Proceedings of the Society of British Neurological Surgeons

The 78th Meeting of the Society of British Neurological Surgeons was held in Glasgow on 19 to 21 September 1968, as a joint meeting with the Association of British Neurologists.

THE MANAGEMENT OF INTRACRANIAL HAEOMORRHAGE

From the physician's standpoint JOHN MARSHALL (London) considered the investigation of intracranial haemorrhage. As most cases of intracranial haemorrhage were admitted to general hospitals he felt that it was necessary to look at the problem from the point of view of the general physician. This was done by analysing 111 cases diagnosed as intracranial haemorrhage who were admitted to a large general hospital in 1967. The diagnosis rested on clinical grounds alone in 66 of the 111 cases, because lumbar puncture was not done. Fifty-five of the 66 died, necropsies being obtained in 26 only. In 25 the clinical diagnosis of intracerebral haemorrhage was confirmed but the 26th patient had a meningioma. Six of the 45 patients who had the CSF examined had no blood staining; of the 39 in whom the CSF was blood stained 18 were referred for angiography, 11 having aneurysms. Of the 21 not referred for angiography, 10 came to necropsy, two having aneurysms. While, therefore, advanced age and poor prospect of survival might make any investigation superfluous, in the majority of cases cerebrospinal fluid examination was an essential stage in the rational management of intracranial haemorrhage.

Surgical aspects ALISTAIR PATerson (Glasgow) discussed the surgical aspects of intracranial haemorrhage under two headings—spontaneous intracerebral haemorrhage and spontaneous subarachnoid haemorrhage. He described some of the diagnostic difficulties associated with spontaneous intracerebral haemorrhage: the patient with a slowly developing hemiplegia without disturbance of consciousness, or the patient who suddenly develops a state of confusion, with headache but little or no disturbance of consciousness or of other neurological function. When the presence of a space-occupying haematoma was suspected, angiography and sometimes even ventriculography were necessary investigations. The author analysed 100 cases ranging in age from 16 months to 85 years in which an intracerebral haematoma had been evacuated, and came to the conclusion that if the haemorrhage had a gradual onset and if the patient was in a fairly responsive state, then the results of surgery were good both as regards survival and function. The opposite applied in the treatment of the unconscious patient. He felt that co-operation between physician and surgeon was essential in the management of these cases, in contrast with the treatment of subarachnoid haemorrhage, which he regarded as a surgical responsibility. He favoured early angiography followed, except in the unconscious patient, by operative treatment aimed at excluding the aneurysm from the cerebral circulation, if possible by clipping its neck. He appealed for the opportunity for the surgeon to persist in his efforts to improve the treatment of those aneurysms which still showed a high surgical mortality and pointed out that 10 years ago basilar aneurysm was regarded as an inoperable condition, but in recent years many of these lesions had been successfully dealt with and the scope of surgery continued to extend.

MYASTHENIA GRAVIS AND THE THYROID GLAND

J. A. SIMPSON (Glasgow) showed that myasthenia gravis was a rare complication of thyrotoxicosis, being encountered only once in 3,000 cases, whereas thyrotoxicosis occurred in about 5% of myasthenics. It might precede, follow, or occur simultaneously with the myasthenia, but an apparent 'see-saw' relationship was spurious. There was no good evidence that thyrotoxicosis caused myasthenia or, conversely, that there was an inverse relationship between them. Hypermetabolism might be deleterious to myasthenia in the same way as other forms of stress. Other types of thyroid disease—non-toxic goitre, Hashimoto's disease, and primary myxoedema—were at least as common as hyperthyroidism in myasthenic patients. The incidence of all thyroid diseases in a myasthenic population was 22% in females and 10% in males and was not influenced by the type of thymic pathology. Many other myasthenics had serum antibodies against thyroid tissue. He concluded that there was a correlation between myasthenia gravis and all non-malignant disorders of the thyroid. There was evidence that the link was a genetic one, acting through a hypothalamic-pituitary mechanism.

EPILEPSY FOLLOWING THE BRAIN WOUNDS OF WORLD WAR II—20 YEARS AFTER

W. RITCHIE RUSSELL (Oxford) stated that among 560 survivors of penetrating brain wounds the incidence of post-traumatic grand mal (PTEp) was over 40%, but after 20 years fits were occurring in about 25% only, and usually with a very low frequency. Frequent fits (over six fits a year) occurred in only 5% of the total 64
patients. Small penetrating Rolandic wounds had a low incidence of PTEp, especially those involving the para-central lobule.

FURTHER STUDIES IN TRAUMATIC EPILEPSY

W. BRYAN JENNETT (Glasgow) had further explored factors predisposing to late traumatic epilepsy. He defined early epilepsy as that occurring within the first week; the vast majority of fits occurring within eight weeks of injury began during the first week. Although only a quarter of patients with early epilepsy went on to develop late epilepsy, this was four times the risk in patients without an early fit. When the post-traumatic amnesia (PTA) lasted longer than 24 hours the risk of epilepsy increased, but this proved to be associated with other complications such as intracranial haematoma or depressed fracture. Prolonged PTA alone in the absence of these complications or early epilepsy did not predispose to late epilepsy. The influence of depressed fracture on late epilepsy was explored in 550 patients from Oxford and Glasgow, with the assistance of a grant from the British Epilepsy Association. The risk of epilepsy varied from 80 to 3% according to the multifactorial influence of dural penetration, early epilepsy, and prolonged PTA. An accurate prediction depended on information about these three factors and allowed certain patients to be reassured that the chances of developing epilepsy were very small, while those in high risk categories could be given anti-convulsants for two years or so.

PAIN AND PERSONALITY

MICHAEL BOND (Glasgow), in a study of 52 women with advanced carcinoma of the cervix, related the communication of pain to medical and nursing staff to two basic personality factors—namely, neuroticism and extraversion-introversion, employing the Eysenk personality inventory. High scoring on the neuroticism (N) scale was related to liability to neurotic breakdown under stress and to a low level of physiological stimulation required to arouse an autonomic response. Extraversion and introversion were indicated by high and low scores respectively on the extraversion (E) scale and were linked with the psychological and social reaction to stimuli. Patients were asked to make an assessment of pain experienced and a record of requests for analgesics and of their administration was kept.

Analysis of these records revealed three groups, each of which differed with respect to personality structure. The first group had low N and high E scores, indicating a low degree of arousability coupled with extraversion and the ability to communicate freely. This combination of factors appeared to be linked with a delay in the development of awareness of disease. The second group experienced pain but did not communicate it to the staff. They had high N and low E scores, indicating a high degree of arousability with introversion. The third group experienced pain and communicated it to the nursing staff who responded by administering analgesic drugs. These patients had both high N and E scores. The first and second groups were regarded as 'non-complainers', and the third as 'complainers'. The author felt that there were a number of factors influencing the freedom with which pain was communicated to the staff, one being the personality of the patient and another the personalities of medical and particularly of nursing staff.

CYANIDE INDUCED OPTIC NEUROPATHY

W. S. FOULDS, I. A. CHISHOLM, JOAN BRONTE-STEWART, and T. M. WILSON (Glasgow) described the aetiology of the toxic optic neuropathies (tobacco amblyopia, the optic neuropathy of pellagrous anemia, the optic neuropathy of diabetes, and Leber's hereditary optic atrophy). The clinical features of the various neuropathies were described and it was noted that tobacco amblyopia appeared to have a dual aetiology; the effects of a toxin derived from tobacco smoke together with a disturbance of B12 metabolism. In a group of 65 cases of tobacco amblyopia, evidence of faulty B12 metabolism was present in some 40%. A correlation was demonstrated between tobacco intake and serum B12 level and between tobacco intake and B12 absorption (as tested by the Schilling test). Amblyopia occurring with a low consumption of tobacco where the serum B12 level was low and only with a heavy consumption of tobacco where the serum B12 level was normal. The points in favour of cyanide as the toxic factor from tobacco were presented and it was pointed out that the visual defect in tobacco amblyopia and in the optic neuropathy of pellagrous anemia or of diabetes responds to large doses of intramuscular hydroxocobalamin but not to the cyanide containing cyanocobalamin. In tobacco amblyopia, thiocyanate levels in the serum and urine were lower than in normal smokers but increased on treatment with hydroxocobalamin, further indicating a disturbance of cyanide metabolism in these patients. The results of treating 14 patients with Leber's hereditary optic atrophy with large doses of hydroxocobalamin were presented. Many of the patients had had atrophy for a number of years; three patients in whom the condition had been present for up to four years showed an improvement in visual acuity on treatment; four cases among those of longer duration had shown some improvement in visual field. As in tobacco amblyopia there was an increase in serum thiocyanate and urinary thiocyanate excretion on treatment with hydroxocobalamin, adding support to the view that Leber's hereditary optic atrophy was also a cyanide induced neuropathy. The results of the investigation and treatment of a group of patients with optic atrophy of indeterminate type were also presented. Some of these patients, too, improved on treatment with hydroxocobalamin.

THE NATURE AND DISTRIBUTION OF THE ISCHAEMIC LESIONS RESULTING FROM BRAIN SWELLING

J. HUME ADAMS (Glasgow), J. B. BRIERLEY (London), and J. A. N. CORSELLIS (Chelmsford) described the neuropathology of systemic hypoxia as being characterized by diffuse neuronal necrosis in the cerebral cortex and selective necrosis in the Ammon's horns. They argued that if post-hypoxic brain swelling could, by a hypo-
The compression of arteries, contribute materially to the damage in both regions, and similar diffuse pathology should be encountered in cases of unequivocal brain swelling without previous hypoxia. In the absence of detailed neurohistological studies to this end, a series of cases with proven brain swelling due to supratentorial expanding lesions was examined to determine (a) whether brain swelling with presumably increased intracranial pressure could give rise to diffuse ischaemic neuronal damage by interference with cerebral circulation, (b) if the alterations were focal, what was their distribution with special reference to the territory of the posterior cerebral arteries and to the occipital cortex and Ammon's horns in particular, and (c) the relationship between tentorial herniation and lesions in the territories of the posterior cerebral arteries.

Their material was derived from 32 cases; in 28 there was a unilateral supratentorial expanding lesion, while in four it was bilateral. The intact and sectioned brains were carefully assessed for evidence of brain swelling. Large bilateral blocks from temporal and occipital lobes and cerebellum were examined histologically but microscopical examination showed no evidence of diffuse ischaemic neuronal damage in any case. In 24 of the 32 cases they found ischaemic foci in one or both posterior cerebral arterial territories. In the 28 cases with unilateral lesions focal ischaemic alterations (but never typical hypoxic necrosis) were seen in the Ammon's horn of 16 (bilateral in seven, ipsilateral in seven, and contralateral in two) and in the occipital cortex of 19 (ipsilateral in 13, bilateral in six). Lesions in the Ammon's horn and in the occipital cortex correlated with tentorial herniation, but in neither case was the converse true. Further, lesions in the Ammon's horn were associated with lesions in the occipital cortex of the ipsilateral but not of the contralateral hemisphere.

The authors concluded that diffuse ischaemic alterations did not result from supratentorial expanding lesions (uni- or bilateral), although focal alterations in the posterior cerebral arterial territories were common. Brain swelling after a hypoxic episode in man might be absent and this conclusion was supported by physiological and pathological evidence in the Rhesus monkey. In the present series focal ischaemic lesions in Ammon's horns and occipital cortex implied a reduced blood flow in the posterior cerebral artery (or arteries) alone. A selective increase in intracranial pressure around these vessels was inconceivable, so that local mechanical factors involving stretching and kinking of these vessels must be of greater pathogenetic importance than the absolute level of the intracranial pressure.

**EXPERIMENTAL STUDIES IN INTRACRANIAL PRESSURE AND HYPERBARIC OXYGEN**

J. Douglas Miller (Glasgow) reported a study of the effect of hyperbaric oxygen at two atmospheres absolute on raised intracranial pressure in anaesthetized, ventilated dogs. In five dogs intracranial pressure was raised by inflation of extradural balloons. Hyperbaric oxygen reduced the intracranial pressure in all animals, the mean fall in pressure being 37%. However, this reduction in intracranial pressure depended on the cerebral blood vessels being responsive, as shown by a rise in intracranial pressure when CO₂ was administered. When inflation of the balloon proceeded until intracranial pressure approached systemic blood pressure and both pupils became widely dilated it was found that CO₂ would not raise the intracranial pressure, nor would hyperbaric oxygen reduce it.

In seven dogs a cryogenic brain lesion was made by applying a metal cylinder filled with liquid nitrogen to the intact skull. This produced a haemorrhagic, oedematous lesion associated with a stable raised intracranial pressure. Hyperbaric oxygen invariably caused the intracranial pressure to fall, the mean reduction being 35%. In six of these dogs the oxygen content of arterial and cerebral venous blood was measured. There was a significant rise in the arteriovenous oxygen content difference on changing from air to hyperbaric oxygen breathing corresponding to the reduction in intracranial pressure. This was estimated to be equivalent to a 19-5% reduction in cerebral blood flow. Thus cerebral vasconstriction was thought to be responsible for the reduction in intracranial pressure produced by hyperbaric oxygen.

**HISTOLOGY OF THE CONTENTS OF BLOCKED VENTRICULAR DRAINAGE TUBES**

D. Doyle (London) reported the results of the histological examination of the contents of 10 Pundenz drainage systems which had become blocked. In seven, the ventricular end was occluded and in three, the cardiac end. Four of the ventricular tubes contained plugs of viable glial tissue which comprised mainly astrocytes with large fibrous processes, capillaries, and histiocytes. The glial tissue and the capillaries extended through the side holes, presumably to become continuous with brain tissue into which the tip of the tube must have penetrated. This penetration may have been a consequence of reduction of the size of the ventricles after control of the hydrocephalus. Three of the blocked ventricular tubes contained choroid plexus, although the tubes had been inserted so that their tips lay anterior to the choroid plexus of the lateral ventricles. The choroid plexus tissue was normal-looking in one, necrotic in another, and the third comprised normal and atrophic fronds. The approximation of the tube tips and the choroid plexus could have followed either relative shifting as the brain grew, or reduction of ventricular size. Along the surface of contact with the silastic tubing the plugs showed a giant cell, foreign body reaction, with small round cells, and histiocytes. The giant cells were particularly large. In one case some of these had apparently passed through the pump and settled in the cardiac end of the tube. Three of the cardiac ends were blocked by fragments of tissue, erythrocytes, and giant cells which had probably become detached from tissue in the ventricular end.

**EIGHT CASES OF HEMIBALLISMUS: HISTOLOGICAL FINDINGS**

Marion Smith (London) described the post-mortem findings in eight patients, all of whom had had hemi-
ballismus, diagnosed by competent neurologists or neurosurgeons, during the weeks immediately preceding death. She referred to the now widely accepted association of hemiballismus with a lesion of the contralateral subthalamic nucleus but with an intact globus pallidus and pallido-fugal system. Slides of the relevant histological findings were shown and described in detail. In three cases there was a typical haemorrhagic lesion of the subthalamic nucleus. The next two cases showed partial destruction of the subthalamic nucleus by metastatic carcinoma which extended into the internal capsule, and in one case across the capsule into the globus pallidus. In these cases both cells of the subthalamic nucleus and its connections with the pallidum had been destroyed. In the sixth case hemiballismus followed a stereotaxic lesion limited to the ventro-lateral nucleus of the thalamus in a case of post-encephalitic Parkinsonism. The subthalamic nucleus was intact. The final two cases showed ischaemic lesions in the putamen. Both were diabetics who had recently changed from oral hypoglycaemic agents to the use of insulin. In all the cases demonstrated, the lesion provoking hemiballismus destroyed some part of the basal ganglia or the thalamus, but only the first two cases described gave support to the classical conception of the pathology of this condition.

RECEPTIVE APHASIA IN CHILDHOOD

F. Clifford Rose (London) described a group of 38 children who fail to learn, or may lose speech in spite of normal intelligence, hearing, and mental stability. He mentioned that some authorities disputed whether children who had not learned language should be called aphasic, but since there was a failure of symbolic understanding in this group, the term 'receptive aphasia' seemed reasonable, although there were over 30 synonyms. The group of 38 cases fell into two main divisions: those whose language development had been normal with normal babble and no history of birth trauma until the age of 2½ to 6 years, when their parents thought that they had suddenly gone deaf and within days or weeks the children had lost their speech and had epileptic attacks. Radiographs of the skull, cerebrospinal fluid examination, air encephalogram, and even brain biopsy in one case, were normal. The only abnormality was seen in the electroencephalogram, where high voltage slow rhythms were seen over both temporal regions. This abnormality, like the fits, tended to resolve spontaneously within a matter of a few months or a year or two but the language defect was much slower in recovery. There were 14 patients with this acquired syndrome and 24 in the 'developmental' group.

The sex incidence in the acquired group was equal but developmental dysphasia showed the same predilection for the male sex as did stammering, dyslexia, and most speech disorders in children. Of the 24 cases in the developmental group, six were twins, whereas there were no cases of multiple pregnancy in the acquired group. Birth trauma and prematurity were far more common in the developmental group, as were cerebral ambilaterality and neurological signs. Fits also were more frequent in the acquired group.

From his study of these cases the author suggested that the disability in the congenital group was due to brain damage.

PERCUTANEOUS CERVICAL CORDOTOMY

S. Lipton (Liverpool) stated that it was generally held that the most effective method of relieving the intractable pain of cancer was by surgical cordotomy. Unfortunately, the surgery involved seemed to deter not only patients but their doctors also, so that many patients who might need operation did not have a neurosurgical opinion. In the author's view, percutaneous electrical cordotomy could provide the benefit of surgical cordotomy without the use of surgery and appeared to be more acceptable. An added advantage was that electrical cordotomy could be repeated even in very ill patients. He went on to describe the techniques of Mullen, Rosomoff, and Lin and a film was shown of the technique used in Liverpool based on their work. Rosomoff and Mullen used an interlaminar approach to the cord between C1 and C2, while Lin's method, although technically more difficult, created a lesion in the anterolateral quadrant of the cord below the outflow to the phrenic nerve and was therefore free from the risk of respiratory complications. The procedure was carried out with local anaesthesia and sedation, the patient remaining fully conscious so that sensory and motor testing could be carried out. In unilateral cases the lesion was placed in the upper cervical cord but, if a further lesion was required on the other side, this was made on a subsequent occasion and at the lower level.

The technique consisted of placing a Harris needle in the subarachnoid space with its point adjacent to the anterolateral quadrant of the cord. By injecting air and/or a Myodil-saline emulsion around the cord at this site, the dentate ligament (Myodil) and the anterior border of the cord (air) were outlined under the image-intensifier in the lateral plane. By manipulating the needle hub the point could be aimed accurately at the anterolateral quadrant. When in position, an insulated electrode was passed along the lumen of the needle into the cord, penetration being controlled by A-P views. Stimulation up to 1 ma would show if the electrode were in the neighbourhood of the pyramidal tract by causing movement of the ipsilateral limbs. When the electrode was in the optimum position a lesion was made using radio-frequency coagulation.

A. K. Frazer (Liverpool) then described the results and complications of 97 patients treated by 127 cordotomies. Nine patients had had bilateral cervical cordotomies and two of these died from respiratory failure. The results, categorized into complete pain relief, partial pain relief, or no pain relief, were compared with those of surgical cordotomy and found to be much the same. However, there were fewer complications and the incidence of ipsilateral limb weakness was considerably less than in the surgical series, since power in these limbs could be tested while the lesion in the anterolateral quadrant of the cord was being made.
PINEAL REGION TUMOURS

T. A. H. HIDE (Glasgow) reviewed 40 cases of pineal region tumours which had been diagnosed by strict radiological criteria. There was an overall mortality of 40% and all but three deaths occurred within the first year. The author stressed the absence of papilloedema among those cases not responding to radiotherapy and among those who died before radiotherapy was possible. No other clinical factors appeared to correlate with one year mortality. All cases except one were treated with ventricular drainage procedures and all but four had radiation therapy. The presenting symptoms in those cases without papilloedema were described. It was suggested that direct surgical attack upon these patients should be considered, as at post-mortem examination several in the younger group had well-encapsulated true teratomata and 20% of irradiated cases relapsed within one year.

THE EFFECT OF VOLATILE ANAESTHETIC DRUGS ON INTRACRANIAL PRESSURE IN PATIENTS WITH SPACE OCCUPYING LESIONS

D. GORDON MCDOWALL (Leeds) with W. B. JENNETT, W. FITCH, and J. BARKER (Glasgow) referred to the previous finding that volatile anaesthetic drugs increased cerebrospinal fluid pressure in patients with normal CSF pathways at constant normal arterial carbon dioxide tension (McDowall, Barker, and Jennett, 1966). In the present paper they reported observations of intracranial pressure made in patients with space occupying lesions, mainly cerebral tumours. Cerebrospinal fluid was recorded from the lateral ventricle in such patients during general anaesthesia with controlled ventilation to produce normocapnia. After obtaining control values during anaesthesia with nitrous oxide-oxygen alone, the volatile anaesthetic under study, halothane, trichloroethylene, or methoxyflurane, was administered for 10 minutes and the resultant changes in intracranial and arterial blood pressure recorded. It was found that, in every patient studied, administration of any one of these volatile anaesthetics elevated intracranial pressure and that the increases produced were markedly greater than those seen in patients with normal CSF pathways. The extent of the increase in intracranial pressure was also related to the administered concentration of the anaesthetic—that is, after 10 minutes of administration, 0.5% halothane increased intracranial pressure by 180 (± 119) mm H₂O; 1% halothane by 278 (± 169) mm H₂O; 0.9% trichloroethylene by 279 (± 129) mm H₂O; 0.5% methoxyflurane by 71 (± 28) mm H₂O; and 1% methoxyflurane by 390 (± 135) mm H₂O. Since halothane and methoxyflurane depressed the arterial blood pressure much more than did trichloroethylene, the former anaesthetics produced greater reductions in cerebral perfusion pressure (that is, mean arterial blood pressure minus mean intracranial pressure). The increases in intracranial pressure produced by volatile anaesthetics might be disadvantageous if these resulted in gradients of intracranial pressure and thereby increased the likelihood of brain herniation. No such increases of intracranial pressure occurred if neuroleptanalgesic drugs were used in place of volatile anaesthetics to supplement nitrous oxide-oxygen (Fitch, Barker, Jennett, and McDowall, 1968).

REFERENCES


THE ANATOMY OF AGGRESSIVENESS

ERIC TURNER (Birmingham) discussed posterior cingulotomy, a psychoplastic operation for chronic aggressiveness. He observed that certain types of aggressiveness and mental instability were unaffected by either frontal or temporal operations. While ictal outbursts of rage were abolished by bilateral temporal lobotomy, and depression with or without irritability responded to inferior frontal lobotomy, chronic hostility, perpetual malevolence, constant resentment, the 'passion of hate' was undiminished by either of these procedures, and frequently was increased.

Attention was turned to the cingulate area, for ablation of this in animals had produced a decrease in aggressiveness. Experiments on monkeys had resulted in a differentiation between anterior and posterior cingulate gyri, the posterior gyri being the area which produced tameness. Ablation was accordingly performed of the posterior cingulate region bilaterally in human beings. At first the operation was confined to epileptic patients who showed the appropriate temperament abnormalities together with focal discharges in the parasagittal areas. Improvement in temperament was so striking that operation was extended to cases with appropriate aggressive features of such degree as to render them unable to live in society. The area was concerned also with sexual aberrations, and incontinence was sometimes seen in epileptic attacks. A small number of schizophrenics were operated on. Posterior cingulotomy alone did not permanently cure the delusions and hallucinations, nor did a combination of this with a modified frontal lobotomy. Posterior cingulotomy produced no observable effect on intellectual function, including all forms of memory. The type of aggressiveness under consideration was consistently reduced in 10 epileptics and one case of aggressive psychopathy without epilepsy.

A tabulation of various forms of aggression in terms of parts of the limbic lobe was proposed. The operation was described together with the anatomical background.

*This paper was given at the Preston meeting of the Society of British Neurological Surgeons in May 1967 and due to an oversight was not included in the relevant Proceedings.*