Relation of hypothalamo-hypophyseal system to diabetes insipidus.—

With the aid of the Horsley-Clarke stereotaxic instrument, lesions were placed in various parts of the hypothalamus in 40 adult cats. In 10 instances prolonged and permanent polyuria developed, while in two cats transient polyuria was observed. In seven of the cats with permanent polyuria transient diuresis which preceded the onset of the permanent phase occurred. The transient polyuria was followed by an interval during which normal conditions of water exchange prevailed. In six instances the interval between the day of operation and the onset of the permanent polyuria varied from eight to 12 days. This interval has been called the latent period.

The transient polyuria has been found to differ from the permanent polyuria in the following respects. It develops much more rapidly and reaches a peak much sooner than does the permanent polyuria; the polyuria is primary to the polydipsia during the permanent phase, while the intake of fluid usually exceeds the output of urine on the first day of the transient phase; during the transient phase the output of urine and the intake of fluid may reach proportions never observed in the permanent phase.

The polyuria and polydipsia in the 10 diabetic cats lasted from two to nine months and appeared to be permanent. The output of urine and the intake of fluid for the animals with the most severe diabetes insipidus were five or six times greater than the values for the control animals. As the polyuria increased in intensity the specific gravity of the urine became correspondingly lower. Deprivation of water for several days brought about a reduction in the output of urine to a normal level and deprivation of food resulted in a reduction of the output of urine to about one-half the previous level. During the course of the experiments on the deprivation of water, the animals lost considerable fluid and a negative water balance developed, suggesting that the polyuria is primary. Repeated small doses of pitressin injected subcutaneously caused a reduction in the urine output and the fluid intake to normal levels.

Permanent polyuria was found to occur only in the cases in which there was bilateral injury to the supraoptico-hypophyseal system. Such
injury resulted in marked atrophy of the supraoptic nuclei and marked atrophy of the posterior lobe. Unilateral damage to this system did not cause polyuria, nor did lesions in any of the other parts of the hypothalamus. The possibility that transient polyuria may be related to irritative lesions of the anterior lobe was discussed.

The evidence from this investigation supports the theory that diabetes insipidus is a hormonal disturbance, caused by a deficiency in the secretion of the antidiuretic principle by the posterior or the intermediate lobe of the pituitary gland. The view is set forth that the supraoptico-hypophyseal system sends secretory impulses to these divisions of the hypophysis and that damage to this system at one of three points, the nucleus, the fibre tract and the pars intermedia and pars neuralis, results in diabetes insipidus.

R. M. S.


An analysis of 12 cases of cerebral varix, including the two reported in this paper, showed that they are equally divided between the sexes, six occurring in males and five in females. The sex was not reported in one case. The disease is more common in young persons, eight patients being 25 years or under: two new-born infants, two children and four adults between the ages of 22 and 25. The ages of the remaining four patients ranged from 38 to 50.

Clinically, the presence of cerebral varix was unsuspected in all the cases reported. In one instance the varix gave no clinical symptoms; it was accidentally discovered at autopsy. In the two cases of new-born infants, death was produced by increased intracranial pressure caused by a rapidly expanding varix. Rupture of the varix with fatal haemorrhage occurred eight times; without fatal haemorrhage once. Sudden increases in intracerebral pressure preceded fatal rupture four times; twice during eclamptic convulsions and twice during severe physical exertion. In one case in which the initial symptom followed trauma of the head sustained during the war, symptoms of pressure persisted for seven years before death from rupture occurred.

The sites of these vascular lesions were of particular interest. Three occurred in the anomalous ophthalmomeningeal vein of Hyrtl, while in a fourth case there was congenital malformation of the varicosed vein of Galen. In a total of seven cases there was involvement of the vein of Galen or of its tributaries or terminus. The pontine vein was varicosed in one case. There was only one instance of involvement of cortical veins.

Cellular infiltration of the wall of the vein was present in three cases. Amsler reported lymphocytic reactions in one case, while in both the present cases a similar invasion of the wall of the vein was observed. The authors do
not believe that the cellular reaction is evidence of a primary inflammatory process or that inflammation is productive of varices. In their studies the most striking picture was that of sclerosis of the vein. Connective-tissue replaced in varying degrees the muscle and elastic tissue elements of the venous wall; the internal elastic membrane, when it was not completely destroyed, was split into lamellae, and the intima in its thickening and hyalinization resembled closely that of an arteriosclerotic vessel. Probably the cellular reaction was a result rather than the cause of the degenerative process. In 11 of the cases in which analysis was made no evidences of inflammation were observed.

Congenital defects in the wall of the vein are probably the most important aetiological factor in the production of cerebral varix.

R. M. S.


The cerebrospinal fluid pressure is usually increased in cases of tumour of the brain. In this series of 182 cases normal pressures were common in cases of tumour of the brainstem or of the pituitary fossa and in those of glioma of a cerebral hemisphere with areas of cystic degeneration. Tumours located on the convexity of the hemisphere, especially meningiomas, may be accompanied by normal or only slightly elevated pressure, since such tumours do not interfere greatly with the circulation of the cerebrospinal fluid.

The cerebrospinal fluid was xanthochromic in 28 per cent. of the cases. Xanthochromia was usually associated with a high protein content. In a few cases the xanthochromia was due to destruction of red blood cells which had been extravasated by the tumour.

The cell count was usually normal in the lumbar cerebrospinal fluid. In 29 per cent. of the cases there were more than six cells, and in only 17 per cent. more than 10 cells, per cubic millimetre. Pleocytosis in the cerebrospinal fluid was due in most cases to an aseptic meningeal reaction to necrosis of brain or tumour tissue near the ventricles. Most reported instances of marked pleocytosis in the cerebrospinal fluid have been cases of tumour of the frontal lobes involving the corpus callosum. Marked pleocytosis is occasionally found in cases of tumour in the region of the pituitary fossa. The pleocytosis in these cases is probably due to localized meningitis resulting from erosion of the nasal sinuses by the tumour.

The protein content of the lumbar fluid was elevated in cases in which the ventricular walls had been involved by the tumour and in cases of tumour in the posterior fossa. The increased protein content of the lumbar fluid in cases of tumour of the brain is apparently due to exudation from vessels in the tumour, and in those of tumour in the posterior fossa, to stagnation of the fluid in the lumbar sac. In cases of supratentorial tumour
increased protein content was almost always observed when the tumour was in the corpus callosum or third ventricle. In cases of neuroma of the acoustic nerve an increased protein content of the lumbar fluid was almost constantly found, which was usually much higher than that of the fluid in cases of glioma of the cerebellum or fourth ventricle. It is probable that in cases of neurofibroma protein is more likely to be exuded into the fluid than in cases of cerebellar glioma.

The determination of the protein content of the fluid of the lateral ventricles and a comparison of the results with the protein content of the lumbar fluid are frequently invaluable in the localization of the tumour. In many cases the information thus obtained will obviate the necessity of a ventriculogram. This is especially true when the protein content of the lumbar fluid is increased.

In order to prevent the admixture of the fluid from the two lateral ventricles, the fluid should be withdrawn from the ventricles at the same time. Correction must be made for blood in the ventricular fluid. It is not necessary for lumbar puncture to be performed at the same time as the ventricular punctures. A satisfactory comparison can usually be obtained if the lumbar puncture is performed within from 24 to 72 hours before the ventricular punctures. Analysis of the protein content of the lumbar fluid or the ventricular fluid is of no value after the injection of air, since this is followed by aseptic meningitis.

The following conclusions derived from the findings may be summarized: (1) When the protein content of the lumbar fluid is high and that of the fluid of both lateral ventricles is normal, the tumour is in the posterior fossa. The increased protein content of the lumbar fluid is due to exudation from the tumour in the posterior fossa and to stagnation of the fluid in the lumbar sac. (2) When the protein content of the fluid from the lumbar region and of that from both lateral ventricles is high, the tumour is in the third ventricle or the corpus callosum, or there are multiple (usually metastatic) tumours. (3) When the protein content of the fluid from the lumbar region and that of the fluid from one lateral ventricle is high, and that of the fluid from the other lateral ventricle is normal, the tumour is on the side in which the ventricular fluid shows the increased protein content, and the tumour extends deeply enough in the hemisphere to involve the ventricular wall.

R. M. S.


Specimens of cerebrospinal fluid from 28 patients were analysed for lead by the Fairhall hexa-nitrite method.
In one of 16 cases of multiple sclerosis the fluid showed a positive result. The patient had been given sodium iodide, and the urine also showed lead.

Of 12 other cases of various conditions, in only one, a case of lead intoxication, were abundant crystals found in the cerebrospinal fluid. In this case there was 0.2 mg. of lead per litre of urine.

Taking all three studies into consideration, there is no adequate proof for, and ample evidence against, the theory that lead is an aetiological agent in cases of multiple sclerosis.

R. M. S.

SENSORIMOTOR NEUROLOGY


In this presentation an attempt has been made to show that 'central pain' and other subjective sensory disturbances occur not only in cases of pure thalamic lesions, but also in cases of peripheral, spinal cord, bulbar and cerebral lesions.

In the authors' case of lesion of a cranial nerve the subjective sensory disturbances consisted essentially of a burning sensation along the distribution of the trigeminal nerve. The neurofibroma compressing the nerve was undoubtedly the irritating factor for the production of that type of sensation.

In the four cases of lesions of the spinal cord, in addition to 'central pain' there were other subjective sensory disturbances such as burning, 'vibratory electric-like sensations' and distorted thermal sensations, e.g. cold being called hot. In most of the cases superficial sensation was impaired below the level of the lesion. The pain and temperature dysaesthesias were associated with impairment of those forms of sensation. Histological examination showed that the spinothalamic tracts were implicated. In the first case there was also slight involvement of the posterior columns in the region of the cervical enlargement. As in some of Holmes' cases, vibratory stimuli produced painful sensations. That a lesion of the posterior columns might produce 'central pain' is a remote but unlikely possibility.

The thalamus, lesions of which give the classic picture of 'central pain' and other forms of subjective sensory disturbances, was implicated in four cases. Superficial sensation (pain, touch and temperature) was impaired in most of these cases. This, best demonstrated in the first case, is of significance, as in most of the cases recorded in the literature there was more impairment of the deeper than of the superficial forms of sensibility. In the authors' first case a small vascular lesion was confined to the posterior part of the external nucleus of the thalamus along its inferolateral surface. In the other three cases the thalamic involvement was due to compression by neoplasm...