isolated seizures were, in many instances, a benign disorder requiring the minimum of investigation.

ANOTHER THEORY OF THE AETIOLOGY OF THE SYRINGOMYELIC CAVITY

A. R. TAYLOR (Dundee) noted that there were four theories regarding the method of formation of syringomyelic cavities. They were:

1. That they were primary congenital dilatations of the central canal, or that they were secondary to obstruction of a spinal blood vessel.
2. That they resulted from diversion of ventricular CSF down the central canal of the cord in the presence of obstruction to the exit foramina of the fourth ventricle.
3. That CSF was driven down the central canal of the cord if jugular venous pressure were increased in the presence of blocks to the exits from the foramen magnum and fourth ventricle as a result of impaction of the cerebellar tonsils.
4. That areas in the postcentral areas of the cord became filled by CSF after necrosis in these situations. The spaces filled with fluid became enlarged in an upwards direction under the influence of increased spinal pressure caused by coughing or any other manoeuvre simulating the Valsalva phenomenon.

In the investigation described, plastic tumours had been implanted in the subarachnoid space of monkeys and rats at different levels to cause venous congestion and small haemorrhages in the grey matter only at distances cranial or caudal to the obstructions. It was concluded that the venous drainage of the grey matter runs downwards below the first dorsal spinal segment and upwards above this level. There were three longitudinal segments in the pattern of venous drainage, the first being from the foramen magnum to the first dorsal segment level, the second being from the first dorsal to the eighth dorsal segment level, and the third being from the eighth dorsal to the fifth sacral segment. Placement of expanding lesions at the watershed sites produced no venous changes in the monkey or the rat. Syringomyelic cavities spread in the same pattern—namely, upwards in the lumbar and thoracic segments and downwards in the cervical segments. It was suggested that they were formed as a result of venous congestion, haemorrhage, and necrosis, and that they spread under the influence of central venous pressure fluctuations conveyed to the subarachnoid space by way of the vertebral venous plexus. Unilateral cavities caused motor and sensory loss on the same side of the body and this could be brought about only by anterior and posterior lesions at the same level.

EXPERIENCE OF OPERATIVE ANGIOGRAPHY VIA THE SUPERFICIAL TEMPORAL ARTERY

R. A. C. JONES (Salford) noted that, while attitudes

towards control angiography in the treatment of cerebrovascular lesions might vary considerably and might have been modified by the advent of microscopical surgical techniques, a system of check angiography seemed desirable and an intraoperative method was more logical than a postoperative one. Fifty-two patients were submitted to operative angiography *via* the superficial temporal artery. Fifty of these had aneurysms (12 multiple), and two had arteriovenous malformations, each supplied from a single carotid system. The procedure was simple and did not add significantly to operating time. No complications were noted. Films were satisfactory in 50% of cases, and in the majority of the aneurysm cases a single arteriogram film was sufficient. Serial films were used when monitoring the vascular malformation operations. The procedure either failed or was unsatisfactory in 50%, the majority of failures being explained by small calibre or tortuosity of the superficial temporal artery, although vasospasm had interfered with the arteriographic demonstrations. Late check angiograms, when performed, had confirmed the operative findings.

PREDICTING CEREBRAL ISCHAEMIA AFTER CAROTID LIGATION

K. JAWAD, J. D. MILLER, W. FITCH, and J. BARKER (Glasgow) believed that measurements of the changes in cerebral blood flow (CBF) and internal carotid artery pressure (ICAP) during temporary clamping of the common carotid or internal carotid artery provided an excellent guide to the expected tolerance of permanent carotid ligation by indicating the 20% of patients who might be expected to develop hemiplegia. The authors compared measurements of jugular venous blood gases (PO_2 , PCO_2 , pH, O_2 saturation) and cerebral arteriovenous oxygen content difference to see whether they carried equal predictive powers.

Spontaneous angiographic cross filling (indicating adequate crossover channels) and bilateral wide bore (fetal) posterior communicating arteries were looked for in a retrospective study of the carotid angiograms in 87 patients. The presence or absence of these features was compared with the incidence of ischaemic complications, with the rejection rate for permanent ligation (depending on changes in CBF) in all patients, and with ICAP changes in 41.

Jugular venous blood gas measurements were carried out in 15 patients and were found to provide a poor guide to tolerance of carotid occlusion, failing to signal even severe reductions in CBF. The presence of angiography cross filling was of no predictive value whatsoever in forecasting capacity to tolerate unilateral carotid ligation. The presence of bilateral fetal posterior communicating arteries was

an unfavourable factor, being associated with a greater fall in ICAP during carotid clamping but, since most patients with this abnormality did accept carotid ligation without developing hemiplegia, the presence of this vascular anomaly was not a contra-indication to carotid ligation but merely a warning that caution must be observed.

DIFFERENTIAL DIAGNOSIS OF CEREBELLOPONTINE ANGLE LESIONS BY VERTEBRAL ANGIOGRAPHY

LINDSAY SYMON and BRIAN KENDALL (London) had used vertebral angiography for the past five years as a definitive investigation for providing details of the sites and probable natures of cerebellopontine angle lesions. Advantages of angiography were its performance under basal sedation with consequent diminution of the disturbance of intracranial dynamics consequent on anaesthesia, and its potential use as an elective investigation not necessarily urgently followed by surgery. The angiograms of 141 cerebellopontine angle masses were reviewed and plain film changes were mentioned. Erosion of the internal auditory meatus was demonstrated in 63 of 78 patients with acoustic neuromas and in two of the 22 posterior fossa meningiomas. Sclerosis of the petrous bone was evident in five of these posterior fossa meningioma cases. The six trigeminal neuromas were associated with fairly typical apical petrous erosions with smooth edges. These findings were also present in one patient with a chordoma and in one with a meningioma. Plain radiographs of the skull were normal in 15 of the acoustic neuromas, 10 of the meningiomas, and in a variety of other less common conditions.

The importance of adequate study of the venous phases in angiography was emphasized. Small masses usually first displaced a complex of veins running towards the petrosal vein which passes from the lateral aspect of the pons to the superior petrosal sinus. The typical arterial displacements associated with acoustic neuroma and trigeminal neuroma were demonstrated. The occurrence of pathological circulation in two-thirds of the acoustic neuroma cases was emphasized, and the importance of adequate visualization of both external carotid and internal carotid circulations in the detection of pathological vessels in meningiomas and acoustic neuromas was emphasized. The common source for the pathological circulation in acoustic neuromas was found to be either the dural distribution of the vertebral supply or the meningeal or ascending pharyngeal branches from the external carotid circulation. The blood supply of the angle meningiomas usually emanated either from the hypophyseal branch of the internal carotid artery or the

external carotid circulation. The vertebral circulation sometimes, but not invariably, played a part.

INTERNAL CAROTID BIFURCATION ANEURYSMS AND THEIR TREATMENT BY DIRECT SURGERY

R. P. SENGUPTA (Newcastle) described nine cases of aneurysm at the bifurcation of the internal carotid artery. All were submitted to direct intracranial surgery on the neck of the sac, and eight aneurysms were occluded by spring clips. No patient died and there was only one poor result. From an analysis of these cases and also from a review of the literature some of the characteristics of these aneurysms emerged. They accounted for about 7% of all intracranial aneurysms, appeared to be usually on the left side, and were equally distributed between the sexes. When associated with other aneurysms those at the carotid bifurcation were responsible for haemorrhage. Demonstrations of small aneurysms might be difficult, and accurate angiographic definition of the aneurysm neck might not be possible. The position of an internal carotid bifurcation aneurysm was either on the origin of the anterior cerebral artery or on the origin of the middle cerebral artery. With careful exposure and appropriate positioning of the head, it was possible to occlude these aneurysms with clips. Carotid ligation might not prevent further haemorrhage in this group of aneurysms.

RESULTS OF SURGICAL TREATMENT OF PERICALLOSAL ANEURYSMS

D. G. T. THOMAS and A. PATERSON (Glasgow) reported a series of 36 cases of pericallosal aneurysms admitted for neurosurgical management between 1960 and 1974. Direct clipping of the aneurysm had been performed in 30 patients with an operative mortality of 23% (seven deaths). A further seven (23%) had considerable disability, but 54% (16) did well. Two patients were excluded from surgery and four died before operation as a result of recurrent haemorrhage at two, six, eight, and 16 days after the initial bleed. Preoperative clinical grading, age, and sex did not correlate with the outcome. Fifteen patients (50%) were found at operation to have significant haematomas, but the results of treatment were no worse in these cases, and five of them before operation were alert without focal signs. Surgery within the first week of haemorrhage was not associated with an increased mortality and was considered to have forestalled death from recurrent haemorrhage. The results of previous series were reviewed and the evidence was considered to indicate that pericallosal aneurysms had a rather poor prognosis, although operative treatment was superior to conservative methods.