and visualization of the middle cerebral artery in the compression angiograms facilitated the discrimination of the anterior cerebral artery and its branches from the middle cerebral artery and its branches because of the variation of contrast filling.

Subtraction of compression angiograms one from the other resulted in the presentation of flow patterns in one picture and may add to better identification of particular vessels.

REFERENCE

- Verbiest, H., and Dokkum, Th. A. (1970). Artificial slow flow carotid angiography. Carotid angiography with compression proximal to the site of injection. *J. Neurosurg.*, 32, 180-196.

CORRELATION BETWEEN REGIONAL CEREBRAL BLOOD FLOW AND ANATOMICOPATHOLOGICAL DATA

L. SYMON, J. E. REES, and J. MARSHALL (London) stated that current methods of examining regional cerebral blood flow have given valuable information about physiological mechanisms responsible for maintenance and variation of cerebral blood flow in relation to changing arterial blood pressure and pCO_2 . They described a method of isotope clearance using $^{133}\text{Xenon}$ with particular stress on accuracy of collimation to study cerebrovascular disease, especially in relation to subarachnoid haemorrhage. The details of collimation were described and the two-compartmental analysis used to obtain five analytical values for each of 16 regional detectors was outlined. Comparisons of abnormal cases with a group of normal cases in which the standard deviation of each variable for each individual area had been worked out produced valuable information which correlated well with circumstances of cerebral infarction seen either at operation or necropsy. Five illustrative cases were described and the potential of the method for the detailed analysis of regional cerebral blood flow in relation to subarachnoid haemorrhage was outlined. It seemed possible that, with increasing experience, the adequacy of perfusion of areas of brain supplied by branches of the major cerebral vessels might be subject to a reasonable assessment.

INFLUENCE OF THE CIRCULATION ON THE CSF PRESSURE WAVE

H. PONSSEN and G. C. VAN DEN BOS (Amsterdam) had investigated the influence of the pulsatile phenomena of the circulation on the form of the CSF pressure curve recorded in the cisterna magna in anaesthetized dogs. Common carotid artery flow, central arterial pressure, central venous pressure, and CSF pressure in the cisterna magna were recorded. To illustrate the interaction between the circulation and the CSF a simple model was described. The skull was represented by a rigid cylinder with two compartments. One of these represented the intracranial blood volume, the other the CSF space. They were separated by a piston which simulated the blood vessel walls and the surrounding brain tissue. The CSF space continued into the spinal canal, where it was contained in

the expandable dural sac. The spinal canal was simulated by a rigid cylinder also, but with a small cross-sectional area. The dural sac was considered in association with the peridural veins and because both were situated in the spinal canal, expansion of the former was possible only at the expense of the latter. With an increase of intracranial blood volume, the piston drove CSF into the spinal canal with a higher velocity than that of the piston, due to the difference in cross-sectional area of the cylinders. The CSF pressure in the cisterna magna was the result of the intracranial blood volume, the impedance of the spinal canal for the inflow of CSF during systole, and of the pressure in the peridural veins related to the central venous pressure. In the CSF pressure wave an arterial and a venous component could be recognized.

From the relationship between the acceleration and the deceleration phases of the flow velocity in the carotid artery and prominent points in the CSF curve it was concluded that inertial forces occurring when the CSF was driven into the spinal canal were the cause of the form of the curve at these points. These forces could occur because a small change in volume of the brain caused a larger change of the velocity (= acceleration or deceleration) of the CSF. The a-wave of the central venous pressure curve appeared in the CSF pressure curve as a small peak, just before the arterial peak. Transmission of this peak to the CSF occurred via the peridural veins. The venous CSF pressure peak never disappeared, not even when the CSF pressure was higher than the central venous pressure. The authors concluded that the peridural veins never became totally compressed and that the dural sac was always expandable.

MONITORING OF INTRACRANIAL PRESSURE IN NEUROSURGICAL PATIENTS

A. HULME, J. C. CHAWLA, and R. COOPER (Bristol) presented their experiences with continuous monitoring of intracranial pressure (ICP) and described some of its applications in clinical management. Most of the studies had been made with implanted subdural pressure transducers and only a small number of ventricular fluid pressure recordings had been made. Different types of miniature transducers suitable for insertion through a burr hole into the subdural space were described. In many cases additional recordings were made of local cortical blood flow and cortical available oxygen monitored by subdural thermistors and gold electrodes. Data were recorded by means of a modified 16 channel Beckmann EEG machine and also on analogue tape fed into a Link 8 computer.

Recordings for periods between a few hours and several weeks had been investigated by implanted transducers in 75 patients. The largest group consisted of patients with intracranial space occupying lesions, mainly tumours. In conjunction with clinical and other observations these records provided useful information of the patient's progress and the effect of therapeutic measures as they influenced ICP. Examples of such records were shown and the most obvious feature was the large plateau waves which were always pathological and represented a partial breakdown in intracranial compensation, their frequency and amplitude increasing in deteriorating

clinical states. It was demonstrated in polygraphic studies that the large plateau waves coincided with a reduction in local cortical blood flow and available oxygen. Clinical improvement after the administration of steroids was reflected in the record by the virtual disappearance of high pressure waves 12 hours after commencement of treatment. Similar records were obtained from patients with communicating hydrocephalus and 'benign intracranial hypertension'.

Eleven cases of so-called normal pressure hydrocephalus were monitored. The majority had flat pressure recordings but a few showed levels above normal at certain periods. Operation was performed in eight patients, half of whom subsequently showed improvement in intellectual performance. Two of these cases had suffered a previous subarachnoid haemorrhage.

The third group of cases were those with severe closed head injuries. Recordings were found to be particularly valuable where there was no evidence of an immediately remediable lesion since it was this group in which an insidious deterioration commonly occurred. On several occasions craniotomy had been undertaken because the recordings showed a rising ICP in the absence of clinical change. Examples were given in each group and the technical problems in relation to a fully reliable pressure transducer were described.

RELATIONSHIP BETWEEN INTRACRANIAL PRESSURE AND WATER CONTENT OF THE BRAIN IN EXPERIMENTAL BRAIN OEDEMA

J. W. F. BEKS and H. P. M. KERCKHOFFS (Groningen) had produced experimental cerebral oedema in cats by the application of cold to a circumscribed area of cortex through the intact dura. Four groups of animals were described.

Group A consisted of controls. No lesion was inflicted and hyperosmotic solutions were not administered.

Group B were killed when, after infliction of the lesion, intraventricular pressure had increased by 500 mm H₂O above its initial value. Hyperosmotic solutions were not administered.

Group C. The same procedure was followed, but when intracranial pressure reached 500 mm H₂O above its initial value a hyperosmotic urea solution was administered. The animals were killed at the lowest pressure subsequently achieved.

Group D was the same as the preceding group, except for the substitution of a hyperosmotic mannitol solution for urea.

The results of this investigation showed:

1. There was no increase in water content of the injured portion of cerebral cortex compared with the uninjured cortex.
2. There was a significant increase in water content of the white matter underlying the injured cortex.
3. There was no decrease in cortical water content after administration of hyperosmotic solutions.
4. Dehydration of the abnormal white matter was more marked with urea than with mannitol.
5. A close correlation between cerebral water content and increase of intracranial pressure was not found.

SOME ASPECTS OF SUBARACHNOID HAEMORRHAGE IN PREGNANCY

J. L. ROBINSON and C. J. HALL (Liverpool) reviewed 26 cases of spontaneous subarachnoid haemorrhage which had occurred during pregnancy. Of the 24 lesions demonstrated to be responsible for the haemorrhages, 13 were aneurysms and 11 were arteriovenous malformations. Eight were aneurysms of the internal carotid artery and of these five were operated upon during pregnancy (four by a direct approach and one by carotid ligation). The remaining eight aneurysms and all the malformations were managed conservatively. Two of the malformations were subsequently treated surgically after recurrent haemorrhage. In the whole group there had been six previous episodes of subarachnoid haemorrhage in five patients, all with malformations. Only one of these had occurred unrelated to pregnancy. Of the 13 subsequent haemorrhages, seven occurred in seven patients with malformations, and six in five patients with aneurysms.

The authors had analysed their cases in conjunction with similar series in the literature and came to the following conclusions. A demonstrable cause of the haemorrhage was more likely than in non-pregnant cases and the incidence of aneurysms and malformations was about equal. The malformations were seen in younger women (20 to 25) and most commonly bled between 15 to 20 weeks. The aneurysms occurred in an older age group (30 to 35) and caused haemorrhage usually between 30 to 40 weeks, but rarely in labour or in the early puerperium. Maternal prognosis was the same as in non-pregnant cases, except that the malformations seemed to have a greater tendency to recurrent haemorrhage. The foetal prognosis was related to that of the mother in the case of aneurysm, but was unexpectedly poor in the malformation cases both in the current and in subsequent pregnancies. The possible causes of these differences were discussed and details of neurosurgical and obstetric management were given.