which might be investigated with this machine were postoperative haematomas, tumour recurrences, metastases, porencephalic cysts, and treated hydrocephalus.

Computerized scanning had already shown itself to be capable of playing a very significant role in the investigation of paediatric neurosurgical patients.

TWO CASES WHERE ABNORMAL VASCULAR ELEMENTS OBSTRUCTED OUTFLOW FROM THE THIRD VENTRICLE

R. J. BRUCE (Middlesbrough) described first a woman aged 38 years who presented clinically with symptoms and signs suggestive of right frontal tumour of two months' duration. Carotid angiography revealed evidence of ventricular dilatation. The internal cerebral vein was displaced to the right. Subsequent ventriculography showed a mass in the third ventricle posterior to the interventricular foramina. Exploration with a needle showed that the mass contained arterial blood. Vertebral angiography showed a large basilar aneurysm. After ventriculoatrial shunting there was marked improvement but the patient died six months later. It was assumed that expansion of the sac had been responsible for the fatal outcome.

In the second case, a man aged 58 years showed features of Parkinsonism with tremor of the left arm and leg associated with cogwheel rigidity. Response to L-dopa was poor, and subsequent clinical examination suggested that there might be neoplastic infiltration of the basal ganglia. Right carotid angiography showed a cerebellar arteriovenous malformation supplied from the external carotid artery. There was no indication of a contribution from the vertebrobasilar system. The venous elements formed large masses in the interpeduncular system, obstructing and displacing the proximal portion of the aqueduct. After ventriculoatrial shunting and external carotid artery ligation dramatic improvement took place but the patient died 15 months later from a subarachnoid haemorrhage.

DELAY IN DIAGNOSIS OF OPTIC NERVE AND CHIASMAL COMPRESSION

G. NEIL-DWYER (London) and J. GARFIELD (Southampton) had studied 29 patients with unilateral failing vision, subsequently shown to have optic nerve or chiasmal compression. The diagnoses were chromophobe pituitary adenoma in 16, suprasellar meningioma in 11, craniopharyngioma in one, and internal carotid aneurysm in one. In only five patients had the correct early diagnosis of a compressive lesion been made. In four patients visual deterioration had been considered to be due to refractive error. All these had chromophobe pituitary adenomas but none had undergone skull radiography or examination of the visual fields before referral. Five patients had been initially diagnosed as having retrobulbar or optic neuritis. Two of these had experienced some visual deterioration. One initially had a central scotoma but in none was there any phase of optic disc swelling or abnormality of pupillary reflexes. Four patients had abnormal skull radiographs when referred. In seven patients no diagnosis had been made and no follow-up had been instigated. In eight patients to whom miscellaneous diagnoses had been attached it was disturbing to find that the diagnosis in two had been hysterical amblyopia.

The importance of visual field examination in the diagnosis of chiasmal compression has been emphasized previously yet in this study only six patients had fields charted at initial visits. Of these six, one had a central scotoma and two had bitemporal hemianopias yet they were not referred for further investigation. The chief errors leading to faulty diagnosis had been too ready acceptance of the diagnosis of neuritis, failure to chart visual fields, failure to consider the possibility of compression in the absence of a demonstrable intraocular cause for failing vision, and failure to follow-up patients in whom no diagnosis had been made. A contributory factor had been the neglect to undertake skull radiography.

ISOLATED FITS—A NEUROLOGICAL DILEMMA

MICHAEL SAUNDERS (Middlesbrough) reported a study of 39 patients referred to an EEG department with an isolated fit. The group was compared with 39 patients with recurrent seizures matched by age and sex. Thirteen attacks were nocturnal during sleep, eight occurred just before waking, 17 were diurnal, and the time of one attack was unknown. Thirty-six presented with a major seizure, two had focal fits, and one had a temporal lobe attack. Fifteen EEGs were normal, one showed spike and wave activity, 16 showed temporal lobe abnormalities, and seven had a general excess of slow activity. For the 22 patients who were known to be well and receiving no anticonvulsants, the follow-up period varied from 10–52 months, the mean follow-up period being 26 months. Thirty-four isolated attacks were of unknown cause, one was associated with the Shy-Drager syndrome, and one was associated with birth trauma. Two were thought to be due to cerebrovascular disease and one was due to primary subcortical epilepsy.

No clear guides to prognosis were found. In this study, the majority of patients with isolated fits appeared to have a low tendency to further seizures. EEG recordings were unhelpful in distinguishing between those likely to have further attacks and those likely to remain well. It was suggested that

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

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Notes