Proceedings of the Society of British Neurological Surgeons

The 80th Meeting of the Society of British Neurological Surgeons was held in Cambridge on 18-20 June 1970 as a joint meeting with the Deutsche Gesellschaft für Neurochirurgie.

HEAD INJURIES IN THE 1970S

WALPOLE LEWIN (Cambridge) stated that newer diagnostic methods might allow a better understanding of the several complications that followed head injury. Illustrations of these methods and their limitations were given. He expressed the view that the emphasis in the immediate future might well rest on the recognition that after head injury a dynamic process was set in train with resultant secondary effects, some of which could be prevented and others corrected.

INTERRELATIONSHIP OF RESPIRATORY DISTURBANCES AND CHANGES IN BLOOD AND CSF GASES AFTER SEVERE HEAD INJURY

R. A. FROWEIN and A. KARIMI-NEJAD (Köln) reported that their clinical observations and analysis of blood and CSF gases—even with a free airway—had demonstrated two forms of respiratory insufficiency after head injury or raised intracranial pressure. The first was depressed respiration—namely, alveolar hypoventilation with arterial hypoxia, hypercapnia, depressed pH, and partly decreased base excess. The other resulted in hyperventilation with hypoxia but with arterial hypocapnia, normal or increased pH, and in contrast CSF acidosis. They had observed the same changes in meningitis and in acute cerebrovascular disturbances. They suggested that the cause of arterial hypoxia in this hyperventilation syndrome might be a functional intrapulmonary ventilation perfusion inequality.

Both groups of respiratory disturbances led to hypoxia and both, therefore, required respiratory therapy. In depressed respiration the indication for assisted ventilation was usually based on the increased PaCO_2 . In hyperventilation, however, the decreased PaCO_2 indicated hypoxia and represented complex central as well as pulmonary disturbances. Treatment was not only by neuroplegics but, if the respiratory rate rose higher than 30/min, was also by assisted controlled ventilation—that is, by IPPB and slight negative expiratory pressure. The effect of assisted ventilation could be demonstrated by continuous blood gas monitoring. The results obtained in 381 patients were encouraging, even in the older age groups, but they showed the limitations of treatment in cases in which serious aspiration had occurred initially.

ELECTROLYTES, FLUIDS, AND ENERGY METABOLISM IN TRAUMATIC BRAIN OEDEMA

H. J. REULEN, M. SAMII, K. FENSKE, and K. SCHÜRMANN (Mainz) described a systematic analysis of the different factors contributing to the development of cerebral oedema as a basis of treatment of this serious complication of severe head injury. Focal cortical oedema was produced in dogs by the application of cold as described by Klatzo. The oedematous areas were outlined by vital staining by Geigy blue dye. Twenty-four hours after the infliction of the lesion, extracellular space water, electrolytes, phosphates, and some of the metabolites of glycolysis were measured in stained and unstained areas of the injured hemisphere and in tissue from the contralateral uninjured hemisphere. The cold injury produced a sharply limited necrosis of the superficial cortex, surrounded by oedematous tissue which had taken up the blue dye. In the stained cortex adjacent to the lesion a slight increase in the water, sodium, and chloride content was found but there was a much greater increase in the subjacent deeply stained white matter.

The authors developed a method of estimating the cerebral extracellular space in order to distinguish between intracellular and extracellular accumulation of fluid. This depended upon achieving the same concentration of extracellular indicator (radioactive labelled sodium-thiosulphate) in both serum and CSF by parallel intravenous and ventriculo-cisternal infusions. An increased extracellular space, compared with normal tissue, was found in both damaged cortex and white matter and was two to three times greater in the latter.

Study of the distribution of electrolytes between extra- and intracellular spaces showed that intracellular Na and Cl increased in oedematous grey and white matter while K decreased. A relationship between an increase of intracellular Na and intracellular water was then demonstrated leading to the conclusion that the increase of intracellular sodium content was one factor responsible for cellular swelling.

Results of the analysis of intermediate metabolites of glycolysis revealed a disturbance between energy production and consumption in oedematous tissue. Anaerobic ATP production by means of an accelerated rate of glycolysis appeared insufficient to meet cellular energy requirements and the energy reserves of the affected

cells were depleted. From the levels of metabolite concentrations it was not clear if the cause of the decrease in the energy-rich phosphates and the increased anaerobic glycolysis could be found in a specific disturbance of oxidative phosphorylation—for example, ferment inhibition—or in insufficiency of oxygen supply. The latter seemed the most likely explanation.

On the basis of these investigations it was concluded that the twin aims of the therapy of cerebral oedema were the removal of increased extracellular fluid in the white matter and restitution of normal vascular permeability. It was also pointed out that oxygen deficiency, due to respiratory embarrassment, shock, or hyperthermia was likely to cause irreversible damage to oedematous tissue more readily than to normal tissue.

The preceding two papers were discussed by W. Wesemann (Giessen) and A. R. Taylor (Belfast).

DIRECT MEASUREMENT OF INTRACRANIAL PRESSURE AFTER OPERATION

E. MARKAKIS, E. BUES, and A. SPRING (Kiel) described a method of direct measurement of intracranial pressure by means of a transducer incorporating a new semiconductor element. The transducer consisted of a disk 1 mm thick and 3 to 6 mm in width. Transducers, which were encased in an ultra-thin capsule of beryllium-copper, were coated with silastic rubber as protection from body tissues. Implantation up to four weeks was possible and the transducers were designed to be compatible with all types of standard strain-gauge instrumentation. The results pointed to the importance of direct pressure measurements in clinical practice by continuous monitoring. Increased intracranial pressure could thereby be detected early and treatment started immediately.

HYPOTHALAMIC LESIONS IN HEAD INJURY

C. S. TREIP (Cambridge) reported the histological examination of the hypothalamus in 16 cases of fatal head injury, some of whom showed clinical evidence of hypothalamic-pituitary disturbance, such as diabetes insipidus, electrolyte imbalance, and persistent hypothermia. The principal lesions found were:

1. Haemorrhage into and disruption of the supraoptic nucleus, due probably to shearing strains imposed by sudden movements of the brain (Treip, 1970). The end result of this acute lesion, in long survivors, was atrophy with loss of magnocellular (neurosecretory) neurones. As bilateral involvement of the supraoptic nucleus was very rare, diabetes insipidus of any severity was correspondingly uncommon (one case in this series). The paraventricular nucleus was less directly involved by trauma than the supraoptic nucleus.

2. Acute infarction of the infundibulum, due probably to interruption or spasm of the upper branches of the superior hypophyseal artery ring. This lesion, if in the midline, could also lead to diabetes insipidus. Bilateral infarction of the ventromedial nuclei occasionally occurred in association with severe electrolyte imbalance (hypernatraemia); this might have been related to disturbance of production of corticotrophin releasing factor in the infundibulum.

3. Widespread damage to the periventricular grey matter of the third ventricle was in three cases associated with persistent hypothermia. These clinicopathological correlations might be of assistance in the recognition and study of hypothalamic injury during life.

REFERENCE

Treip, C. S. (1970). In *Symposium on the Pathology of Trauma*, Royal College of Pathologists (in press).

DIAGNOSIS AND TREATMENT OF SPINAL ANGIOMAS

H. W. PIA (Giessen) described a total of 88 angiomas in 70 patients which had demonstrated that 60% of angiomas were solitary and 40% were complex, consisting of vertebral-extradural angiomas, more rarely extradural and intradural angiomas, and occasionally the simultaneous occurrence of angiomas and angio-blastomas.

Epidural angiomas, mostly cavernous and racemose, were more frequent than usually thought. The most frequent intradural angioma was the subarachnoid arteriovenous cirroid type fed by the dorsal radicular arteries.

As in the case of cerebral angiomas, the most important and constant disturbance was the local reduction of blood flow due to the A-V shunt, a spinal 'steal syndrome'. Primary compression with spinal block was unusual, even with large vessels but secondary compression of the cord was found in 50% of cases. Adhesive and cystic arachnoiditis probably caused by unsuspected haemorrhages, were the usual cause of spinal block in these cases. In the intradural lesions, however, early compressive symptoms predominated.

The difficulties of diagnosis in this condition were very apparent. In an earlier report of 54 cases, which was referred to, no correct diagnosis had been made in cases of extradural angioma and the suspicion was expressed in only three cases of intradural angioma. In more than half of the cases described acute spinal apoplexy and a relapsing apoplectic course were seen and were regarded as strongly suspicious of a spinal angioma.

The special diagnostic procedures indicated in this condition were described with particular reference to spinal ossovenography before myelography and in certain selected cases angiography according to the technique of Djindjian and Di Chiro. Myelography, however, had remained indispensable in this diagnosis.

The radical removal of these lesions, wherever possible, was recommended, and the author's experience confirmed that, although this was only possible in the past in about half the cases, at present two-thirds yielded to this method of treatment. He recommended the use of magnification, bipolar coagulation, and microsurgical instruments. He was not in favour of occlusion of feeding vessels without excision of the main lesion because 'the demand for blood' of the angioma persisted and the reduced blood flow might act to the detriment of the cord.

SPONTANEOUS HAEMORRHAGE IN THE SPINAL CANAL

TH. SCHAAKE and E.-R. SCHÄFER (Göttingen) described