cells were depleted. From the levels of metabolite concent-
trations 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 phosphorylization—for example, ferment in-
hibition—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 semi-
conductor 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 monitor-
ing. Increased intracranial pressure could thereby be
detected early and treatment started immediately.

HYPOTHALAMIC LESIONS IN HEAD INJURY

C. S. TREIP (Cambridge) reported the histological exa-
mination of the hypothalamus in 16 cases of fatal head
injury, some of whom showed clinical evidence of hypo-
thalamic-pituitary disturbance, such as diabetes insipidus,
electrolyte imbalance, and persistent hyper-
thermia. 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 (Treib, 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 corre-
spondingly 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 hypophysial artery ring. This lesion, if in the
midline, could also lead to diabetes insipidus. Bilateral
infarction of the ventromedial nuclei occasionally oc-
curred in association with severe electrolyte imbalance
(hypernatraemia); this might have been related to dis-
turbance 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 associ-
ated 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 extra-
dural and intradural angiomas, and occasionally the
simultaneous occurrence of angiomas and angio-
blastomas.

Epidual angiomas, mostly cavernous and racemose,
were more frequent than usually thought. The most
frequent intradural angioma was the subarachnoid
terovenuous cirsoid 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 syn-
drome'. Primary compression with spinal block was un-
usual, 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 Chiuro. Myelography,
however, had remained indispensable in this diagnosis.

The radical removal of these lesions, wherever possible,
was recommended, and the author's experience con-
firmed 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