field apparently little recognized, but one of considerable importance. Although parenteral infection is universally known to be an important cause of "D. and V." in infants, only too often it gives place to the diagnosis of gastro-enteritis after the more easily detectable and better-known sites of infection have been excluded. I would like to cite the case of an infant recently admitted to the Southend General Hospital. Besides being a practical example of the point made above, this case tests the truth of most of the criteria laid down by Mr. Leathart for enabling a diagnosis of suppurative mastoiditis to be reached in the ward instead of in the post-mortem room. It is to be regretted that the diagnosis suspected in this patient had to be confirmed in the latter situation instead of on the operating-table. It may be hoped, however, that the lesson learned has helped to offset a fatal end-result.

HISTORY OF CASE

The baby was first seen on July 27, 1943, with a history of vomiting after feeds since birth. Apart from rapid delivery, the previous history was negative. His birth weight was 6½ lb. and he was breast-fed. Stools were "loose" but otherwise normal. Examination revealed a healthy infant, weighing 7 lb. 14 oz. No abdominal mass was felt, nor was peristalsis seen. He was admitted for observation and weaning, and was discharged a fortnight later, having gained 1 lb. and having been taking five milk feeds daily without vomiting.

He was readmitted on Oct. 30, aged 4½ months. The mother stated that for one week he had vomited after every feed, that he "pumped it up," and that much clot was present in the vomit. As previously, examination was entirely negative. He looked a healthy, well-developed infant, and weighed 12 lb. 14 oz. He fed eagerly, even greedily, and was given boiled water and chloral before feeds. Vomiting was completely controlled after five days, while weight was gained after a small initial drop. During this time the stools remained normal in frequency and colour. Nov. 6: Weight 13 lb. 1 oz., feeding well but greedily. Nov. 11: Pyrexia (100° F.) noted; stools, though not frequent, were green, and vomiting occurred once; general condition good. From this time onward a remittent temperature to 102.4° was maintained; stools were frequent, green, and offensive (up to 7 in 24 hours), with occasional vomiting. The infant looked pale, but lay quietly and showed no signs of dehydration. Glucose-saline was given by mouth combined with a course of succinyl-sulphathiazole.

Meanwhile investigations were carried out. Radiographs of chest, examinations, and culture of the urine were negative. Blood examination showed Hb 84%, W.B.C. 13,600, with 59% polymorphs. Culture of stools revealed no pathogenic organisms, except on the second occasion, three days before death, when a few colonies of *B. proteus* were found.