flexures of colon, but apart from this no definite evidence of organic disease."

The blood count showed: red blood cells 4,500,000 per c.mm., white blood cells 8,000; polymorphonuclears 74 per cent., large mononuclears 4 per cent., lymphocytes 20 per cent., eosinophils 2 per cent.; haemoglobin 90 per cent.; colour index 1.1. No abnormal cells were seen.

COMMENTARY

The interesting point about this case is that the clinical, radiological, and microscopical examinations appear to exclude all the commoner conditions giving rise to a haematemesis of such severity. The patient stated that she had never suffered from any gastric symptoms in her life, nor did the radiological examination show any evidence of gastric or duodenal ulcer, nor had she been treated for either at any time. There is certainly a possibility that the deformity of the duodenal cap was due to an ulcer as well as to adhesions. The past clinical history undoubtedly points to the latter cause. At the same time it seems incredible, if an ulcer did exist, that it would give rise to no clinical symptoms save a sudden and severe haematemesis, which did not recur and for which no special treatment was given.

There had never been any evidence of cirrhotic changes in the liver. Thrombosis of the portal vein can be eliminated, as the other usual sequels—namely, enlargement of the spleen and ascites—were entirely absent, and the haemorrhage had not recurred. The normal blood count, together with a normal spleen, excluded early splenic anaemia and leukaemia. What, then, was the cause of the haematemesis? We appear to be left with a gastrostaxis occurring by diapedesis. Might this not be the result of a very sluggish liver of long duration giving rise to a more or less constant hyperaemia of the gastric mucosa? However sceptical one may be in regarding the haematemesis as a sequel of the amoebic hepatitis, it is difficult to exclude the contingency occurring in such an obstinate and long-standing case.

It must be borne in mind that the patient is a nurse on duty in a fair-sized hospital, where she has always had constant and immediate medical attention from a highly competent staff of medical practitioners, so that any gastric disturbances would have been promptly treated.

I have to thank the Director-General of Medical Services, Ministry of Pensions, for permission to record this case.

CONGENITAL ANEURYSM OF THE BRAIN

BY

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The problems of differential diagnosis are nowhere more strikingly illustrated than in aneurysms of the base of the brain. It is now well recognized that besides those instances in which the condition is associated with infective endocarditis or cerebral arterio-sclerosis, there is a group of cases in which the aneurysm is congenital, or depends on a congenital weakness in the arterial wall at certain points. To this congenital weakness of the arterial wall the case reported owes its interest.

CASE NOTES

History.—The patient, a married woman aged 33, was quite well until a month ago, when she had a slight attack of vomiting and complained of a vague headache. Since

then she had not felt really well. On September 8th she fainted twice and vomited without warning. During the following week she complained of low occipital headache and pain in the back, and felt "feverish." On September 16th she walked to her doctor's house at 8 p.m. While in the surgery she suddenly collapsed and became comatose. The doctor applied artificial respiration and made arrangements for her admission to hospital.

Condition on Admission.—The patient was deeply comatose. The face and body were pale and cold. The muscles were flaccid and all reflexes absent. The pupils were widely dilated. Examination of the discs showed nothing abnormal. The systolic blood pressure was 110, and the heart rate 70, irregular, but not fibrillating. The urine contained no sugar, but a trace of albumin. No incontinence. Lumbar puncture obtained blood.

Progress.—Artificial respiration was maintained until 2.30 a.m., when intratracheal air was given. Fibrillation was noted at 3 a.m. Death occurred at 3.30 a.m.

Necropsy.—A complete post-mortem examination was made with the following findings. The convolutions of the cerebrum were flattened, and there was a large amount of blood clot in the pia-arachnoid at the base of the brain. The haemorrhage extended along the sulci over the right side of the cerebrum. The fourth ventricle contained blood clot, the lateral ventricles blood-stained cerebro-spinal fluid. Over the junction of the right anterior cerebral and anterior communicating artery was a pale fibrinous clot, about the size of a pea, evidently the remains of an older haemorrhage. The two frontal lobes were adherent to each other in this area. On lifting off this clot there was found a saccular aneurysm measuring 1 by 0.5 by 0.5 cm. occupying the space between the two anterior cerebrals and springing from the junction of the above arteries. The aneurysm showed a white fibrous-looking wall, thinned on the lower side, where it was ruptured. The other blood vessels of the brain showed no abnormality. The pericardium was adherent to the posterior aspect of the left ventricle by some old cord-like adhesions over an area of 2 cm. square. No other abnormality was found in the body. Microscopical examination of the liver and kidneys revealed no sign of disease. The Wassermann reaction of the blood obtained at the post-mortem examination was negative.

COMMENTARY

On reviewing the case in the light of the necropsy, it is evident that the attack on September 8th was due to a leakage from the aneurysm causing a small subarachnoid haemorrhage, which became arrested before causing material damage to the brain or cranial nerves. The subsequent attack was due to a sudden large haemorrhage causing a rapid and fatal compression of the medulla. No evidence of the presence of an aneurysm was detectable before rupture.

The clinical symptoms of a congenital aneurysm may be conveniently divided into symptoms prior to rupture and those following rupture.

Before rupture, localizing symptoms causing pressure on neighbouring structures may be recognized. In the region of the anterior half of the circle of Willis the second, third, fourth, fifth, or sixth nerve may be irritated. Thus a case has been reported where an intractable and apparently idiopathic trigeminal neuralgia was the only symptom. Again, an irregularity of the pupil on one side might be the only tell-tale sign. In the posterior half of the circle of Willis, pressure on the brain stem, in addition to paralyses of the last four cranial nerves, might give rise to paraplegia, cerebellar symptoms, dysarthria, dysphagia, or contralateral hemianaesthesia.

After rupture, leakage of blood into the subarachnoid space at the base of the brain gives rise to clinical signs upon which a diagnosis may be made with reasonable certainty, although meningitis or influenza may be closely simulated. Depending upon the rate of the haemorrhage, the intensity of the symptoms varies. Loss of consciousness passes on to deep coma with a severe haemorrhage, while with a small one, giddiness, headache, and vomiting may be the only

effect. Moderate pyrexia, stiffness of the neck, and positive Kernig's sign may be present, so that except for the difference in the cerebro-spinal fluid or the suddenness of onset or a possible previous attack, a diagnosis of meningitis might well be made.

C. P. Symonds gives a full report on this type of haemorrhage, and points out that Sir William Gull in 1859 was the first to recognize its comparative frequency.

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Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

LACTATION IN ACCESSORY BREASTS

While the occurrence of accessory breasts is not uncommon, a case in which they actively functioned may deserve recording.

I attended, in September last, the confinement of a primipara, aged 23. She had a normal pregnancy up to the third month, when she noticed for the first time a lump in the right axilla immediately behind the anterior fold. At the eighth month a similar lump appeared in the left axilla. These lumps presented no true nipples, but in each there was a small dilated pore in the middle of the overlying skin. On the right side, midway between the true nipple and the umbilicus, was a small elevated nipple without any appreciable breast tissue beneath it.

On the second day after delivery the axillary lumps swelled concurrently with the true breasts, and reached the size of small oranges. A considerable quantity of milk exuded from the central pore of each. There was no change in the right accessory nipple. The accessory breasts gave an insufficient supply of milk to be of use to the child, and caused inconvenience to the mother by reason of pain and wetting of the clothes. They decreased in size in the absence of the sucking stimulus, and there was an uneventful puerperium.

I have seen several other cases of accessory breasts, but it is so rare for lactation to occur in axillary breasts that I think this case is of much interest.

I am indebted to Dr. E. Robertson for permission to publish this short account of the case.

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A CASE OF GLANDULAR FEVER

In view of the recent correspondence in the *Journal* on this subject, the following case may be of interest, especially in view of the fact that at no time during the illness was there any rash, a symptom which appears to have been a feature in some of the cases described by your correspondents.

J. M., aged 13, was taken ill on June 14th, 1930. When I first saw him he was listless and suffering from general malaise and a severe headache. He had a temperature of 103° F.; pulse 80. He gave a history of being on the river in a boat in strong sunshine, wearing only his school cap. After a short interval of three or four days, during which his temperature became normal, he again suffered from a rise of temperature, and complained of sore throat, pain in the neck, and general malaise.

On examination the throat was slightly red, tongue furred and dry, chest organs normal, but the spleen was distinctly palpable. The cervical lymph glands were enlarged on both sides, and the glands in both the carotid and posterior triangles were involved. Further search revealed slight enlargement of the lymph glands in the axillae. As the illness progressed the cervical glands became larger, very

swollen and tender, and extremely painful. This persisted throughout the disease, during which there was considerable pyrexia. I enclose the temperature chart, from which it will be seen that the range was from 97° to over 104°. After a preliminary diagnosis of heat stroke, which had hurriedly to be revised, I came to the conclusion that I was dealing with a case of glandular fever, and this diagnosis was borne

out by the blood count, the details of which are as follows: red blood cells, 6,000,000 per c.mm.; haemoglobin, 80 per cent.; colour index, 0.7. White blood cells, 18,000 per c.mm. Differential white cell count: polymorphs, 9,180 (51 per cent.); lymphocytes, 8,640 (48 per cent.); large mononuclears, 180 (1 per cent.); eosinophils, nil.

After lasting for twenty-two days, the temperature fell by crisis, and the glands gradually began to recede in size. Recovery has been uneventful, except that the convalescence has been slow, and the boy has even now not yet regained his usual spirits. There was never at any time any suspicion of a rash.

In my view the chief points of interest in this case are: (1) the blood count, which revealed a moderate degree of leucocytosis, but mainly of the small lymphocytes, whereas some of your correspondents found that the increase was in the large lymphocytes; (2) the absence of rash.

I am indebted to Dr. W. H. McKinstry, pathologist to the Willesden General Hospital, who kindly did the blood count, and to Dr. C. R. Box, F.R.C.P., physician to the London Fever Hospital, who came at my request and confirmed the diagnosis.

London, N.W.2.

E. C. WHITEHALL COOKE.

Reports of Societies

THE TREATMENT OF PNEUMONIA

At the meeting of the Medical Society of London on March 10th, with Dr. R. A. YOUNG in the chair, a discussion took place on the treatment of pneumonia.

LORD DAWSON OF PENN said that the treatment of lobar pneumonia had for centuries caused concern and controversy, justified by the prevalence and high mortality of the disease, and by the way in which it attacked both the robust and the unhealthy. One out of every 1,300 of the population succumbed to it every year. The attitude of mind towards the treatment of pneumonia changed from the time when it was established as an infection by a pneumococcus usually localized in the lung and pleura. He believed that specific therapy gave the greatest hope. The development of the serum treatment of pneumonia had followed the classification of the pneumococcus by Cole and other workers into four etiological groups. Of these the first, second, and third were specific types, while the fourth was a motley collection of serological strains. The serum contained protective substances similar to those which the patient's body was trying to produce, and did in fact produce, at the crisis in large amount if prognosis was favourable. These substances were antibacterial, not antitoxic. Their efficacy was commonly tested by their ability to protect mice from death when injected simultaneously with a lethal dose of organisms. Groups 1 and 2 accounted for 50 to 75 per cent. of the total cases. If, therefore, something could be done for these two groups, material progress would be made. Yet, in practice, serum treatment had not made much headway; probably