Medical Memoranda

Cysticercosis Epilepsy treated with Sulphathiazole

The recent articles by Blythe (*British Medical Journal*, 1941, 1, 401) and Ewing (*ibid.*, 1941, 2, 263) prompt me to record the following case under my care.

PREVIOUS HISTORY OF THE CASE

A man aged 29 was found unconscious in Sutton Park at 11.50 p.m. on June 29, 1941. He was taken to Sutton Coldfield Cottage Hospital, and transferred to Great Barr Park Colony the following day. He remembered leaving camp on June 29 at 4.30 p.m. and going to Birmingham, and nothing more. There was a slight smell of alcohol in his breath.

The personal history was as follows. No illness as a child. In 1930 he joined the Royal Navy and saw service in the Mediterranean, in 1932 in the Baltic, and in 1935 in China. In 1937 he returned to civilian life, where he worked in Bedfordshire, labouring until 1939. In 1939 he joined the Army and has served at home since.

In 1935, while serving in China, he suffered from "a tropical disease"; he did not know what it was and he is unable to remember the symptoms. Subsequent to this on seven occasions he has had an attack of loss of consciousness with complete amnesia of the incident. Since 1935 he has complained of headaches and mental depression. He has also noticed that on each occasion preceding his seizure the headaches have increased in severity and culminated in the fit. There was no history of vomiting or otorrhoea; he has never suffered from meningitis.

CONDITION ON ADMISSION

His I.Q. falls within normal limits; he is dull and somewhat apathetic. He sleeps quite a lot; often asks for some tablets to make him sleep, but he is asleep before the tablets can be procured. His memory on the whole is fairly good, but he is unable to give many details about himself. No family history of mental disease or of convulsions could be obtained.

Examination of Nervous System.—Pupils equal and responded to light and to accommodation, and ophthalmoscopic examination showed blurring of both disks on the nasal side; also venous pulsation on the right side. Nothing else abnormal found on examination of the cranial nerves. Upper extremity: Power and muscle tone normal and equal on both sides; reflexes average and equal on both sides; no sensory loss; joint sense and muscle sense normal; co-ordinated movements normal; no astereognosis. Abdominal reflexes were normal on the right side but diminished on the left. Lower extremity: Power and muscle tone appeared normal, with no perceptible difference on the two sides; no sensory loss; co-ordinated movements normal; joint sense and muscle sense normal; knee-jerks somewhat increased on both sides, especially the left; ankle-jerks increased, especially on the left side; plantar reflexes, right flexor, left extensor; ankle clonus not obtained; no Rombergism; sphincters normal.

General Examination.—Nothing abnormal found on clinical examination of the heart, lungs, and abdomen; no albuminuria; no glycosuria. Blood pressure: systolic 130, diastolic 88. Blood: Wassermann, negative; Kahn, negative; gonococcal fixation test, negative. Blood film, differential count of leucocytes: polymorphs—neutrophil 58%, eosinophils 6%; monocytes 19%, lymphocytes 17%. A slight increase of eosinophils and a marked increase in the monocytes.

Cerebrospinal Fluid.—Colourless and clear, definitely under increased pressure, degree not measured; cells, 2 per c.cm.; protein, 35 mg. per 100 c.cm.; chlorides, 720 mg. per 100 c.cm.; reducing substance present; Lange gold curve, no change; Wassermann negative in all directions; direct film, no acid-fast bacilli or other organisms seen; culture, no growth.

X-ray Examination.—Head, thorax, and extremities were all radiographed for the presence of cysts. In the left lung there was a cyst about the size of a five-shilling piece, which showed

the features of a hydatid cyst. There was also a suggestion of an infected left maxillary antrum. Nothing else abnormal was found.

In view of these findings it was considered that the symptoms were due to the presence of a hydatid cyst of the brain.

TREATMENT ADOPTED

The report of a case, reached me (verbally) of a hydatid cyst of the liver which, at operation, had secondarily infected the peritoneum and had been exposed to sulphonamide treatment (given primarily for a superimposed secondary infection). In this case it was found that the daughter cysts in the peritoneum were being discharged through the wound in a shrivelled condition, and, further, the percentage of sulphonamide in these daughter cysts was greater than that in the blood stream. The effect of chemotherapy was therefore tried in this case, the preparation used being sulphathiazole (M & B 760).

On July 9 the patient was given four tablets (2 grammes), and this was followed after six hours by two tablets (1 gramme) four-hourly for forty-eight hours. This in turn was followed by two tablets six-hourly for forty-eight hours, and the course was completed by giving two tablets three times a day for a further three days. At the end of the week (July 16) the man was re-examined. He was no longer complaining of headaches. There was no mental depression and he was no longer apathetic, nor did he sleep to an abnormal degree. There was a great change in his personality. He became bright and cheerful, and he took a natural interest in the comings and goings in the wards. His abdominal reflexes on the left side, which had been sluggish, reacted normally and equally with those on the right. His left plantar reflex, which had been extensor, now became flexor. His ankle-jerks and knee-jerks on the left side became equal with those on the right, but both remained rather more active than normal. Both disks showed blurring on the nasal side.

The findings were thought to be so promising that it was decided to repeat the course of sulphathiazole. On July 27 this was given as before, except that instead of terminating on the seventh day the course was continued until the twelfth day. On August 8 the patient was re-examined. The improvement had persisted, and the only abnormal findings were that the disks showed some blurring on the nasal side and the knee-jerks and ankle-jerks on both sides were still somewhat overactive. A differential leucocyte count was made: polymorphs—neutrophil 61%, eosinophils 0%; lymphocytes 34%, monocytes 5%. The monocytes had returned to a normal percentage. No eosinophils were formed. A further radiograph of the lungs was taken under conditions (kV, mA, time exposure, and distance) similar to the first picture. It was found that the cyst was less obvious than in the first picture; it was, however, still present. The size as depicted on the radiograph did not appear to be materially altered.

I fully realize that enough time has not elapsed since the treatment was started to allow any conclusions to be drawn. However, in view of the recent articles on cysticercosis epilepsy and the possibility of an approach to it by chemotherapy, I thought this case interesting enough to quote. I would be grateful for any comments.

I must thank Dr. D. M. Macmillan, medical superintendent of this Colony, for permission to publish this case, and also my colleagues for their advice and helpful criticism.

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Giant Hydrocele successfully treated by the Sclerosing Method

I have now treated seventy-four cases of hydrocele of the tunica vaginalis, some at the Injection Clinic at the Southend General Hospital and some in private practice, with complete success in every instance, although in about one-third of the cases more than one injection proved necessary in order to effect a complete cure. It takes on an average about three months for a hydrocele to be cured by this method.

The technique is simplicity itself; it consists in tapping the hydrocele and withdrawing all the contained fluid, and then

injecting into the sac 4 c.cm. of lithocaine (lithium salicylate 30% and tutocain 1%), followed by 2 c.cm. of quinine-urethane. The parts are thoroughly massaged after the injection, and the patient is ordered to wear a testicular support. A week later the fluid which accumulates in the tunica vaginalis as the result of chemical irritation of the delicate endothelial lining is cautiously aspirated.

The following case is of great interest, as it proves conclusively that a hydrocele of enormous proportions and of many years' standing can be treated successfully by the sclerosing method.

CASE REPORT

The patient, aged 42, consulted me on August 2, 1939, complaining of an "enlarged testicle." He had first noticed the swelling in 1928, but it had not increased much in size until the last three years. It then grew rapidly and caused a great deal of embarrassment on account of its weight and gigantic size. In spite of the fact that he was short, stout, and very stockily built, with consequently loose-fitting clothing from the waist down, it was apparent the moment he entered the room that there was something very abnormal amiss, and when he sat down it looked as if he was carrying a football in his left trouser pocket.

Examination revealed a giant hydrocele of the following dimensions: length 10½ inches, circumference 17½ inches. When tapped, 64 oz. of typical straw-coloured hydrocele fluid was evacuated. After the tapping was completed 5 c.cm. of quinine-urethane was injected, followed by 10 c.cm. of lithocaine. This mixture was thoroughly agitated inside the sac, and the patient was then sent home and told to retire to bed early in the evening.

On September 16 a second tapping was carried out, and 24 oz. of fluid was withdrawn. This time I injected 2 c.cm. of quinine-urethane and 8 c.cm. of lithocaine into the hydrocele sac. On October 23 a further 12 oz. of fluid was withdrawn, and another combined injection was given. The patient was told to return in six months' time, and when he did so, on April 24, 1940, I was disappointed to find that the hydrocele, although much smaller, was still present and appeared to have a very thick wall. He was tapped once again, 10 oz. of fluid being obtained, and a further injection of the two sclerosing media was undertaken. I did not see him again until May, 1941, when I was agreeably surprised and pleased to find that I had at last effected a complete cure by the injection method. The testicle on the left side was then of normal size, and there was no undue thickening or discomfort subsequent upon the extensive fibrosis which had occurred.

The patient was examined by Mr. Rodney Maingot at the Southend General Hospital in July, 1941, when it proved impossible on clinical examination to ascertain on which side the hydrocele had been, thus suggesting that a complete cure had been achieved.

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Transient Blindness following Blood Transfusion

The following is an interesting case of blindness after blood transfusion.

CASE REPORT

A man aged 42 was admitted to hospital on May 29, 1941, complaining of flatulent dyspepsia, loss of weight, and weakness. Clinical examination showed marked anaemia and obvious loss of weight. There were no abnormal physical signs in the respiratory, cardiovascular, or central nervous system. There was, however, an indefinite mass palpable in the epigastrium with some tenderness and muscular guarding over this region. He was put on Meulengracht's diet with alkalis.

The reports of the radiological and laboratory investigations were as follows. Barium meal: marked delay in emptying of stomach; pathological stenosis of the pyloro-duodenal region, nature not manifest, but could be the result of ulceration or of neoplasm, the former being the more likely. Blood picture (May 30): Red cells 2,700,000; white cells 8,000; haemoglobin 50%; film showed anisocytosis and central pallor of the red cells. Fractional test meal showed low total acidity, rising to a

maximum in two and a half hours: free HCl 0.2%; total acidity 0.45%. Wassermann reaction negative. Blood pressure 108/62. Urine: deposit consisted of urates only; no albumin or sugar present.

On June 9 the patient was given a blood transfusion of Group O (IV) blood with a view to improving his condition preparatory to laparotomy. The blood was given at the rate of 2 c.cm. per minute approximately, without incident. Fifteen minutes after the completion of the transfusion he had a very slight rigor, but no rise in temperature; this lasted about ten minutes, after which the patient slept. An hour later he awoke and complained that he was blind. On examination there was no perception of light, his pupils were dilated but reacted to light; there was no evidence of arteriospasm or of retinal haemorrhages on examination of the ocular fundi, and the disks appeared normal. Fifteen minutes later perception of light returned, and after a further thirty minutes he could perceive objects. His vision then improved steadily until one and a half hours after the onset of the condition it was normal. The patient's general state remained very good throughout, and no other physical signs were discovered in his central nervous system. On the following day the urine contained a considerable quantity of blood and albumin, but this cleared up in twenty-four hours.

Clinically, the patient appeared to be very much improved after the transfusion, but the blood count showed: red cells 2,000,000, haemoglobin 50%, blood urea 64 mg. per 100 c.cm., blood non-protein nitrogen 55 mg. per 100 c.cm.

Unfortunately it was not possible to test the donor blood against that of the recipient. The recipient's group, however, was O (IV), as was the donor's blood.

DISCUSSION

The possible causes of this reaction, in view of the transient nature of the blindness and the absence of positive findings by ophthalmoscopic examination, were considered to be: (1) oedema of the optic tracts; (2) arteriospasm associated with hypertension; (3) arteriospasm following severe blood loss; (4) air embolus; (5) uraemic blindness.

1. It is difficult to see how oedema of the optic tracts would have affected the optic nerves without producing other evidences of oedema in the central nervous system and elsewhere, and one would expect to find some evidence of such a condition on ophthalmoscopic examination.

2. Arteriospasm associated with hypertension can be excluded, since the patient's blood pressure was 108/62 before the transfusion and not materially different twenty-four hours later.

3. If arteriospasm following severe blood loss was the cause one would expect to find evidence of arteriospasm in the retinal vessels, but this was not present; also there was no sign of recent severe haemorrhage.

4. While air embolus is a possibility, air which enters the circulation during transfusion is almost certainly absorbed before reaching the arterial circulation and usually produces symptoms in the right ventricle or pulmonary circulation. Further, the bilateral blindness in the absence of other symptoms is difficult to explain on these grounds.

5. Uraemic blindness is also a possibility in view of the somewhat raised blood urea and N.P.N., but no other signs or symptoms of uraemia developed subsequently.

From these observations it will be seen that no definite aetiological factor has been decided upon. It would be interesting to hear if any readers have had similar cases, and whether they have been able to explain them. One fact should, however, be noted—that is, in this case vision was normal in about three hours, whereas in cases of transient blindness previously reported it has persisted for longer periods, even up to twenty-four hours.

I am indebted to Prof. T. B. Davie for his assistance and advice, and to Dr. R. A. Grant, medical superintendent of the Birkenhead Municipal Hospital, for his permission to publish this case.

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