atrophy of the anterior lobe of the pituitary as a result of necrosis due to thrombosis.

Apart from cystic degeneration in tumours and those arising in association with craniopharyngiomata, cysts of the pituitary large enough to cause pathological symptoms are rare, though small cysts arising in the tubules of the pars intermedia are not uncommon. An unusual feature of this case was the almost complete destruction of the anterior lobe by a haemorrhagic cyst. In view of the definite history of the onset of symptoms dating from the time of a difficult labour there can be little doubt that the pituitary destruction was related to the delivery.

It is difficult to see how haemorrhage alone could cause such a well-defined and circumscribed cyst, and a haemorrhage large enough to produce it would almost certainly have at the time of its occurrence caused severe optic chiasma pressure signs, of which there is no record. It is probable that the cyst was present before the pregnancy, causing some pressure atrophy of the anterior lobe, and that necrosis of this occurred at the time of delivery. There had undoubtedly been haemorrhage into the cyst on occasion; one of these was recent, but fibrosis and organization were too advanced to allow of any estimate of when others occurred. These changes also obliterated any evidence of the exact nature of the cyst.

Complete destruction of the anterior lobe seldom if ever occurs in Simmonds's disease (Sheehan, 1939) and the residual anterior-lobe tissue is usually composed mainly of chromophobe cells as in this case. The recent haemorrhage into this area may well have played a part in the patient's death. This haemorrhage, as also the other recent haemorrhages from gums and into the vertebral and femoral marrow, may have been secondary to the aplastic anaemia.

Cause of the Aplastic Anaemia

The association of the marrow aplasia with Simmonds's disease may be fortuitous, but in view of the absence of any other likely cause, and knowing that Simmonds's disease gives rise to anaemia, it seems reasonable to assume that the long-standing pituitary damage in this case resulted in the aplastic anaemia. It is known that pituitary hypofunction can give rise to a gastrogenic pernicious anaemia, and the aplastic anaemia may be the end-result of such a condition: unfortunately we have no knowledge of the previous state of the blood or whether achlorhydria existed. In this case, apart from the reduced size of the thyroid gland, there was little microscopical evidence of severe atrophy and the acini showed normal colloid formation. It seems unlikely, therefore, that thyroid hypofunction could have been responsible for the marrow hypoplasia. There was atrophy of the adrenals and the ovaries, but, so long as such uncertainty exists regarding the action of the endocrines on haemopoiesis, cases such as this can only give rise to speculation.

Our thanks are due to Dr. C. D. Coyle, medical superintendent of the Archway group of hospitals, for permission to use the clinical notes; and to Dr. Mohun, senior assistant pathologist, Archway group laboratory, for his co-operation and for providing the reports on the haematology and biochemical findings. We also wish to thank Dr. A. B. Bratton and Prof. H. L. Sheehan for their helpful criticism.

REFERENCES

- Bomford, R. R. (1938). *Quart. J. Med.*, n.s., 7, 495.
Escamilla, R. F., and Lissner, H. (1942). *J. clin. Endocrinol.*, 2, 65.
Jones, R. M. (1940). *Amer. J. med. Sci.*, 200, 211.
Sheehan, H. L. (1939). *Quart. J. Med.*, n.s., 8, 277.
Snapper, I., Groen, J., Hunter, D., and Witts, L. J. (1937). *Ibid.*, n.s., 6, 195.
Watkinson, G., McMenemey, W. H., and Evans, G. (1947). *Lancet*, 1, 631.
Witts, L. J. (1942). *Ibid.*, 2, 307.

Medical Memoranda

Typhoid Meningitis

A male Chinese aged 25 years was brought into Klang Hospital, Malaya, at 10.30 a.m. on Aug. 9, 1946, complaining of severe headache and pains over his whole body for ten days before admission. Before July 31, 1946, he felt perfectly well.

CASE HISTORY

On July 31 he felt unwell and developed a fever, severe frontal headache, vomiting, insomnia, and a continual aching sensation throughout his whole body, particularly in his legs. The headache was continuous until admission, and he vomited two or three times a day. His appetite was poor and he lost weight. There was neither diarrhoea nor constipation, and micturition was normal.

On Aug. 8 he found that his eyes were very sore in the light. There were no other symptoms.

Examination revealed a well-built Chinese; he was conscious, and showed no skin rash, jaundice, or other abnormalities. He liked to lie curled up on his left side and disliked light on his eyes. The temperature was 103° F. (39.4° C.), pulse 96, respirations 24. There was no neck stiffness, and Kernig's sign was negative. Clinical examination of the central nervous system, chest, and heart revealed nothing abnormal except the photophobia. Loss of weight was evident in both thighs. There was no muscle wasting. Abdomen: no masses or engorged veins were present; no tenderness or rigidity; liver and spleen not palpable; nothing abnormal per rectum.

Investigations on Aug. 9.—Blood film: negative for malaria. Blood count: haemoglobin, 70%; W.B.C., 10,000 (polymorphs, 69%; lymphocytes, 30%; monocytes, 1%). Urine: no albumin; no sugar; film negative. It was decided to observe him and repeat blood-film examinations for malaria.

On Aug. 10 his temperature was 103.4° F. (39.7° C.), pulse 92, and respirations 22. Condition unchanged. Blood films negative. On Aug. 11 temperature was 103.6° F. (39.8° C.), pulse 86, and respirations 22. He suddenly became worse about midday, complaining of a sudden and severe increase in the severity of the headache. His vomiting returned and became much worse. He now had neck stiffness, a positive Kernig's sign, and extensor plantar reflex. On lumbar puncture the cerebrospinal fluid was slightly turbid and under pressure. It was sent to the Institute of Medical Research, Kuala Lumpur, for full examination, and in the meantime the case was treated as a meningitis (our own laboratory film revealing pus cells but no organisms).

Penicillin, 100,000 units daily, was given, with sulphathiazole tablets (initial and second doses being 2 g., followed by 1 g. every four hours). As the patient showed no response, this treatment was discontinued on the morning of Aug. 14. In the afternoon of the same day the I.M.R. report was received.

Report from I.M.R.—Smear: moderate number of pus cells and Gram-negative bacilli. Culture: *B. typhosus* isolated in pure culture. Globulin: Nonne-Apel test positive. Protein: 200 mg. per 100 ml. Chlorides: 680 mg. per 100 ml. Cells: 550, of which 80% are polymorphs.

A sample of blood was taken for Widal and Weil-Felix tests. The result was T 1/480A-O. The Weil-Felix, as expected, was negative. This finding supported a diagnosis of typhoid meningitis.

On Aug. 18 the patient, being treated as a case of typhoid fever, began to improve. On Aug. 31 he developed cystitis and his urine was found to be full of pus cells. This condition disappeared four days later as rapidly as it came.

On Sept. 9 he was much better and complained only of a slight persistent frontal headache. He developed a right facial palsy. His white-cell count was now 11,000 (polymorphs, 73%; lymphocytes, 23%; monocytes, 4%). On Sept. 20 he felt fit except for the right facial palsy, which still persisted. His white-cell count was now 4,500 (polymorphs, 63%; lymphocytes, 32%; monocytes, 3%; eosinophils, 2%).

He was discharged on Sept. 30 fit except for slight facial palsy. Before discharge three consecutive faecal examinations were done, but all were negative.

COMMENT

I have reported this case because true suppurative meningitis due to *B. typhosus* is relatively rare. True, one out of ten patients get meningism in typhoid fever at the onset of the disease, but an actual meningitis is rare. When it does occur, recovery is rarer still, because early involvement of the nervous system, as in this case, is usually an indication that the prognosis is poor.

I should like to thank Dr. R. B. MacGregor, Director of the Medical Services, Malayan Union, for his kind permission to publish this case.

W. E. STUART,
Medical Officer,
Malayan Union.