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