NEURO-SYPHILIS WITH SEVERE LARYNGEAL CRISIS: TRACHEOTOMY.

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CONSIDERATION of the symptoms presented in the case here reported points to an irritative cortical lesion as the most likely cause of the severe laryngeal crisis. The experiments of Krause, Semon, and Horsley have shown that there is a centre for adduction of the vocal cords in each cerebral hemisphere at the anterior part of the lower end of the ascending frontal convolution. Irritation of either centre results in adduction of both cords, while abduction though represented is more difficult to produce, and is rather governed from the lower centres in the floor of the fourth ventricle.

A pensisner, aged 35, was admitted about 3 a.m. on January 15th, 1922, as an urgent case suffering from respiratory embarrassment and cyanosis, having been found in this condition by the police. I saw him first about 8.15 a.m., in response to an urgent call, as his symptoms were becoming worse. He was then propped with pillows in bed in a semi-recumbent position, had a haggard and anxious expression, and looked seriously ill. The pulse was regular, 72 a minute, and of good quality and tension. Respiration seemed shallow and difficult, but there was no stridor. He was apathetic and apparently unable to raise his voice above a whisper. In a few minutes respiration ceased altogether, and his pulse then became weaker and ceased to be palpable. Tracheotomy instruments had meantime been got ready, and a low tracheotomy was then performed by a single incision of the scalpel. The tracheotomy tube was inserted without delay, but respiration did not begin, and it was noticed that there was no bleeding from the tracheotomy wound. Artificial respiration by compression of the lower ribs (Howard's method) was resorted to, and in a short time spontaneous breathing returned. A few seconds later, and before consciousness returned, there were convulsive movements of the arms and legs sufficiently violent to throw the tray with the instruments off the bed and to give some trouble to keep the patient from being jerked out of bed. He then appeared to waken up, lay down quietly, and was able to phonate quite well when the finger was placed over the tracheotomy tube. Three hours later, when I saw him again, he had lost his anxious look and expressed himself as feeling well. The tube, which had slipped out of the trachea, was replaced in position.

Examination of the larynx showed that the epiglottis was infantile in type and hid the greater part of the vocal cords. The arytenoids moved normally, and there was no oedema or other sign of abnormality. There was a superficial ulcer round the fraenum of the tongue. Knee-jerks were exaggerated, especially on the left side, and there was slight clonus of the left ankle. There was no noticeable "scanning" speech. The pupils were equal, moderately dilated, and reacted well to light. The urine contained some albumin and a trace of blood. On January 18th direct laryngoscopy with cocaine anaesthesia showed the movements of the anterior parts of the vocal cords as far as the vocal processes to be normal, but the posterior one-third moved sluggishly and did not approximate fully. There was some redness and oedema of the mucous membrane of the arytenoids and false cords, but this was considered to be due to passive congestion, and not inflammatory. There were no further laryngeal symptoms, and in ten days the tube was removed and the wound healed up. On January 30th examination revealed a painless swelling the size of a hazel nut in the left anterior pillar of the fauces. A week later this gumma had completely gone, and another had appeared at the anterior border of the right sterno-mastoid muscle, causing some pain and stiffness of movement of the neck. This also subsided within a week, leaving no trace. Dr. Douglas Adams, under whose care the patient was, has kindly supplied the following further details. He had frequent diplopia, lightning pains in legs, girdle sensation, frequency and precipitancy of micturition, difficulty in walking in the dark. The knee-jerks became slightly sluggish, but were never absent, and there was impairment of tactile sensation in the legs. The pupils continued to react actively to light. The Wassermann reaction for blood and cerebro-spinal fluid was positive. The cerebro-spinal fluid gave a normal cell count and protein content. The colloidal gold reaction showed a strong luetic curve—namely, 33221100000.

The patient was discharged from hospital on March 3rd, 1922, to continue antisyphilitic treatment, which had been confined to mercury and iodide on account of the albuminuria.

The occurrence of general convulsions during recovery in this case would be explained by extension of the irritation to neighbouring centres, as frequently occurs in laryngismus stridulus. The appearance and rapid subsidence of gummata elsewhere supports the view of a cortical lesion, while the paucity of localizing symptoms points to a gummatous formation in the membranes covering the cortical centre rather than in the brain substance. Had there been a lesion in the neighbourhood of the pons or fourth ventricle other cranial nerves could scarcely have escaped being involved.

At the same time the possibility of early tabes has strong claims. It is known that such crises are an early—and sometimes the earliest—symptom of this disease, and that the spasmodic attacks vary greatly in frequency, occurring sometimes only two or three times in the course of years, and sometimes daily or oftener.¹ Yet a diagnosis of early tabes does not negative the probability of the laryngeal crisis in the present instance being due to an irritative cortical lesion caused by a gumma.

Another point worthy of note is the quick recovery, even after the cessation of both circulation and respiration. The question at once arises as to how soon after apparent death measures for resuscitation must be regarded as of no avail. A recent paper in the BRITISH MEDICAL JOURNAL,² in which among other cases one of resuscitation two hours after apparent death is recorded, is of interest in this connexion. It seems reasonable to conclude that, at least in cases where there is not an obvious cause of death, measures for resuscitation should not be lightly abandoned. Particularly does this apply in cases of sudden death under chloroform or other general anaesthetic, in which the patient is usually comparatively vigorous, and in which perhaps a rapidly performed tracheotomy followed by Howard's method of artificial respiration might be tried more often with success. Free entrance of air into the lungs is assured, there is less disturbance of the patient than with Sylvester's method of artificial respiration, while external massage of the heart is at least as efficient; and finally, it is much less drastic than direct massage of the heart through an incision into the pericardium.

I am indebted to the Minister of Pensions for permission to publish the above.

REFERENCES.

- ¹ Allbutt and Rolleston: *System of Medicine*, 1908 edition, pp. 280, 281.
² Dr. D. E. Anderson: Resuscitation after Apparent Death, BRITISH MEDICAL JOURNAL, March 11th, 1922, p. 416.

THE TREATMENT OF ASYLUM DYSENTERY BY MEANS OF ANTIDYSENTERY SERUM.

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THE results in the following cases of asylum dysentery, treated at this institution during the past two years, indicate the value of antidyenteric serum. The matter is of importance, since during the second half of 1921 the number of persons in mental hospitals who were attacked by the disease was 728, and of these 126 died.

The serum mainly used was supplied by Messrs. Burroughs, Wellcome, and Co., and was prepared by injecting horses with cultures of Shiga's, Flexner's, and Kruse's bacilli. In Cases 5 and 6 a serum of a similar nature prepared by Messrs. Evans, Lescher, and Webb was administered.

Case 1.—Female, aged 48. The patient, who was in an extremely debilitated state, developed diarrhoea. On the fifth day of the attack the dejecta contained blood and mucus. No previous attack of dysentery had been recorded. The milk diet was stopped and a diet of albumen water, Benger's food, and Valentine's meat juice substituted. At the same time the patient was put on a bismuth mixture and iodoform enemata. This treatment was continued for six days without improvement; 40 c.cm. of the serum were then injected into the pectoral regions. In three days' time the blood and mucus had disappeared, and the patient showed a marked physical improvement. Convalescence was somewhat gradual, but no further traces of blood or mucus were detected, although diarrhoea continued in a mild form for two weeks after the injection. There was no pyrexia during the illness. Nine months afterwards the patient developed slight diarrhoea, cardiac weakness, and pulmonary congestion, and died. At the necropsy the mucous membrane of the large intestine was much injected, but there was no ulceration.

Case 2.—Female, aged 44. For three months this patient had suffered from intermittent diarrhoea, which had been treated with salines and bismuth. Blood and mucus then appeared in the dejecta. Antidyenteric serum, 45 c.cm., was administered. The dysenteric symptoms disappeared in three days' time. A week after the injection the bowels had not opened for twenty-four hours; twelve days after injection the stools were normal. No pyrexia was recorded during the course of the disease. The patient one year after treatment has had no recurrence.

Case 3.—Female, aged 21. This patient had suffered from diarrhoea for three days when blood and mucus were observed. There was slight pyrexia, ranging round 100°. Antidyenteric

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.

BY

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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.