serum, 25 c.cm., was given hypodermically. In five days the temperature had fallen to normal and the evidences of colitis had disappeared.

Case 4.—Female, aged 46. This patient developed a severe dysenteric attack, with blood and mucus, much abdominal pain, and pyrexia up to 103°. She was given 25 c.cm. of the serum, and after five days no further looseness of the bowels was recorded.

Case 5.—Male, aged 74. This man suffered from a severe attack of dysentery with slight pyrexia. Small doses of salines were given at frequent intervals. After two days of the saline treatment no blood or mucus was observed in the dejecta, and the temperature was found to be subnormal. The diarrhoea, however, continued, and two days afterwards there was a recurrence of the pyrexia and traces of blood and mucus were again detected in the motions. The patient was then given an injection of 25 c.cm. of serum, and after an interval of two days only two evacuations were recorded in the twenty-four hours, one of which was formed. There was a marked physical improvement twenty-four hours after the administration of the serum.

Case 6.—Male, aged 72. This patient, who had two years previously suffered from dysentery, again suffered from an attack of the disease. He was at the same time suffering from pernicious anaemia in a state of remission. For the first two days there were frequent evacuations containing blood and mucus, and associated with hyperpyrexia ranging up to 105.4°. On the third day the temperature was found to be subnormal, but the colitis continued unabated. On the fourth day the temperature rose to 100° and 40 c.cm. of antidyenteric serum were injected. On the following day the temperature was normal and the evacuations had decreased in number, but the blood and mucus were still present. Next day the number of motions was not diminished but blood and mucus had disappeared. In a further three days' time he had only one formed action, and shortly afterwards he was complaining bitterly of constipation.

Case 7.—Male, aged 53. This patient developed an acute dysenteric attack with a temperature ranging up to 104°. Twelve hours after the commencement of the attack 40 c.cm. of the serum were given. The temperature fell to normal within twelve hours after the injection, and during the day only three dysenteric motions were passed, although eight had been passed on the previous day. Two days afterwards he had only one formed motion.

Case 8.—Male, aged 67. This inmate suffered from a moderately severe dysentery with an even temperature of 101°. Only 25 c.cm. were used. The condition at once cleared up and at the end of a week he was again in his usual state of health.

Case 9.—Male, aged 82. This man developed dysentery at the same time as the previous case. The same treatment was used, and the course of the disease was exactly similar to that recorded under Case 8.

Case 10.—Female, aged 44. This patient was found to be suffering from a severe attack of dysentery, associated with blood and mucus, tenesmus, much abdominal pain, very frequent motions, and high pyrexia. She was given 20 c.cm. of serum daily for four successive days. The temperature had fallen to normal by the fourth day, and the tenesmus and pain had disappeared, while the number of motions had greatly decreased. By the fifth day mucus and blood had disappeared. The patient rapidly recovered, although a slight diarrhoea lasted for a week afterwards. This patient two years previously had had an attack of equal severity, which took a month to clear up under salines.

It is somewhat difficult to dogmatize on such a small number of cases, but owing to the comparative freedom of this institution from dysentery it has taken over two years to collect these records. The results are certainly of a more satisfactory nature than any obtained in this institution before the introduction of serum treatment. Apart from the rapid clearing up of the blood and mucus and the cessation of the diarrhoea, the rapid fall in the temperature and the immediate and marked physical improvement have been outstanding features.

FURTHER OBSERVATIONS UPON EXTERNAL HYDROCEPHALUS.

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IN THE BRITISH MEDICAL JOURNAL of July 16th, 1921 (p. 66), and elsewhere, I have described typical cases of acquired external (occult) hydrocephalus and the treatment adopted. These clinical pictures have been found with great constancy in numerous subsequent cases. The treatment, however, has been simplified in many cases. A striking fact has presented itself, in that congenital external hydrocephalus exhibits certain differences of character from the acquired form, and, since the operative outlook is much worse in the congenital cases, it is important to be acquainted with diagnostic methods other than the history as given by the parents.

A congenital external hydrocephalus is frequently associated with microcephaly. The reason for this is that the

brain pulsation is a great factor in the normal development of cranial capacity. When there is a layer of fluid external to the brain this expansile pulsation is diminished. Curiously, the ventricles do not enlarge as a rule in external hydrocephalus, at least not to any extent. The brain instead shrinks, the skull is often arrested in development, and the subarachnoid and subdural fluid stays abundant because the foramina of Magendie, Key, and Retzius are patent, and often there is no tela choroidea or arachnoid covering the fourth ventricle at all.

Many patients have normal-sized heads, but appear mentally defective with some bulbar paresis, and varying degrees of spastic or flaccid diplegia or paraplegia. Invariably the legs are more affected than the arms, since, next to the cerebellum, the region of greatest pressure seems to be the leg centres at the vertex where a puddle collects around the arachnoid villi, in the same way as rain collects and eddies at the mouth of a partially blocked drain. An opening in the dura in this region unless promptly closed is almost inevitably fatal in these cases, for cerebro-spinal fluid escapes in a jet and continues in spite of a vertical posture of the head and with ever-increasing frequency of respiration until the respiratory centre fails or multiple capillary cortical haemorrhages occur. The safest approach is a graduated suboccipital deduralization in chronic cases, which is also the more logical. Supratentorial measures are subsequently required in a certain proportion of cases. Many cases of so-called Little's disease are therefore not due to haemorrhage nor to trauma at the vertex, the vertical symptoms being secondary to defect in function of the basal membranes.

I operated upon a four-days-old infant suffering from continuous right-sided Jacksonian fits following a prolonged forceps labour, and found excessive clear fluid at the vertex, but no haemorrhage, as was expected. The dura was left open over the Rolandic area in this case, and the child made a perfect recovery and is now normal. Such a procedure would have been followed by a very severe reaction in a chronic case.

It is not easy to say why a uniform increase of external fluid produces a flaccid and sometimes a spastic diplegia, but, contrary to expectation, the flaccid type of this disease has often a better prognosis than the spastic. This latter fact possibly indicates that the cerebellum has been more compressed than the cerebrum in the well-nourished flaccid cases. Another point is that the congenital cases do not have fits, as far as my observation goes, whereas the acquired cases of external occult hydrocephalus always do, even when the disease has been acquired within the first few days of extra-uterine life. I believe that the congenital cases have had their fits *in utero*, or the cerebral devitalization has progressed beyond the convulsive stage before birth.

In a series of 29 cases of external hydrocephalus operated upon there has not been one with either clinical or serological signs of syphilis, nor with appreciable enlargement of the head. On the other hand, cases of internal hydrocephalus due to cerebello-medullary adhesions show a high percentage of positive Wassermann reactions, and, of course, great cranial enlargement. Such cases are scarcely worth surgical consideration.

The acute specific diseases, especially tonsillar infections, as well as birth trauma, appear to be the usual causes of primary external hydrocephalus. Without doubt, the frequent textbook statements regarding "ex vacuo" accumulations of fluid are true of local collections in certain instances, but I believe that when extracerebral fluid is general, the brain has shrunk because the fluid accumulated owing to defective powers of absorption of the basal arachnoid, and that the fluid did not accumulate to fill the vacuum left by an otherwise unaccountable atrophy of the cortex. In other words, there seems to have been a tendency in the literature to put the cart before the horse in this matter. Why the brain shrinks and the ventricles do not dilate in external hydrocephalus is purely a question of physics.

THE third conference of the International Union against Tuberculosis will be held in Brussels from July 11th to 13th. The subjects for discussion include: (1) Tuberculosis in the child, (a) before school age, and (b) during school life; (2) antituberculosis prophylaxis in the home by the visiting nurse; and (3) the work of the tuberculous during and after cure. Visits will be organized to the different antituberculosis establishments in Belgium. Those who wish to become members are requested to send their names and addresses with subscription (£1) before June 26th to the secretary, International Association for the Prevention of Tuberculosis, 20, Hanover Square, W.1.