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