Scientific Programme of the 11th Canadian Congress of Neurological Sciences 11e Congres Canadien des Sciences Neurologiques

Winnipeg, Manitoba
June 23-26, 1976

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https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

Brain Damage After Head Injury — Initial Severity and Ultimate Outcome

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K. G. McKenzie Award — Metabolic Disturbances After Head Injury — Abnormalities of Sodium and Water Balance and Serum Cortisol

P. Steinbok, Vancouver

Eighty-eight patients with craniocerebral trauma were studied in order to assess their metabolic status after the injury. Abnormalities of serum sodium and osmolality occurred in 11 patients out of 76 who were on the study for more than 24 hours. Hypernatremic hyperosmolar states occurred as frequently as hyponatremia and hypo-osmolality. The syndrome of inappropriate ADH secretion was present in at least four patients. Abnormalities of sodium and water balance occurred only in patients with a significant head injury.

The results suggest that the ideal fluid intake after head injury is between 1500 and 1800 ml/24 hours. However, since hyponatremia tended to occur in association with middle fossa fractures, the presence of such a fracture might be an indication to use a lower fluid intake. On the other hand the presence of pyrexia or a very elevated serum cortisol, both of which were associated with hypernatremia, might be a reason for an increased fluid intake beyond 1800 ml/24 hours. The electrolyte balance should be monitored continuously following a significant head injury for up to two weeks, since hyponatremic states sometimes developed more than one week after injury.

The serum alcohol was measured on admission, and the level of serum alcohol correlated well with the serum osmolality on admission such that the degree of elevation of serum osmolality was a very good guide to the serum alcohol level. Alcohol intoxication was also associated in some instances with elevated serum sodium levels on admission. However, there was no correlation between alcohol intoxication or chronic alcoholism and the late development of serum sodium and osmolality disturbances.

Serum cortisol estimations were done in 49 patients. In 22 patients abnormalities of cortisol were noted later than 24 hours after injury. The serum cortisol was elevated or showed a reversal of the normal diurnal rhythm, with abnormalities sometime persisting for more than one week after injury. The occurrence of abnormalities was related to the severity of the craniocerebral trauma and perhaps also to the presence of an associated middle fossa basal skull fracture.

Autoradiographic Study of Muscular Dystrophy, Motor Neuron Disease and H.M.S.N.

G. Monckton, H. Marusyk, Edmonton

It has been known for a number of years that muscle protein turnover in mouse muscular dystrophy is increased. Some years ago we were able to show increased uptake of labelled amino acid into Duchenne muscular dystrophy biopsies and an abnormality in polysomal distribution with an increased incorporation of amino acid into the larger polysomes. Recently it has been shown (Nihei et al., 1973) that there is reduction of myosin synthesis in mouse muscular dystrophy.

The present study involves the incorporation of tritiated leucine into Duchenne and Becker Type II dystrophies, motor neuron disease and HMSN muscle biopsy material. The observations to be described demonstrate a difference in incorporation pattern in motor neuron disease and HMSN as compared to the Duchenne and Becker Type II dystrophy. The patients with neurogenic atrophy there were demonstrated a reduction of incorporation of tritiated leucine into myofibrils, but no associated alteration in cytoplasmic protein amino acid incorporation. These differences appear to support the view that the Duchenne and Becker Type II dystrophies are not neurogenic.

Role of Myoinositol in Alterations of Nerve Conduction Following Galactose Feeding in Rats

P. K. Thomas, A. K. Sharma and J. Jefferys, London, England

We have been able to confirm the observation of Gabbay and Snider (Diabetes 21: 295, 1972) that nerve conduction velocity becomes reduced following galactose feeding to rats. This is not explicable in terms of structural alterations in the myelinated nerve fibres. The reduction in conduction velocity is accompanied by an accumulation of galactitol and water in nerve and a reduction in the myoinositol content. Polyphosphoinositides may be involved in the regulation of ionic channels through the axolemma.

Nerve conduction velocity is reduced in experimental diabetes where the reduction is also not explicable in terms of morphological alterations. It is associated with an accumulation of sorbitol in nerve and again with a reduced myoinositol content. Greene et al. (J. Clin. Invest. 55: 1326, 1975) have recently found that the addition of myoinositol to the diet prevents the reduction in conduction velocity in experimental diabetes. In order to assess the possible role of myoinositol in influencing nerve conduction, experiments will be reported on the effects of the addition of myoinositol to the diet in galactose fed animals.

Sodium Pump Sites in Nerve and Muscle

G. M. Bray and A. J. Aguayo, Montreal

The plasma membrane $Na^+ - K^+$ - ATPase activity and its associted Na^+ - pump function are essential for the maintenance of resting membrane potentials in excitable tissues. Because the binding of tritiated-ouabain has been used to measure the density of Na^+ - pump sites, the present study applied this technique to the investigation of the pathophysiology of experimental disorders of nerve and muscle.

Segments of mouse skeletal muscle and rat sciatic nerve were incubated with tritiated ouabain. The amount of binding per mg protein was determined by liquid scintillation spectrometry.

Results: 1. At ouabain concentrations of less than 10^{-6}M, more than 50% of the binding in both nerve and muscle is K^+ - sensitive (specific); at higher cencentrations, most of the binding is K^+ - insensitive (non-specific).

2. Specific ouabain-binding sites are more dense in red (soleus) than in white (superficial gastrocnemius) muscles.

3. The density of ouabain-binding sites is reduced in soleus muscles from dystrophic mice.

4. In sciatic nerves from diabetic rats, in which conduction velocities are slow but histology is normal (metabolic neuropathies), specific ouabain-binding is not reduced.

This technique is now being applied to other experimental disorders of nerve or muscle; it could also be used to study human tissues obtained at biopsy. Because of the physiologic importance of Na^+ - pump mechanisms, it is anticipated that the measurement of ouabain-binding will be a useful technique to assess functionally-abnormal tissues prior to the appearance of frank histologic changes.

Human Sensory Nerve Refractor Period: A Simple Technique For Its Determination

C. Melmed, A. Eisen, D. Schomer, Montreal

Measurement of the refractory period in a peripheral nerve might indicate dysfunction earlier, and at times when more routine electromyographic testing is normal. Previous attempts to measure the refractory period in man have been few, and limited by the fact that the recording position was far from the point of stimulation along a mixed nerve (allowing for relative recovery of the amplitude of a test potential). Recently Tackmann and Lehmann (Europ. Neurol. 12:277 1974), have described a technique for measuring the refractory period in human sensory fibers. This technique has been modified in the present study to concentrate on an accurate measurement of the end of the relative refractory period. (For reasons that will be discussed, it is extremely difficult to make an accurate measurement of the absolute refractory period).

Double supramaximal shocks were delivered to the index finger. Fine needle electrodes were used to pick up 16 to 32 averaged responses from the median nerve at the wrist. The shock interval was reduced in a stepwise manner from 5 msec to 0.5 msec, and the amplitude of the second response was expressed as a percentage of the first.

The data from 20 normal subjects were found to fit a power curve, in which

$Y = ax^b$, or as a linear equation: $\ln Y = b \ln x + \ln a$.

The correlation coefficient r was 0.99.

Stimulus interval (msec)	1	2	3	4
Observed % of test shock	82.0	87.5	92.1	95.6
Calculated % of test shock	81.1	88.1	92.5	95.8

The calculated termination of the relative refractory period (100%), was found to be 5.72 msec. The ability to calculate this value from a regression line allows for an accurate determination of the relative refractory period and converts the test into one, that is relatively simple, and not prolonged. Results in various diseases will be discussed.

Incomplete Recovery of "Neurapraxic" Lesions

A. R. M. Upton, Hamilton

"Complete" clinical recovery within a few weeks or months and an absence of denervation potentials in weak muscles more than two or three weeks after the development of a peripheral nerve lesion are usually taken to indicate that the lesion was "neurapraxic". Partial "neurapraxic" lesions may show a slowing of nerve conduction across the site of the lesion.

Neurophysiological studies are rarely repeated after "complete" clinical recovery even though it has been shown that "complete" clinical recovery may occur if more than 10% of nerve axons are functional (McComas, Sica, Campbell and Upton, 1971).*

Serial neurophysiological studies of patients with lesions of the radial (5), anterior interosseus (10), median (10) and peroneal nerves (4) included assessment of motor and sensory nerve conduction velocities, motor unit counts, the amplitudes of sensory action potentials and needle sampling of muscles; these studies revealed that there was incomplete neurophysiological recovery in all of the patients with "complete" recovery of neurapraxic lesions, in two cases of radial palsy there was a reduction in the numbers of functional motor and sensory nerve fibres over the course of a few months **after** the onset of the original lesion. There were demonstrable neurophysiological abnormalities up to two years after "complete" clinical recovery, serial neurophysiological studies being compared over time and with the contralateral limb.

Although clinical treatment and outcome depend on whether a peripheral nerve lesion is predominantly axonal or neurapraxic, these studies may be relevant to the cumulative effects of serial trauma at one site and may well be relevant to the delayed onset of sensory and motor symptoms after a traumatic event.

Schwann Cell Kinetics in Experimental Nerve Grafts

L. Charron, A. J. Aguayo, G. M. Bray, Montreal

Although nerve grafts are used to repair injured peripheral nerves little is known about the dynamics of Schwann cell proliferation and migration in host and graft nerve segments during regeneration. The present communication describes ultrastructural and radioautographic findings in experimental autogenous sural nerve grafts.

Methods: In 36 adult mice a segment of the right sural nerve was resected and discarded. Two days later these animals received a single dose of tritiated thymidine intraperitoneally (4μC/gm/body weight) to label dividing Schwann cells in the stumps. On the third day after transection, a 5 mm portion from the opposite, left, sural nerve was grafted between both stumps of the right sural nerve (Aguayo et al, Brain Res., 105: 1-20, 1976). Animals were sacrificed from 4 days to 6 weeks after grafting. By these methods it is possible to establish: a) the population of dividing Schwann cells at the height of proliferation, b) cell migration from the stumps of the host nerves to the graft.

Results: As early as one month after grafting there was widespread regeneration and myelination of nerve fibres along the graft. By phase microscopy radioautography it was established that, in the host nerve, cells were heavily labeled in the last 3 mm of the proximal stump and also along the distal stump. The grafts contained few or no labeled cells, an indication that there had been no significant migration of Schwann cells from the stumpt o the graft.

Thus, in grafted nerves, indigenous rather than migratory Schwann cells within the graft are the supporting cells for regenerating axons. These findings underline the restricted mobility of Schwann cells along nerves regenerating in continuity.

Correlation of Electrophysiological and Morphometric Findings in Neuromuscular Disease

A. Hahn, C. F. Bolton, J. J. Gilbert, J. P. Girvin, London

The superficial peroneal nerve (sensory) and lateral fascicles of the deep peroneal nerve (motor) and a portion of the anterior tibial muscle were biopsied in 16 patients, for diagnostic reasons or as part of research programs in uremic and carcinomatous neuropathy. Six patients had no evidence of neuromuscular disease according to clinical and electrophysiological findings, eight had motor and sensory polyneuropathy of varying degrees of severity, one had motor neuron disease and one had myotonic dystrophy. Electrophysiological testing, performed in the same limb as the biopsies, included nerve conduction studies of peroneal motor and sural sensory nerves, motor unit estimates of extensor digitorum brevis (EDB) muscle and concentric needle electrode studies of lower limb muscles. Motor and sensory nerves were examined by the teased fiber technique and determinations were made of the density and spectrum of diameters of myelinated fibers. Qualitative changes in the nerves and the anterior tibial muscle were assessed by bright field and electron microscopy.

There was good correlation between the pattern of abnormalities in motor and sensory conduction studies and, in teased fiber preparations; particularly clear was the relationship between reduced conduction velocity and the incidence of paranodal and segmental demyelination. Moreover, a direct correlation was demonstrated between motor unit estimates of the EDB muscle and the density of large myelinated fibers in the branch of the deep peroneal nerve supplied by that muscle. However, needle electrode studies also showed a reasonably good correlation with muscle biopsy results; one exception, a case of mild motor and sensory polyneuropathy (documented by morphometric and electrophysiological tests of peripheral nerve) was shown by muscle biopsy to have an associated polymyositis, not disclosed by needle electrode examination. The patient who had typical muscle biopsy and needle electrode evidence of myotonic dystrophy had normal motor and sensory conduction studies, motor unit estimate, and normal peripheral nerve morphometry.

Bilateral Internal Carotid Artery Occlusion — Clinical Considerations in Survivors

C. W. McCormick, H. J. M. Barnett, London

The possibility of effective revascularization of the brain has led to reappraisal of clinical cases afflicted with occlusion of the cerebral arteries and particularly those with multiple artery occlusions. A continuing study of patients afflicted with cerebral ischemia has, in fact, demonstrated that a small but significant number of individuuals can survive bilateral internal carotid artery occlusion as well as rare instances where the vertebral arteries may be impaired or occluded at the same time. The pathogenesis of symptoms including diffuse cerebral dysfunction, the occurrence of transient ischemic episodes and the occurrence of unusual manifestations of bilateral occlusive disease must be carefully assessed in these patients so that the rationale for any proposed treatment will be understood.

Ten cases of bilateral internal carotid artery occlusion have been studied in University Hospital during a three year period and form the basis of this report.

In common with most other series all ten patients were males, with ages ranging from 48 to 66, five were diabetic. The internal carotid occlusions occurred at, or ultimately involved the origin of the arteries. Eight of the ten patients were afflicted with dementia. All cases came to medical attention because of some type of focal neurological problem and the dementia was recognized coincident with this. None came to attention because of dementia alone. None of the patients were markedly hypertensive and none mimicked lacunar states.

Unilateral neurological symptoms presented in all cases although six of the ten or initial examination evidenced bilateral signs. Most of the patients (seven of ten) had no prior clinical neurological symptoms related to the original or opposite internal carotid occlusion. Seizures occurred in this group with a frequency that was at least twice that of cerebral infarction due to unilateral carotid disease. The marked paucity of transient cerebral ischemic events once the bilateral occlusion became known will be stressed. This is in contradistinction to other reports and the important reasons for this will be discussed.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

This series will be contrasted and compared with the available literature on the subject. The histories and findings in these patients will be reviewed in relation to the possibility of the E.C.-I.C. anastomoses.

Dynamic Positron Emission Tomography For Study of Cross Section Cerebral Hemodynamics in Humans

Y. L. Yamamoto, C. J. Thompson, E. Meyer, W. Feindel, Montreal

A ring of 32 scintillation detectors is designed and constructed with the purpose of obtaining the sequential tomographic distribution of a tracer in a cross section of the head following inhalation of the positron emitting inert gas, Krypton-77 and the intravenous injection of the non-diffusible tracer Gallium-68-EDTA.

It is well recognized that the positron annihilation coincidence detection offers the basic advantage of depth independent and depth equal responses by back to back emission of 511 Kev gamma rays as compared to the photon detecting system.

In contrast to other tomographic devices, including the EMI scanner, gamma tomography and the hexagonal positron device, all of which require a number of rotations of the detecting system to obtain the final image, our system is stationary and is therefore most suitable for cerebral hemodynamic studies.

This tomographic dynamic study has the capability of studying the blood flow or perfusion rate in the territory of each cerebral artery as well as the subcompartments of the internal capsule or subcortical nuclei area without any interference from other territorial blood flow.

Preliminary studies on occlusive cerebrovascular disease, arteriovenous malformations and brain tumors, in correlation with the results from the gamma camera dynamic studies and the EMI scans, were performed in over 50 cases. This study indicates that the dynamic positron emission tomographic study is particularly useful for the determination of the hemodynamics of the posterior cerebral circulation and subcortical nuclei as compared to the other techniques.

Cardiac Arrhythmias in Acute Stroke

J. W. Norris, V. C. Hachinski, Toronto

118 patients with acute cerebrovascular lesions had continuous cardiac monitoring in addition to evaluation of other cardiac parameters, in an acute intensive care stroke unit. 37 patients initially admitted to the unit and later found not to have strokes, acted as controls.

53 (44%) of the stroke group were found to have various cardiac arrhythmias compared to 6 (16%) of the controls. The commonest arrhythmias encountered were ventricular ectopic beats and atrial fibrillation. Approximately the same proportion (12%) of stroke patients had chronic atrial fibrillation compared to the control group but paroxysmal atrial fibrillation was seen only in stroke patients. The same proportion (43%) of arrhythmias were seen in brain stem and hemispheric lesions but the incidence was less in transient ischemic attacks than in completed strokes.

In 76 stroke patients with concurrent cardiac disease the cardiac lesion may have accounted for the cerebral lesion in 26 (34%) cases mainly due to embolism. In only 6 cases (8%) could the cerebral lesion be attributed to hemodynamic causes. The insertion of cardiac pacemakers in two of these patients abolished their transient ischemic attack.

These preliminary results suggest that cardiac monitoring may aid the understanding of pathogenesis in acute stroke syndromes and the early detection and treatment of cardiac arrhythmias may reduce morbidity and mortality in acute cerebrovascular lesions.

Extracranial-intracranial Arterial By-pass in the Treatment of Dementia and Multiple Extracranial Arterial Occlusion

G. G. Ferguson, S. J. Peerless, London

The authors have had experience with 10 patients presenting with dementia and multiple extracranial cerebrovascular occlusion in whom superficial temporal artery to cortical artery anastomosis has been performed to revascularize the brain. The degree of intellectual impairment was moderate to severe in all cases. None of the patients was able to work, and two were institutionalized. In two cases the dementia had existed for more than two years. In every case co-incident focal cerebrovascular symptoms (TIA's or RIND's) were a prominent feature.

Pre-operative angiographic assessment revealed bilateral internal carotid and vertebral artery occlusion in three cases, bilateral internal carotid artery occlusion in five cases, a common carotid artery and vertebral artery occlusion in one case, and a combination of an internal carotid occlusion, a severe contralateral siphon stenosis, and bilateral vertebral stenoses in one case. Fourteen anastomoses were performed. The average follow-up is 2.8 years (7 months to 6.5 years).

Five patients are dramatically improved, two having returned to their regular work, three being able to do limited work. In two of these cases the dementia had existed for more than two years. In four cases there has been less dramatic improvement, in that none have returned to work. One case is unchanged, and remains institutionalized. The results suggest that in some instances the dementia associated with multiple extracranial cerebrovascular occlusion may be reversible to a greater or lesser degree with cerebral revascularization.

The Vulnerability of the Optic Nerve to Hypotension: A Cause of Ischaemic Optic Neuropathy

V. P. Sweeney, S. M. Drance, Vancouver

The optic nerve head has been shown to receive its blood supply through the ciliary system and not from the retinal circulation. The posterior ciliary arteries, in spite of a rich anastomes, supply the disc in a sectoral manner, thus predisposing the patient to sectoral ischaemia and nerve fibre-bundle infarction. Such micro-infarction produces arcuate scotomata similar to that seen in glaucoma. Discreet small haemorrhage in a sector of the disc is occasionally seen in association with such episodes of infarction and development of field loss compatible with optic neuropathy.

The commonest cause of ischaemic optic neuropathy is small vessel disease, such as is associated with hypertension, diabetes or giant cell arteritis. However, a larger majority are unexplained, and because of the typical field loss, are labelled as normal pressure glaucoma. We have studied 45 patients with unexplained typical arcuate scotomata matched for age and sex with patients with increased intraocular pressure. A substantially higher proportion of the former group had a history of previous shock, or a low systemic or ophthalmic blood pressure. 10 patients with preceding shock, due to bleeding, myocardial infarction or cardiac arrest, had typical nerve fibre-bundle defect type scotomata which did not progress. The majority of those without a history of haemodynamic crises showed progression of field loss presumably due to local vascular insufficiency.

It is proposed that shock may produce ischaemic optic neuropathy with changes at the optic nerve head and visual field loss indistinguishable from those caused by local vascular pathology. The peculiar sectoral distribution of the blood supply to the optic nerve head accounts for this vulnerability to hypotension. The optic nerve should be added to the recognized list of organs compromised by the shock state.

Carotid Endarterectomy: Factors Contributing to Changing Morbidity and Mortality

D. W. Griesdale, R. Fleming, H. Schutz, Toronto

A series of 221 consecutive carotid endarterectomies performed over the last 15 years has been analysed in order to detect trends in the surgical morbidity and mortality rates and the overall benefit to the patients. A comparison is made between results in the earlier years, when pre and postoperative oral anticoagulation was routine, and those in later years when anticoagulation has not been used. The surgical morbidity and mortality, and the early and late incidence of postoperative, transient ischemic attacks and strokes in these two groups is presented.

Contrary to the findings of the Joint Study of Extracranial Arterial Occlusion (J.A.M.A. 211, 1993-2003 March 23, 1970), our figures show that the presence of bilateral carotid lesions does not increase the morbidity or mortality of surgery on one of these lesions.

Current anesthetic techniques, stump pressure measurement to determine the need for bypass shunting, intraoperative heparin and other technical advances have been contributing factors to the declining surgical morbidity and mortality and improved long term results.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

Comparison of Pattern and Rate of Cerebral Blood Flow Before And After Anastomosis of the Superficial Temporal Artery to Middle Artery

P. Murray, W. Feindel, L. Yamamoto, Montreal

In recent years micro-vascular techniques have allowed neurosurgeons to perform micro-vascular anastromoses in an attempt to increase cerebral blood flow. Since little information is available in the literature concerning flow patterns and micro-regional blood flow, following extra cranial-intra cranial anastomosis, the following experiments were performed. 25 mongrel dogs weighing 45-50 pounds were anaesthetized with IV Nembutal and intubated. Pulse and blood pressure were kept constant, mean arterial blood pressure being 100 mm. of mercury. PO_2 and PCO_2 were kept constant, PCO_2 being 40 mm. of mercury. Regional cerebral blood flow was determined by the intracarotid Xe^{133}washout technique. Flow patterns were studied by Fluorescein angiography. Complete data were recorded in 19 of the 25 animals. Technical failure as a result of occlusion at the anastomosis site occurred in 1 animal. Severe vasospasm of the superficial temporal artery was noted in 1 animal, and technical errors in the recording or monitoring of the physiologic parameters negated the data from the other animals.

Control Studies showed a mean regional cerebral blood flow of 35 cc./100 gm/min. without significant difference from region to region. Fluorescein angiography confirmed a normal flow pattern. After occlusion of the middle cerebral artery the regional cerebral blood flow decreased by an average of 30%. Occlusion time varied from 1.0 to 2.5 hours. Fluorescein angiography showed variable areas of lack of filling in the middle cerebral artery distribution with an increase in the number of collateral vessels.

Post anastomotic studies showed an increase of 15-60% in 14 of the 19 animals with no significant change in the others. Fluorescein angiography confirmed patency of the graft and showed a distal shift of the watershed zone closer to normal than the post-occlusive angiogram. Hypercapnia induced by 5% carbon dioxide inhalation caused a further increase in regional cerebral blood flow. These studies support the hypothesis that focal cerebral ischaemia of 1.5-2.5 hours in the dog can be improved by extra cranial — intra cranial by-pass graft. In addition, a distal shift of the watershed zone of cortical irrigation was noted that resembled the normal flow pattern more closely than the ischaemic cortex with no anastomosis.

The Role of Clonazepam in the Treatment of Epilepsy

N. L. Auckland, Vancouver

The purpose of this open clinical trial was to evaluate the efficacy and tolerance of clonazepam in the treatment of epilepsy.

Subjects were all private patients of the author whose practice is confined to the diagnosis and treatment of the epilepsies. Therefore treatment and evaluation were carried out under the direct supervision of one person.

All patients were treated on an out-patient basis although in a number of cases, the drug was introduced while the patient was undergoing preliminary investigation in hospital.

The duration of this study extends over a four year period and the total number treated exceeds one hundred and sixty. This report however is based upon a somewhat smaller group — a number being excluded for a variety of reasons which rendered accurate evaluation impossible. In order to minimize the possibility of a placebo effect, all patients were subjected to routine E.E.G. monitoring and the report is based only on those who have taken the drug for a minimum of six months.

Methods of use in order of frequency:

1. Added to the pre-existing medications in an effort to improve control.
2. To improve the E.E.G. in those clinically seizure free but with persisting spike wave in the waking or sleep trace — frequency associated with a prominent photo-sensitivity.
3. As the principal drug, although other drugs were introduced because of 'break-through'.
4. As the sole drug.
5. To replace Zarontin in controlled patients who had been on high dosage (2000 mgms.) for several years.

Results indicate clonazepam is of definite value in primary and secondary, generalized epilepsies as well as certain partial epilepsies.

Side effects were found usually to be transient and dose related. Serious adverse effects were not encountered. Clonazepam is considered safe and efficacious in suitably selected cases and a very useful addition to our anti-epileptic pharmacological armamentarium.

Wicket Spikes — Clinical Correlates of a Newly Described Electroencephalographic Pattern

M. Lebel, J. Reiher, Sherbrooke

The purpose of this presentation is to outline the electroencephalographic characteristics and the clinical correlates of a newly described pattern, the wicket spikes. The electroencephalograms of each patients, regardless of age, who underwent recording during wakefulness and sleep in our laboratory through the years 1969 to 1975, were reviewed. Out of a total of 4,458 patients, we have retained for this study only the 39 patients in whom wicket spikes were detected both during wakefulness and sleep, in sufficient number to allow for detailed analysis. The description which follows is based upon an analysis of the 129 EEG traces obtained in those 39 patients and a review of their clinical records.

Wicket spikes are found during wakefulness and sleep, almost exclusively in adults. Their cardinal feature is a changing mode of occurrence through any single trace: from intermittent trains of more or less sustained arciform discharges resembling mu rhythm, to sporadic unitary single spikes. When occurring singly, wicket spikes can be mistaken for anterior or middle temporal spikes, since they predominate in either one area, and since they share with them other characteristics such as amplitude (60 to 210 microvolts), polarity (surface negative), duration and configuration. They are recorded over both temporal regions, independantly on either side, as a rule with a one-sided predominance. Wicket spikes ought to be differentiated from anterior and middle temporal spikes. Contrarily to the latter, interictal patterns commonly found in patients with partial seizures, the former, as an isolated finding, do not correlate with epilepsy, either partial or generalised, nor do they imply localized demonstrable, overt involvement of temporal lobe structures. Furthermore, wicket spikes do not correlate with any uniform symptom complex. Proper recognition of the pattern is a prerequisite for adequate interpretation, and for unbiased management of patients referred to an EEG laboratory.

Pattern Sensitive Epilepsy: The Clinical Spectrum, Postulated Mechanisms and Considerations on Treatment

M. W. Jones, F. Andermann, A. Wilkins, Montreal

In the group of patients with photo sensitive primary cortico-reticular epilepsy, there is a sub-group whose seizures are induced by viewing patterns under average lighting conditions. Our series consist of 8 patients with pattern sensitivity. All but one have normal intelligence and a normal examination. The seizures are virtually all provoked by viewing patterns and it is not yet clear whether these patients have any seizures under other circumstances. Effective triggers were striped clothes, floor grills at store entrances, fences, riding escalators (3/8), changing T.V. channels (4/8), and looking at "OP" art. Some of the patients had an attraction to the television screen "snow" but there is no clear evidence for deliberate self induction in this series of patients. The clinical seizure pattern includes absence attacks and generalized tonic clonic seizures with the clinical pattern age dependent just as it is in other forms of primary generalized corticoreticular epilepsy. Our findings suggest the inheritance of pattern sensitive epilepsy is as specific as in reading epilepsy or in those with photo sensitive non-pattern sensitive epilepsy. The patients are not all equally sensitive and activation by viewing patterns varies in effectiveness from patient to patient and from time to time. All patients were highly photo sensitive with the resting records frequently normal (5/8). Contrasting line patterns were effective activators as opposed to complex patterns such as checker boards etc. Sensitivity was greatly diminished by monocular occlusion. Our series of experiments following upon the work of Hubel and Wiesel suggest that these seizures are triggered by the complex cells of the visual cortex.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

Memory, Seizures and Emotion: Analgies Drawn From the Experimental "Kindling" Model of Limbic System Dysfunction

K. E. Livingston, M. Burnham, R. Racine, R. Adamec, Toronto

In 1967 Goddard demonstrated that it was possible by repeated low intensity stimulation of the amygdala and other limbic structures to induce spreading epileptiform electrographic activity culminating with time in full blown motor convulsive seizure. Massed stimuli do not induce this lasting state of seizure sensitivity which is associated with a persisting lowered after discharge threshold. Repetition of stimulation finally leads to a state of "spontaneous" seizure no longer dependent on electrical stimulus.

Analysis of the spreading electrographic activity leading to this stable seizure sensitive state, suggests that the brain has "learned", retained, and now displays on command a new and in this case abnormal pattern of motor behavior. Recently Adamec has shown in cats that there is a parallel modification of emotional responsiveness in the kindled animal. The physiological parameters of this change in emotional responsiveness parallel those seen in the evolution of motor seizure.

This experimental model provides new insight into some of the mechanisms that may be involved in learning and memory, in seizure disorders, and in some aspects of psychiatric illness.

The principal characteristics of the kindling model will be illustrated and some of the clinical implications will be commented upon.

Bilateral Motor Task (BAMT) in Assessment of Seizures

A. R. M. Upton, M. LeBlanc, D. Longmire, Hamilton

The clinical significance of paroxysmal discharges in the EEG can be estimated by assessment of reaction times, intellectual performance or continuous motor activity at the time of electrical disturbance. Unfortunately, the simplest and least expensive task, repetitive pressing of a switch in one hand, has been discarded because it is insensitive, but we have found that a bilateral and alternating motor task (BAMT) is three times more sensitive than a unilateral task; the BAMT has proved to be valuable over eight years of use, showing abnormalities in 88% of 50 consecutive patients with spike and wave or polyspike and wave discharges. Abnormalities of the BAMT included unilateral or bilateral interruptions and alteration in the rates of switch pressing, synchronisation of the two hands and perseveration. The clinical value of such a simple test was illustrated by the lack of any relationship between the length of the paroxysmal discharges and the number or type of errors in the motor task; even sinple spike and wave discharges can be associated with mistakes; medication may reduce the number of errors in the BAMT even though there is no reduction in the number of paroxysmal discharges.

The BAMT can be used during hyperventilation or photic stimulation and is reliable in patients aged five years or more.

Of interest was a reduction in the number of paroxysmal discharges in the EEG during the period of unilateral or bilateral motor activity.

The BAMT may indicate lateralisation of apparently synchronous paroxysmal discharges in the EEG, without the use of depth electrodes or stereo techniques.

There is often a DC shift in cortical potentials at the time of paroxysmal discharges and errors in the BAMT.

The BAMT provides a simple, inexpensive and relatively sensitive method of assessing the clinical significance of paroxysmal discharges in the EEG. More time consuming assessment of reaction time or intellectual activity can be reserved for those patients without errors in the BAMT; in fact, one patient with normal reaction times made errors in the BAMT.

Cervical Somatosensory Evoked Responses in Human Subjects

H. Berry, P. M. Richardson, W. J. Horsey, Toronto

It is well established that cerebral responses evoked by peripheral nerve stimulation can be recorded from the scalp and that they are altered in disease states. The variability of these responses is considerable and this has limited their clinical usefulness. It is reasonable to predict that evoked responses recorded from lower levels of the ascending sensory pathways will prove to be of greater clinical value. Early attempts to record such potentials from the cervical cord have been reported by Cracco et al. (1973) and Matthews et al. (1974).

Our method of recording the cervical somatosensory responses as evoked by peripheral nerve stimulation in the upper limb, by the surface electrode signal averaged method, is described and the results obtained in twenty-one normal subjects is noted. A complex wave form is present and appears interposed between potentials derived from the peripheral nerve, brachial plexus and the higher thalamocortical sites. Latencies for waves 1, 2, 3, evoked by medial nerve stimulation are 10.22 (SD ± 1.32), 12.32 (SD ± 1.60), 13.71 (SD ± 1.54) milliseconds with mean amplitudes of 0.4, 0.3, 0.1 microvolts respectively and similar values are obtained by stimulation of the ulnar nerve. We have found these potentials to be diminished or absent in patients with lesions of the cervical cord and our experience with this method in various clinical conditions is presented.

EEG Monitoring During Controlled Hypotension at Aneurysm Surgery

W. T. Blume, N. F. Kassell, C. G. Drake, S. J. Peerless, London

We have initiated the use of EEG as a monitor of cortical functioning during controlled hypotension at aneurysm surgery.

The only reliable data correlating blood pressure with the EEG in humans is provided by Trojaberg and Boysen (1973) who monitored the EEG and internal carotid artery stump pressure at endarterectomy. In their series, the threshold for EEG slowing varied from 46-29 mm. Hg.

In our series to date, no EEG changes and no complications have occurred while the mean systemic arterial pressures were lowered to 25-40 mm. Hg. This unexpected absence of EEG change has encouraged us to proceed at these extremely hypotensive levels, thereby facilitating considerably the technical aspects of aneurysm surgery.

The presentation will illustrate the dangers of using, instead of the standard EEG, a single channel "cerebral function monitor" which operates at a low paper speed. Differentiating a cerebrally originating electrical change from artefact is more reliably done using the EEG.

Finally, we will illustrate the marked rhythmic delta activity produced **contra**lateral to traction of the brain, its abatement during continued traction and its recurrence upon traction release.

Other aspects of EEG interpretation in this situation will also be outlined.

Electrophysiological Studies of Patients with Thalamic Stimulators for Treatment of Chronic Pain

M. D. Low, I. M. Turnbull, Vancouver

Three patients with intractable pain associated with injury to sensory pathways were treated by implantation of a chronic stimulating electrode into the principal sensory nucleus (VPM-VPL) of the thalamus. After satisfactory pain relief was obtained for a week by percutaneous activation of the electrodes, the systems were internalized and now the electrodes are activated from an external power source by way of an antenna placed over a subcutaneous receiver.

Recordings were made of the ongoing EEG and Somatosensory evoked potentials (SEP's) prior to surgery. Three or four days after implantation, recordings were obtained from the thalamic electrodes during electric shock stimulation of both Ulnar (or Median) and Posterior tibial nerves. Several months after implantation, scalp-recorded SEP's were again obtained, recording just before and over a 30-60 minute period after use of the thalamic stimulator.

The most significant findings were:

(a) Thalamic stimulation results in a temporary increase in amplitude and scalp distribution of some components of the SEP. This change persists for several minutes after stimulation has stopped.

(b) The changes in SEP parameters appears to be transient. SEP's recorded prior to thalamic stimulation, after several hours of abstinence from use of the stimulator were similar to potentials recorded under identical conditions several months earlier.

Quantitative Assessment of the Effects of Stereotaxic Dentatectomy in Animal Models of Spasticity

F. Gentili, R. R. Tasker, M. Shanlin, Toronto

The management of spasticity continues to be a well-known therapeutic problem. More recently a number of publications have recommended stereotaxic dentatectomy as an effective approach to the

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

treatment of severe spasticity in man. Unfortunately, the various series, lacking quantitative evaluation, have been confusing and often contradictory. The virtual absence of any experimental work on this subject prompted this laboratory study. We have studied the effect of dentatectomy in decorticate spasticity resulting from selective cortical removals in twenty-five squirrel monkeys and in gamma and alpha decerebrate rigidity in seventeen cats. An objective and quantitative method based on the evoked integrated E.M.G. technique has been used to measure spasticity and its reduction. Unilateral dentatectomy resulted in a significantly ($p < .05$) greater drop in tone in the ipsilateral side in nine of ten animals. The mean percent reduction in spasticity in the various ipsilateral muscle groups studied ranged from 57-81%. Further, there was a significant ($p < 0.05$) correlation between the degree of effect and the volume of dentate nucleus destroyed. Bilateral dentatectomy resulted in a significant bilateral reduction in spasticity in twelve of fifteen animals. The mean percent fall in tone for the two sides ranged from 53-78%. There was no significant ($p > 0.05$) difference between the effect in the unilateral and bilateral dentatectomy groups or between the effect on flexor versus extensor musculature. A significantly ($p < 0.05$) greater effect on upper limb versus lower limb musculature was found. Spasticity involving axial muscles and established contractures did not benefit from dentatectomy.

In contrast dentatectomy resulted in no significant ($p < 0.05$) reduction in rigidity resulting from either intercollicluar (10 cats) or ischemic (7 cats) decerebration.

Our findings suggest that dentatectomy would be of benefit in man in those spastic syndromes resulting from cortical damage but not those arising from deeper brain-stem lesions. Further our work indicates that in unilateral spasticity the lesion should be made ipsilaterally. The degree and duration of the effect will likely depend on the volume of dentate nucleus destroyed.

Fibre Systems of the Corpus Striatum in Cat Studied With the Autoradiographic Tracing Method

H. J. W. Nauta, Toronto

The efferent projections of the caudate nucleus, external pallidal segment, internal pallidal segment and subthalamic nucleus were each studied separately in experiments using the autoradiographic tracing method in the cat.

The principal advantage of the autoradiographic tracing method over methods based on axonal degeneration is that only axons whose cell bodies are located in the nucleus under study are labelled; there is no labelling of axon systems which pass through but originate outside the nucleus under study. Such axons in passage confound the results of studies based on older degeneration methods, especially when applied to the basal ganglia whose cell groups are traversed by fibre systems originating from rostral and caudal levels of the neuraxis.

From the data it appears that:

1. The caudate nucleus projects to internal and external pallidal segments and to the pars reticulata and pars compacta of the substantia nigra.
2. The external pallidal segment projects prominently to the subthalamic nucleus, possibly to the nucleus reticularis thalami, and to the substantia nigra.
3. The internal pallidal segment projects to the ventralis lateralis-ventralis anterior complex of the thalamus, to the centrum medianum, to the lateral habenular nucleus, to the substantia nigra pars compacta and to the mesencephalic nucleus tegmenti pedunculopontinus pars compacta.

Computerized Graphic Display of Results of Subcortical Stimulation During Stereotactic Surgery

R. R. Tasker, P. Hawrylyshyn, L. W. Organ, I. H. Rowe, Toronto

A computer programme has been developed for on-line use during stereotactic surgery. First, a display of the appropriate brain section is drawn on the television monitor of the computer terminal in the operating room, stretched or shrunk as need be to fit the individual patients' brain dimensions. Corrections are applied for any deviation of placement of the stereotactic frame from mid-sagittal with respect to the brain or of electrode trajectories from parasigittal. A magnified image of any desired portion of this diagram may be thrown upon the screen at will. Then, as serial stimulation is carried out for purposes of physiological localization while the electrode advances in 2 mm. steps towards a tentative target site, the observed effects are plotted suitably oriented on the brain diagram on the television monitor. Homunculus diagrams with appropriate portions shaded in are used to plot somatosensory and motor information, the quality of response being indicated by symbols, the intensity by depth of shading. Symbols suffice for auditory vestibular visual and other responses. The completed display is inspected, additional data collected if necessary and final target selection made.

All operative data, including lesion sites is then stored in a tape library which is available for scanning at anytime to produce a printout of any particular type of data either by itself or in relation to other data. This is suitably plotted on a series of sagittal brain diagrams, all corrected for variations of the individual patients' thalamic dimensions and for any errors of frame placement. Examples of such printouts tracing various pathways through the midbrain and thalamus will be shown.

Clinical Experience With the Somatosensory Evoked Potential (SEP) in Spinal Cord Injury

D. W. Rowed, J. A. G. McLean, C. H. Tator, Toronto

Somatosensory evoked potential (SEP) studies have been used in the Acute Spinal Cord Injury Unit at Sunnybrook Medical Centre for the past year. The technique is used to study nerve impulse conduction in the dorsal columns of the spinal cord.

The studies are noninvasive. The median, ulnar, and tibial nerves are repeatedly stimulated transcutaneously and the SEP is recorded via scalp mounted EEG electrodes. A mobile signal averaging computer eliminates random background EEG activity and displays the SEP. Studies are done at the bedside without the use of shielding.

Two hundred and three studies have been carried out on eighteen patients with spinal cord injury. Forty-six studies in ten patients with complete spinal cord injury did not demonstrate an SEP when the peripheral nerve stimulated entered the spinal cord below the level of injury. One hundred and forty-seven studies in seven patients with incomplete spinal cord injury demonstrated SEPs that were abnormal in wave form and amplitude. The SEP became more normal in wave form with progressive clinical recovery of the spinal cord injury.

Representative SEPs will be shown and their clinical coorelation and the implications of the findings will be discussed.

Chemical Changes in Injured Spinal Cord: Effects of Steroids and Local Cooling

R. R. Hansebout, E. F. Kuchner, D. I. Mercer, H. M. Pappius, Montreal

During the investigation of treating spinal cord injuries three animal series were conducted using two types of lesions. All animals were observed chronically and at death, spinal cords were analyzed for electrolyte content and dry weight.

Series No. 1: Cats received a T13 cord impact injury and were untreated or received parenteral steroids. Edema and K^+ loss were observed at the site of impact and extended several segments from there. Steroid treatment prevented K^+ loss but not edema ($Na^+\uparrow$ $K^+\downarrow$ and D. W.$\downarrow$) and improved motor recovery which correlated with K^+ preservation.

Series No. 2: Dogs received moderate or severe cord impact lesions and were untreated, received steroids or local cord cooling. Edema was confined to the impact area while K^+ loss was noted even in adjacent segments and this loss was decreased by steroid treatment but not cooling. While K^+ loss was proportionate to the severity of injury, moderately injured dogs recovered while severely injured dogs remained paralysed.

Series No. 3: Dogs underwent a spinal cord compression injury and were untreated, received steroid and/or local cord cooling. Both modes of therapy enhanced motor recovery. Edema presence and K^+ loss were seen only at the site of injury and K^+ preservation was not altered by treatment, but correlated with motor recovery.

Conclusions:

1) Edema following impact injury differs in dogs and cats.
2) Impact and compression injuries cause different biochemical alterations.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

3) K+ loss is prevented by steroid treatment but not by cooling
4) K+ preservation may not be the only cause for functional improvement following spinal cord injury.

Post-Traumatic Infarction of the Spinal Cord

A. N. Sandler, C. H. Tator, Toronto

Quantitative regional spinal cord blood flow (SCBF) was studied in monkeys subjected to acute compression injury of the thoracic cord. SCBF was measured by the ^{14}C-antipyrine autoradiographic technique for 24 hours after moderate or severe injury of the cord. With this method regional SCBF can be measured in areas of the cord as small as 0.1 mm^2, and flow in grey and white matter can be resolved. The cord injuries were produced by the inflatable circumferential extradural cuff technique.

After moderate injury SCBF was reduced for the first hour after trauma in both grey and white matter. In white matter flow returned to normal by 6 hours and by 24 hours there was a hyperaemic response. In grey matter flow remained lower than normal for 24 hours after trauma. After severe injury, SCBF was markedly reduced in both grey and white matter for 24 hours after trauma. Post-traumatic ischaemia of the cord was present over a 5-6 cm segment of the cord although the inflatable cuff is only 4 mm wide. At 24 hours the ischaemic zones could be histologically identified as infarcts. The mechanisms involved in producing post-traumatic infarction of the cord are not known.

The results of this study indicate that blood flow in the spinal cord is markedly altered by trauma, and that post-tramatic infarction of the cord may be an important reason for the poor recovery usually seen after major spinal cord injuries.

Spinal Reflexes and the Concentrations of 5H1AA, MHPG and HVA in Lumbar CSF Following Spinal Lesions in Man

P. Ashby, M. Verrier, J. J. Warsh, K. S. Price, Toronto

In the mammalian nervous system there are a number of neurotransmitter-specific pathways running from the brainstem to other parts of the nervous system. The dopaminergic pathway which originates in the brainstem and modulates activity in the basal ganglia is an example of an ascending neurotransmitter-specific pathway. There are, in the cat at least, descending neurotransmitter-specific pathways from the brainstem that have important modulating effects on spinal reflexes. Activation of the terminals of a descending bulbospinal noradrenergic pathway, for example, permits walking movements in the spinal cat. Are there pathways with similar function in man?

In the present study the assessment of activity in spinal reflex pathways has been combined with the estimation of the lumbar cerebrospinal fluid (CSF) concentrations of 5 hydroxyindolacetic acid (5HIAA), 3 methoxy-4-hydroxyphenylglycol (MHPG), and homovanillic acid (HVA) in 12 patients with complete or virtually complete spinal lesions in order to explore relationships between spinal reflex pathways and specific neurotransmitters in man.

The concentrations of 5HIAA and MHPG in lumbar CSF are reduced following complete or virtually complete spinal lesions in man. This may occur within 18 days of the lesion. MHPG concentrations appear to be inversely related to the level of the lesion. The HVA concentration in lumbar CSF is reduced when there is obstruction of the CSF pathways. There is no apparent relationship between the concentrations of 5HIAA or MHPG in lumbar CSF and the activity in the spinal monosynaptic pathway (estimated from the proportion of the motoneurone pool activated by the H reflex or Achilles tendon reflex) or the activity of a spinal inhibitory mechanism (estimated by the degree of vibratory inhibition of the monosynaptic reflex). Patients with a tonic vibration reflex (TVR) tended to have higher MHPG levels. There appeared to be an association between low CSF HVA and enhanced vibratory inhibition of the monosynaptic reflex in the 9 patients whose spinal lesions were complete.

Experimental Allergic Myasthenia and Cellular Sensitization to AChRP

W. Sheremata, M. Eldefrawi, M. Moscarello, Montreal

Experimental allergic myasthenia (EAMG) has been induced in a number of species using Acetyl choline receptor protein (AhRP). Although antibody production to AChRP has been studied extensively, investigations by Engel et al. have shown the initial damage to the motor end plate to be immunologically mediated by cell elements. We, therefore, sought to determine, using the macrophage migration inhibition factor (MIF) assays, to find an **in vitro** correlate of these observations, and compare the results to those obtained with basic myelin protein (BMP), crude muscle (CM), and crude peripheral nerve (CPN).

Eight Hartley strain guinea pigs were sensitized with 10 μ AChRP (Group I), 8 with 20 μ (Group II), and 8 with 40 μ (Group III) in Freunds complete adjuvant. Peritoneal macrophages of eight normals and test animals were studied concomitantly and challenged with 10 μ (BMP) 0.1% w/v (pn), 0.1% CM., 0.2% CM, and 5 μ, 10 μ, 20 μ, 70 μ/ml of AChRP. The migration inhibition index (MI) was calculated by observations of normal cells with and without antigen.

No significant inhibition was seen in animals sensitized with 10 μ of antigen, but mild clinical illness was seen in the others; except for one which became paralyzed and found dead in the cage. MI for Groups II and III together were 114 for BMP, 71* for PN, 55* for CM, 28* for CM., 74 for 5 μ AChRP, 75* for 10 μ AChRP, 62 for 20 μ. Normals showed 104, 101, 100, 80, 100, 80, and 55 for the respective antigens.

Significant sensitization to AChRP and crude muscle was anticipated but sensitization to crude nerve was not. The data supports the observation of cell mediated hypersensitivity to AChRP in the pathogenesis of EAMG. It also suggests the presence of similar antigen in peripheral nerve.

*$p \leq 0.01$

Myasthenia Gravis, Multiple Sclerosis, and Cellular Sensitization to Acetyl Choline Receptor Protein

W. Sheremata, M. Eldefrawi, H. Triller, I. Heller, Montreal

Sensitization to acetyle choline receptor protein (AChRP) has been shown to induce cell mediated destruction of the motor end plate and antibody production.

Preliminary investigations using lymphoblast transformation assays have shown evidence of cellular sensitization to AChRP in myasthenia gravis, its experimental model, but not in other situations. However, because of the frequent clinical signs of ptosis and proximal muscle weakness in multiple sclerosis (MS), and recent neurophysiological evidence we determined to study a large group with multiple sclerosis, and myasthenia gravis as well as a miscellaneous group (strokes 7, senile dementia 6, Guillain Barre 3, SSPE 2, brachial neuritis 2, encephalitis 2, and hysteria 2).

Twenty-four normals, 24 myasthenics, 24 multiple sclerosis patients and 24 with miscellaneous diseases were studied using the direct cell migration inhibition assay system and purified torpedo AChRP. Normals gave a % migration of 100 $\pm$ 14.5 (S.D.), myasthenics 91 $\pm$ 32, multiple sclerosis 88 $\pm$ 30, and misc. 103 $\pm$ 36. No normals showed significant inhibition, but all but one myasthenic (9 patients) with renewed symptoms showed significant results. One MS patient with acute myasthenic features and 7 others also showed positive results. None with stroke, senile dementia, Guillain-Barre, SSPE, brachial neuritis showed significant results. However, 2 with "hysteria" did.

The results show using another parameter of cellular hypersensitivity that sensitization to AChRP is present in myasthenia, but shows that such sensitization occurs as commonly in MS. The data suggest that myasthenic features in MS may possibly be related to such sensitization. The findings in hysteria underline the limitations of our clinical ability to recognized early nervous system disease.

HLA Typing in Familial Multiple Sclerosis

G. C. Ebers, L. R. Espinoza, J. B. Zabriskie, New York City

HLA-B7 typing was performed in 27 patients with familial multiple sclerosis (MS) from 13 families.

The incidence of HLA-B7 was not significantly increased in this patient group over that seen in non-familial MS. When this antigen was present in a given family, it did not segregate with the disease. Similar findings were obtained in typing for DW2. Affected members within a family were likely to share a haplotype. Linkage analysis with an hypothetical MS gene has been made.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

It is considered that these findings in a small number of patients may reflect the importance of environmental factors in these families or the presence of an additional non-HLA 7 linked genetic factor(s) leading to complementation of the immune response.

Neurological Complications of Gold Therapy

S. H. Huang, E. M. Ashenhurst, V. L. Flatt, D. M. Mitchell, W. E. DeCoteau, Saskatoon

Although neurologic complications are thought to result infrequently from gold therapy, the problem may be more common than generally recognized, as evidenced by several patients that we have encountered in the past few years.

The first patient was a 62-year old male with a two year history of inflammatory polyarthritis, thought to be rheumatoid arthritis (RA). Gold was started in January, 1974. In September, 1974, he developed symptoms of muscle spasm, weakness, weight loss and was found to have severe generalized muscle fasiculations. A clinical diagnosis of motor neuron disease was made, substantiated by electromyographic findings. Gold was discontinued in October, 1974, and he experienced progressive improvement. By September, 1975, he was neurologically well. (A short film documenting the clinical course will be presented.)

The second patient was a 43-year old woman with a two year history of RA. After having received 1 gram of gold, she was noted to have episodoes of confusion, disorientation, memory loss, inappropriate affect and was thought to have presenile dementia. Some muscle fasiculation was also noted. Gold was discontinued and she made a progressive and complete recovery and was found to be mentally normal within four months.

The third patient was a 50-year old female with a two year history of RA. After having received 600 mgm. of gold, she developed a profound loss of the sensations of taste and smell. She made a slow, but complete recovery one year after discontinuation of gold.

We believe that gold toxicity can be implicated in the pathogenesis of all 3 of these patients' neurologic abnormalities for the following reasons: 1) All 3 patients developed the neurologic symptoms and signs after having received significant quantities of gold. 2) After the onset of neurologic disturbance, the first 2 patients noted exacerbations of their neurologic symptoms after each gold injection, until the medication was discontinued. 3) All three patients have made a steady and complete recovery after the discontinuation of gold.

We have also recently encountered a patient with RA and multiple sclerosis (MS). She was treated with gold, and after 10 weeks had a catastrophic exacerbation of the MS. She has made a partial remission after gold was stopped. It is unclear whether gold can be implicated in this patient's disease exacerbation, but since there may be a statistical association between MS and RA, perhaps gold therapy should be administered to such patients with great caution.

Canadian Neurological Society Award — The Role of Blood Velocity in Pathogenesis and Management of Cerebral Vascular Disease

Dr. J. D. Spence, San Francisco

The treatment of high blood **pressure** prevents death from congestive heart failure, hypertensive nephropathy, and hypertensive encephalopathy, and strokes from cerebral arteriolar disease. (Lacunes, haemorrhage from microaneurysms). However, atherosclerosis, manifested as coronary artery disease, is just as frequent a cause of death in well-controlled hypertensives as in poorly-controlled patients. Increasing evidence suggests that increased blood **velocity** by causing turbulence and high shear rates at the endothelial surface of arteries, may be important in the pathogenesis of atherosclerosis. Turbulence has been observed in cerebral berry aneurysms. In order to measure the effects of antihypertensive agents on blood velocity, a new method of analyzing Doppler ultrasound velocity recordings has been developed. Studies in Rhesus monkeys show the following: In doses which reduce diastolic pressure by 13-28%, Propranolol **decreases** mean blood velocity (MV) by 17%, Clonidine decreases MV by 14%, while Methyldopa **increases** MV 12%, and Hydralazine increases MV by 52%. ($p < .00001$). It is hypothesized that enlargement of berry aneurysms, the progression of cerebral atherosclerosis, and embolism from carotid lesions might all be decreased by the selection of antihypertensive agents which decrease blood velocity.

Disciplines of Diagnosis in Neurology

J. C. Richardson, Division of Neurology
University of Toronto, Toronto, Ontario

Carotid Ligation — New Safety For an Old Operation

B. Jennett, Division of Neurosurgery
Institute of Neurological Sciences, Glasgow, Scotland

Thomas Willis and Headache

F. L. McNaughton, Division of Neurology
McGill University, Montreal, Quebec

Experimental Lipid Storage Myopathy

A. K. W. Brownell, Calgary, A. G. Engel, Rochester

In several instances of human lipid storage myopathies not due to carnitine deficiency the biochemical defect has not been elucidated. To investigate mechanisms resulting in muscle fiber lipid excess, 2.5 g/kg brominated vegetable oil (BVO), which gives rise to alpha-brominated medium and short chain fatty acids during beta-oxidation, was given to rats by gavage daily. Sex- and weight-matched animals received corn oil. BVO treated rats became weak during the first week of the study. Morphometric analysis for lipid content of electron micrographs obtained by stratified random sampling revealed an approximately 5-fold increase in soleus, 10-fold increase in diaphragm (p values < 0.001) and no significant change in superficial medial gastrocnemius after 7 or 21 days of BVO treatment. The oxidation of labeled long and medium chain fatty acids and ketone bodies was markedly inhibited (p values < 0.001); oxidation of other substrates and activities of the carnitine transferases were normal. BVO treatment reproduces the morphologic features of human lipid storage myopathies presumably by depressing intramitochondrial beta-oxidation of medium and short chain fatty acyl residues. The present study also provides a scheme for investigating human lipid storage myopathies of unknown cause.

The "F" Wave: Its Physiology, and Its Use in Determining Proximal Motor Conduction Times

D. Schomer, A. Eisen, C. Melmed, Montreal

There are no established methods for determining the velocity of fast conducting **motor fibers** over their central segments ("H" reflex studies using in part sensory fibers). Recent studies by Kimura (Neurology 24:539, 1974, Arch. Neurol 32:524, 1975), have utilized "F" wave conductions to investigate the velocity of central segments of the median and ulnar nerves. The origin of the "F" wave is not entirely clear, although there is some evidence indicating that it is independent of sensory fibers. The present study was undertaken (1) to further investigate the physiological basis of the "F" wave, and (2) having shown that its latency indeed appears to be independent of sensory fibers, determine the velocity of the proximal segments of the median, ulnar, peroneal and posterior tibial nerve motor fibers in man.

"H" and "F" waves were elicited from the soleus-gastrocnemius complex in normal subjects, and patients with absent ankle jerks. In the former group the "H" wave had a latency of about 5 msec shorter than the "F" response. Furthermore the "F" response was obtainable despite the absence of an "H" response (absent ankle jerks), indicating that the "F" wave is independent of IA afferents and is carried in fibers that are of a slightly slower maximum conduction (ie motor fibers).

The distal conduction times determined from "M" wave latencies and "F" wave latencies in a large group of subjects, were very similar for any given nerve.

	Distal "M" wave conduction time (ms)	Distal "F" wave conduction time (ms)	Proximal "F" wave conduction time (ms)
	E - W	E - W	Cord to E.
Median	4.4±0.55	4.1±1.0	6.85±0.63
Ulnar	4.5±0.46	4.1±0.82	7.3±0.68
	K - A	K - A	Cord to K.
Peroneal	7.2±1.1	6.5±0.8	13.4±1.9
Post Tibial	7.9±0.94	7.5±2.1	13.4±1.4

E - elbow, W - wrist, K - knee, A - ankle.

Proximal "F" wave conduction times were in accordance with the expected maximum motor conduction velocity from cord to elbow, and cord to knee respectively, assuming a 1 msec central delay.

The value of determining proximal motor conductions in a number of pathological conditions will be discussed.

Scapulo-Peroneal "Dystrophy"

A. S. Gordon, S. P. Starkman, K. P. H. Pritzker, F. Langer, Toronto

Muscular atrophy in a scapulo-peroneal distribution was first reported by Davidenkov in 1926. Since then, both mypathic and neurogenic forms have been described with autosomal dominant and X-link recessive inheritence. We have recently studied two Italian families in which this condition appears using electromyography, serum creatine phosphokinase, and muscle biopsy. The biopsy was evaluated with histochemistry, histograms, and electromicroscopy. In the "C" family, the diagnosis was difficult because the 50-year-old male proband first displayed pelvic girdle weakness at age 46. It was not until later that the scapulo-peroneal distribution was apparent. Both muscle biopsy and electromyography showed predominantly myopathic changes although there were neurogenic features as well. One biopsy showed type I and II fibre hypertrophy and atrophy of type I fibres. The proband's 20-year-old son had an elevated creatine phosphokinase (CPK) of 150 and paraspinal muscular atrophy, whereas one daughter was normal. In the "M" family, the 12-year-old proband had delayed milestones, a significant foot drop, and proximal pelvic girdle and shoulder girdle weakness. Her CPK was 750 units and the EMG was suggestive of a myopathy. A biopsy of a clinically strong muscle, the gastrocnemius, showed focal muscle necrosis, regeneration and mild hypertrophy. Her 8-year-old brother was similar clinically with a CPK of 762 units. The 10-year-old clinically normal brother had a CPK of 103 units. The mother was normal but the father showed evidence of mild deltoid and paraspinal atrophy and a CPK of 104 units, Genetic studies are continuing in both families but the pattern appears to be autosomal dominant or possibly X-linked recessive in the "C" family and autosomal dominant with incomplete penetrance in the "M" family. The different muscle biopsy results may reflect sampling, stage of the disease as well as etiology.

Ulnar Neuropathies in Rheumatoid Arthritis

A. R. M. Upton, J. Darracott, F. A. Bianchi, Hamilton

There has been much controversy about the possible causes of ulnar deviation of the fingers in patients with rheumatoid arthritis; one hypothesis attributes such deviation to selective impairment of the deep palmar branch of the ulnar nerve.

A neurophysiological study of 33 patients (age 43.3 ± 14 years. Female, 26; Male, 7) with rheumatoid arthritis included assessment of motor and sensory nerve conduction velocities, the amplitudes of sensory action potentials, motor unit counts (thenar, hypothenar, first dorsal interosseus and extensor digitorum brevis muscles) and needle sampling of muscles.

There were carpal tunnel syndromes in 7 (21%) of the patients; other abnormalities included slowing of motor nerve conduction at the elbow in four (12%) patients and a polyneuropathy in five (15%).

Motor latencies from the ulnar nerve at the wrist to the hypothenar and first dorsal interosseus muscles were not significantly different in seven patients with ulnar deviation (2.27 ± 0.17; 3.2 ± 0.27 msec) and those without (2.37 ± 0.28; 31.8 ± 0.27 msec). Motor unit counts in hypothenar and first dorsal interosseus muscles were lower in those with ulnar deviation (229.1 ± 57; 306 ± 71.2) than in those without (314.8 ± 145; 336 ± 121.2) but these differences may be due to the older age (63.9 ± 6.23 cf 54.2 ± 11.97 years) and the longer duration of the disease (15.71 ± 16.8 cf 11.19 ± 12.13 years) in those patients with ulnar deviation of the fingers; in favour of this possibility was the reduction in thenar motor unit counts in those patients with ulnar deviation (149.4 ± 70.9 cf 178.7 ± 88.7).

These results indicate that ulnar deviation of the fingers in patients with rheumatoid arthritis is unlikely to be due to selective impairment of the main trunk or the deep palm branch of the ulnar nerve.

A New Spino-Cerebellar Syndrome With Early Mental Retardation and Seizures

S. J. Rothman, C. W. Olanow, Montreal

A kinship of eight children will be presented. Two died of congenital anomalies in infancy and the remaining six children have been studied extensively.

Each child showed a remarkably similar clinical picture, but the parents were unaffected. Global developmental delay, behavior disorder and seizures were the dominant features in the first five years of life. In the latter half of the first decade, progressive gait ataxia, cortico-spinal tract signs, kyphoscoliosis and pes cavus developed. There were no clinical sensory abnormalities at any time and amyotrophy was not a prominent feature. However, significant mixed peripheral neuropathy of the axonal degenerating type was demonstrated by EMG studies and nerve and muscle biopsies. Intellectual abilities deteriorated and were in the trainable range in five of the six children. The behavior tended to improve with increasing age and the seizure disorder spontaneously resolved. Nevertheless, all required institutional care.

It is felt that these children present a unique variant of an heredofamilial spino-cerebellar degenerative disorder. The clinical picture, the genetic pattern and the pertinent laboratory tests will be discussed.

Emulsified Oil Ventriculography In Early Diagnosis and Management of Hydrocephalus in Newborns

F. B. Maroun, W. D. Heneghan, J. C. Jacob, St. John's

A technique is described of outlining the ventricular system in the newborn, in cases of overt spinal dysraphism. Ethiodan emulsified with C.S.F. is introduced into the lateral ventricles; gentle agitation of the head permits dispersal of the emulsified contrast medium through the ventricular system. A "cast" of the ventricles in then easily and graphically demonstrable by conventional skull radiographic technique.

Serial skull x-rays in neonates (after repair of overt spinal dysraphic lesions) allows a follow up on the ventricular size as the contrast medium adhers oo the ventricular wall and remains for several days.

The usefulness of this simple technique will be illustrated by appropriate clinical material.

Late Onset GM_2 Gangliosidosis (Juvenile Tay Sachs Disease) in Two Lebanese Families

E. Andermann, F. Andermann, S. Carpenter, G. Karpati, D. Grimes, L. S. Wolfe, Montreal and Ottawa

Only 16 cases of late onset GM_2 gangliosidosis have been described in the world literature, 10 of which have been confirmed enzymatically (Brett et al., 1973). The onset of symptoms is either in the late infantile or juvenile period, and the deficiency of hexosaminidase A may be partial, or as profound as in classical Tay Sachs disease.

We have investigated two brothers of Lebanese extraction, aged 5 and 4 years, product of a first cousin marrige, who presented with progressive ataxia, spasticity, dystonia and mental deterioration from the age of 2½ years. They rapidly became mute, developed optic atrophy, but had no seizures. Muscle biopsy revealed denervation atrophy. Nerve and skin biopsies were not diagnostic by light and electron microscopy. Brain biopsy showed lipid storage in neurones and axons. Serum hexosaminidase A levels were very low in both brothers (1.7 and 2.1%), whereas the parents had intermediate values (39.5 and 45.0%), similar to those seen in heterozygotes of classical Tay Sachs disease. In reviewing our cases of cerebro-macular degeneration, we noted another Lebanese boy, also the offspring of first cousin parents, who died in 1953 at the age of 12½ years with an autopsy diagnosis of juvenile cerebral lipidosis, type uncertain. The parents were traced, and hexosaminidase determinations showed that they were also carriers of late onset GM_2 gangliosidosis.

The two families come from adjacent towns in Southern Lebanon, and can probably be traced to a common ancestor. Many of the family members have migrated to the Montreal and Ottawa regions. To date, we have carried out carrier detection and genetic counselling in 37 family members, and are planning to extend this to other individuals in these large families, as well as to other members of the Lebanese community, particularly those originating from the same areas of Lebanon.

A Numerical Taxonomic Study of Aphasia

A. Kertesz, J. Phipps, London

The first numerical taxonomic study of aphasic patients is presented. 142 aphasics with cerebral infarcts were subjected to classification by a clustering algorhythm on the basis of 5 clinical attributes. Fluency, information, comprehension, repetition and naming subtests of the Western Aphasia Battery (WAB) were used. The scores were weighted on a 1-100 scale to represent equal difficulty level.

Ten major clusters were corresponded more or less to recognizable clinical groups of aphasics. These were Global, Broca's, Isolation, Transcortical Motor, Transcortical Sensory, Wernicke's, Afferent Conduction, Efferent Conduction, Semantic and Anomic Aphasia. One of the most interesting findings was the bimodel distribution of the so-called "conduction" aphasia. The contribution of various clinical attributes to the clustering arrangement is mathematically analysed (principal component analysis). The separation of the groups obtained was found to be statistically valid.

The results of taxonomic analysis permits an objective grouping of aphasics and the clinical characteristics defining them. The method is similar to the objective classification of plants and other species. This study attempts to provide objectivity and precision for clinical classification which are very much needed for reliable research in aphasia.

Essential Tremor That Is Not "Benign"

A. H. Rajput, Saskatoon

The benign nature of essential (familial) tremor is so widely recognized that it is frequently referred as "benign essential tremor" (BET). This study was undertaken to evaluate the disability in the patients once diagnosed by a qualified neurologist as BET. Sixty-six patients with average age of onset of tremor at 44.1 years had last examination at average age 59.6 years, which forms the basis of this report. The tremor was fine in 31, medium in 26, and coarse in 9 patients. Other neurological abnormalities included: cerebellar dysfunction in 8, spasmodic torticollis (ST) in 6, and evidence (excluding ST) of two other parts of nervous system involvement (MSA) in 6 patients. Other systemic diseases were seen in 24 cases — the most common was hypertension, in 10 patients (twice the expected frequency). Of the 24 patients eligible for employment, 4 retired prematurely, 1 was demoted, and 6 were seriously considered for early retirement due to neurological problems; indicating that 45.8% of the eligible workers had significant functional neurological deficit. These data demonstrate that all cases diagnosed as BET do not have a "benign" ailment. The high incidence of cerebellar dysfunction and MSA cannot be explained as incidental concurrent illnesses. It is most likely that these patients in fact suffer from another disorder which is indistringuishable from BET at an early stage. The high incidence of ST in the BET patients may indicate some common pathophysiological basis to these two clinical manifestations, since ST cannot be clinically mistaken for BET and vice versa. The diagnosis of BET is made on the basis of clinical features alone, and the effect of alcohol on tremor is of no diagnostic significance (Rajput et al., Can. J. Neurol. Sc. February 1975). Better diagnostic criteria are needed to recognize the patients with unfavourable prognosis early in the course of illness. Distinguishing features of each group with unusual manifestations will be discussed.

Transcutaneous Neurostimulation For the Relief of Chronic Pain

D. Fewer, N. C. Hill, M. Driedger, Winnipeg

Over the past Eighteen months we have evaluated the ability of the Neuro Stim transcutaneous unit to relieve chronic pain states as they have presented to us in a general neurosurgical practice.

A total of Fifty-seven patients were treated, all but three on an outpatient basis. Five patients were considered unevaluable. The overall early response rate was thirty-three percent, with response simply defined as a relief or reduction of pain as reported by the patient. In patients being treated for pain related to cancer, the response rate was the same.

Sixteen of the patients completed a protocol in which each individual served as his own control by being alternately treated by the standard procedure and then by a set up in which the neurostimulator (without its battery) was connected to a machine emitting a combination of light and sound stimuli. In the protocol group, seven of the sixteen did not respond to either the standard stimulus or the standard stimulus with light and sound added. Of the nine who responded to the standard stimulus, all nine also responded to the light and sound stimulus without the standard stimulus. Four of the six of these patients then continued to respond to treatment by the battery-less machine alone, heightened by verbal encouragement.

Our long term home results and conclusions will be discussed.

Post-Traumatic Syringomyelia Occurring 14 Years After a Mild Spinal Cord Injury.

D. Stringer, J. Girvin, H. J. M. Barnett, Winston-Salem, N.C. and London

The development of syringomyelia as a sequela to a serious spinal cord injury is a well known entity. It has been reported to occur in 1.9% of patients who after their initial injury were left with a significant and permanent neurologic deficit. However, the occurrence of post-traumatic syringomyelia after a mild spinal cord injury has been reported infrequently. We are reporting a case of a 45-year-old woman who sustained a thoracic spine fracture in 1956 and who had virtually complete return of function except for mild spasticity of her right leg. She was able to carry out all her normal activities except for participating in vigorous sports. Fourteen years later she developed signs of thoracic spinal cord dysfunction which progressed to spastic paraplegia. She was operated upon and found to have syringomyelia both above and below the level of her injury.

A review of the literature has been made and a discussion of this unusual syndrome will be presented.

Cerebral Hemodynamics in Migraine

V. C. Hachinski, J. W. Norris, P. W. Cooper, J. G. Edmeads, Toronto

At the time of clinically indicated arteriography regional and hemispheric cerebral blood flow studies (CBF) were carried out by the intracaratid ^{133}Xenon method on 4 patients with migraine and on 1 patient with cluster headache.

Three of the 4 migraine patients developed an attack during the procedure. Two of these showed no significant change in the CBF during the headache. The third patient had a decrease in CBF during the aura phase and a pronounced increase in the CBF during the headache phase. The fourth migraine patient who had recovered 3 days previously from a migrainous aura consisting of expressive aphasia and right face and arm weakness showed focal CBF changes in the left frontal region. In the patient with the cluster headache there was an apparent increase in the CBF during the attack.

The cerebral hemodynamics in the migraine syndrome are variable. They may depend on the individual and on the type, severity and arterial distribution of the attack.

The Role of Histamine in the Cerebral Microvascular Response to Injury

C. Dila, E. Myer, L. Yamamoto, W. Feindel, Montreal

The pathogenesis of the early microvascular changes in response to minimal cerebral trauma has been studied in the pentothal-anaesthetized dog. A "minimal" mechanical trauma is applied to the exposed cerebral cortex, cardiovascular parameters and blood gases being monitored throughout and controlled. The lesion so produced is virtually indistinguishable from the surrounding normal brain but microscopically may show scattered petechiae. Blood flow within the lesion and in the surrounding normal brain is studied by means of serial fluroescein angiography and ^{133}Xenon microregional cerebral blood flow (rCBF) determinations.

Group A. Twelve dogs received no pharmacological agent aside from the anaesthetic agent. In this group a biphasic cerebrovascular response to minimal trauma is shown. In the period up to 20 minutes following trauma there is increase in rCBF within the lesion of 33% ($p < 0.001$), as compared with the control values. By 1 hour flow has returned to approximately normal values. This response is seen in normocapnic, hypocapnic and hypercapnic animals.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

Group B. Four animals were treated with the bradykinin blocking agent protamine sulfate. Here the early hyperemic phase (rCBF increase 28%, $p < 0.001$) was again seen, the biphasic response to trauma being virtually identical with that seen in the untreated animals.

Group C. Five animals were pretreated with the antihistamine diphenhydramine hydrochloride. In this group the early hyperemic phase of the response was abolished, rCBF within the lesion being significantly slower by 60 minutes following trauma.

From the evidence of these studies, it appears that the plasma bradykinin system, the most potent endogenous dilator of peripheral blood vessels, has little or no role in the regulation of cerebral microcirculation following minimal cerebral trauma. On the other hand, the data indicate that histamine is an important agent mediating the early vasodilatory and hyperemic phase of the biphasic response.

Microvascular Alterations and Edema in Focal Cerebral Ischemia

J. R. Little, F. W. L. Kerr, T. M. Sundt, Jr., Montreal

The sequential morphological alterations that occur in brains of squirrel monkeys following middle cerebral artery occlusion were studied using light and electron microscopic techniques. Astrocytic swelling was an early finding having a perivascular distribution initially but later spreading in a centrifugal fashion to involve the entire neuropil. Enlargement of the extracellular space was identified in scattered areas of the gray matter and appeared to precede necrosis. Progressive microcirculatory obstruction was identified using the carbon perfusion technique. The obstruction lay at the capillary level and appeared to be the result of capillary compression by the swollen perivascular glial processes and swelling of the endothelial cells. Intravascular "bleb" formation and thrombosis did not appear to be important factors. Advanced neuronal changes frequently were detected before severe impairment of carbon filling became evident and were more widespread than the identifiable areas of microcirculatory obstruction. The findings suggested that obstruction of the microcirculation was not the main factor in the production of an infarct. The role of lysosomes was also assessed using histochemical techniques. The lysosomes appeared to be relatively insensitive to ischemia and release of lysosomal contents into the cytoplasm and neuropil occurred at a stage when tissue injury and oedema were already advanced. The relationship of the morphological changes to previous metabolic electroencephalographic and blood flow studies in the same model will also be discussed.

Autoregulation of Cerebral Blood Flow During Controlled Hypotension

G. G. Ferguson, London

The effect of hypotension on the cerebral circulation has been studied in anaesthetized baboons. Progressive hypotension has been produced either by graded hemorrhage or by the administration of increasing concentrations of hypotensive drugs (halothane alone, halothane plus sodium nitroprusside, or halothane plus Arfonad) sufficient to reduce mean arterial blood pressure in 10 mm Hg decrements. A further group of animals has been subjected to hemorrhagic hypotension under α-adrenergic blockade, produced by the prior administration of phenoxybenzamine (1.5 mg/kg). Cerebral blood flow was determined at each step reduction by the intracarotid injection of 133 Xenon. Control values between groups showed no significant differences.

During hemorrhagic hypotension, autoregulation was lost at a mean arterial pressure of approximately 65 mg Hg, below which cerebral blood flow became pressure passive. With drug-induced hypotension, autoregulation persisted to significantly lower levels (approximately 35 mm Hg mean pressure). Under α-adrenergic blockade, animals subjected to hemorrhagic hypotension showed a persistence of autoregulation to levels similar to those obtained with drug-induced hypotension.

It is postulated that controlled, drug-induced hypotension in man, such as used in aneurysm surgery, may be tolerated because of a blockade of sympathetic activity by the hypotensive drugs used. As demonstrated in this study, the hypotensive drugs allow persistence of cerebral autoregulation to remarkably low levels of mean blood pressure.

Neurological Deterioration Following Subarachnoid Hemorrhage: Prevention, Diagnosis and Treatment

S. J. Peerless, N. F. Kassell, London

Progressive central nervous system deficit is common in patients with aneurysms and subarachnoid hemorrhage and occurs with distressing frequency both pre and postoperatively. When deterioration occurs a diagnosis of "spasm" is often made and a fatalistic attitude adopted since no specific therapeutic measures are available. However, the cause of deterioration in patients with ruptured aneurysms is most often multifactorial. While vasospasm is usually a contributing if not dominant factor, other more readily treatable factors are usually operant. Reversal of these factors may result in dramatic improvement, even in the presence of severe spasm.

Factors responsible for progressive neurological dysfunction in patients with subarachnoid hemorrhage include, among others: cerebral arterial vasospasm, arterial thromboembolism, iatrogenic partial or complete arterial occlusion, venous thrombosis, aneurysmal swelling or shrinkage, intracranial hematoma, hydrocephalus, brain edema, ischemic encephalopathy secondary to intraoperative hypotension, fluid and electrolyte disorders, steroid psychosis, respiratory, renal or hepatic dysfunction, meningitis, epileptic seizures, and "floppy brain" syndrome.

The nature of most of these factors is such that they combine in a compound manner accelerating both the rate of development and the severity of the neurological deficit. Often the deficit is reversible, if definitive treatment is begun before permanent changes in the brain have occurred. Understanding of the underlying pathophysiology and treatment of each individual factor and meticulous monitoring of appropriate parameters is the foundation for effective prevention and early detection.

This report relates the experiences with neurological deterioration, both pre and postoperatively, in a series of 60 patients with aneurysms and subarachnoid hemorrhage. The individual factors responsible for the progressive deficit and the underlying pathophysiology and management of each will be discussed.

Carotid Cavernous Fistula. Evolution of Treatment As Seen in Forty-One Cases Over Thirty Years

T. P. Morley, Toronto

In 1950 at the annual meeting of the Canadian Neurological Society in Halifax Botterell and Cluff reported ten cases of carotid cavernous fistula from the Toronto General Hospital. The present report describes the evolution of treatment and its results in the succeeding period when a further thirty-one cases have been encountered.

There were thirteen spontaneous fistulae and twenty-eight traumatic. Eleven cases received no surgical treatment. Four of these experienced delayed ocular deterioration from several causes. Another four fistulae closed spontaneously.

Proximal ligation, trapping and muscle embolization all carried a significant incidence of failure to seal the fistula and ocular and cerebral complications. In the last eight consecutive cases a muscle plug was introduced via an intracranial carotid arteriotomy into the intracavernous carotid and cavernous sinus. This has been the single most effective treatment in the series. Following this operation there was no neurological deterioration and no visual deterioration (three were blind preoperatively) and in all cases the fistula has been permanently sealed.

The relative merits of this and other more recent methods (balloon, Parkinson, Peterson) will be discussed.

The danger of any procedure which involves carotid occlusion is not diminished by prolonging the interval between the onset of fistula and operation in the hope of giving time for collaterals to open.

The Hidden Cerebral Aneurysm

R. G. Vanerlinden, Toronto

The source of bleeding in subarachnoid hemorrhage is not shown by four vessel angiography in 10-20% of patients. In these cases, it has been assumed that a small aneurysm had thrombosed and failed to fill with contrast or a minute angioma had been destroyed when it ruptured. The prognosis in these cases is thought to be much better than it is when

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

an aneurysm can be demonstrated for Drake and others have reported the incidence of re-bleeding at only 10%.

We are reporting 6 cases of subarachnoid hemorrhage in which an aneurysm was not revealed on initial angiography but was demonstrated on repeated studies and was confirmed at operation. The aneurysms, in three cases, were over 5 mm. in size and probably were occluded by thrombus that later flushed out and exposed the fundus to the cerebral circulation. In the other three cases tiny aneurysms, initially camouflaged among the parent vessels, appeared when spasm reduced the vessel size; with clearing of spasm, the aneurysms were again hidden. An aneurysm was demonstrated in a seventh patient on admission but after subsequent clinical deterioration, repeat angiography revealed intense spasm with non filling of the aneurysm.

Case histories will be presented and x-rays shown to illustrate these three mechanisms whereby ruptured aneurysms may be hidden at the time of first angiography. They mark the importance of treating all patients with subarachnoid hemorrhage and negative angiography as if they were harbouring aneurysms. The cases also emphasize the necessity of repeating the arteriograms if supervening spasms is suspected, rebleeding occurs or prior to mobilizing the patient.

The Value of Computerized Axial Tomography in the Management of Acute Head Injury

B. N. French, A. Dublin, Sacramento

In the 9 month period between May 1, 1975 and January 1976, 203 head-injured patients had computerized axial tomography (CT Scan) to assess the degree of intracranial pathology. Positive scans were present in 98 patients (48%). Abnormalities demonstrated in these 98 patients consisted of 5 epidural hematomas, 42 subdural hematomas, 23 intracerebral hematomas, 4 intraventricular hematomas, 47 instances of contusion, edema, or mass effect, and 28 instances of enlarging ventricles. Forty-two of the 98 patients (43%) had multiple lesions. Two cases had clinically unsuspected posterior fossa hematomas that were successfully evacuated. Nine of 24 patients with the clinical diagnosis of "brain stem contusion" had normal first scans. Twelve of the other 15 patients had a surgical intracranial lesion. The CT Scan failed to identify 1 isodense subdural hematoma, 2 cases of post-traumatic vascular spasm, and 1 case of carotid cavernous fistula. We will show representative cases to indicate that the CT Scan gives far more information about the traumatized brain than other diagnostic tools. Angiography has been eliminated in the assessment of head injury except for unusual circumstances.

Intra-Neoplastic Injection of Chemotherapeutic Agents For the Treatment of Brain Tumors

C. H. Tator, Toronto

Systemic administration of chemotherapeutic agents for the treatment of brain tumors has not been successful in most instances. A series of experiments on intra-neoplastic injection of chemotherapeutic agents has been performed in mice bearing intracerebral implants of a transplantable mouse glioma. The uptake and distribution of tritiated methotrexate (^{3}H-MTX) were studied by autoradiography and liquid scintillation counting. Therapeutic studies of MTX and nitrosoureas (CCNU and methyl-CCNU) were then performed on groups of brain tumor bearing mice. MTX was used with and without systemic leucovorin rescue. All chemotherapeutic agents were administered intracerebrally via a specially constructed stereotactic frame, and the results were compared with groups of animals which had received intraperitoneal injections of the same agents.

It was found that MTX was completely ineffective as a chemotherapeutic agent for this transplantable mouse glioma. Intra-neoplastic injection of MTX with or without systemic leucovorin was of no benefit. In contrast, CCNU and methyl-CCNU were highly effective chemotherapeutic agents, and in many experiments intra-neoplastic injection produced a greater increase in the median life-span and a larger number of long term survivors than did intraperitoneal injection. Further studies are underway to determine the possible clinical usefulness of direct intra-neoplastic injection.

Giant Extradural Sacral Cysts: A New Surgical Treatment

B. Hunt, B. Purves, P. Yue, North Vancouver

Congential extraduarl sacral cysts can arise directly from the thecal sac or by attachments with the dural nerve root sleeves.

The extra dural cyst of the patient presented had an unusual dual origin arising symmetrically from the distal portions of both second sacral nerve root dural sleeves.

Operative observations confirmed the hydrostatic hypothesis of the cyst's growth and production of clinical neurological symptoms and signs.

The unorthodox but highly successful surgical treatment was necessitated by the large size of the cyst as well as the mode of origin of the cyst and its contents.

The pre- and post- operative course of the patient is described including the remarkable cystometrogram changes.

SYMPOSIUM: THE CEREBRAL BLOOD VESSEL

Chairman: F. A. Durity

Introduction.
F. A. Durity, Vancouver

Blood — Brain Barrier and Edema.
H. M. Pappius, Montreal.

Thrombogenesis and Hypercoagulability in Relation to Stroke.
H. J. M. Barnett, London

Cerebral Circulation Following Total Cerebral Ischemia.
W. A. Tweed, Winnipeg.

Changes in the Cerebral Blood Vessel Wall Following Subarachnoid Hemorrhage and Stroke.
S. J. Peerless, London.

The Cerebral Vessel Wall in Hypertension.
H. Dinsdale, D. Robertson, Kingston.

Dynamic Aspects of Cortical Vessel Behaviour Under Pathological States.
L. Yamamoto, W. Feindel, Montreal.

Herbert Jasper Award of C.S.E.E.C.N.

Short Term Sequential Memory Tasks and Patterns of Spelling Errors in Underachieving Children

H. Darwish, C. Netley, A. Rebhen, Toronto

The underachieving child is often subjected to batteries of tests. Deficits on these tests are often cited as the cause for failure to read and spell. In the adult with various cerebral lesions, studies have correlated specific spelling error patterns with location of lesions. In the developmental approach these patterns have been regarded as reflecting individual specific strategies related to the existing handicap. One approach to remediation attempts to define the strategy used by the child in order to gain insight into programming the individual specific corrections needed.

We defined forty underachieving children by using a discrepancy of more than 10 points between their W.I.S.C. and W.R.A.T. scores, an I.Q. greater than 85, and the absence of apparent hyperactivity or significant emotional handicap. The Boder dyslexia screening was used to define a visual or a phonic strategy in reading and spelling. Cumulative short term sequential learning trials were performed. The verbal digit suprascan as well as visuospatial block display pointing tasks were used. Serial position curves, types of errors made, response to two rates of presentation and number of trials to correct learning were noted. An attempt will be made to correlate these with error patterns in these childrens' spelling. The significance of these results in terms of remedial programmes will be discussed.

Isolation and Chemical Nature of Curvilinear Bodies From Batten's Disease

L. S. Wolfe, R. R. Baker, N. M. K. Ng Ying Kin, S. Carpenter, Montreal

It is generally thought that the autofluorescent lipopigments accumulating in neurones and other tissues in the late infantile form of

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

Batten's disease represent lipid peroxides. Our recent studies have failed to reveal a deficiency of leukocyte p-phenylene diamine peroxidase in either the late infantile or juvenile forms of this disease complex. We report here new chemical findings on curvilinear bodies isolated and highly purified from the cerebral cortex shortly after death from a 7-year old child diagnosed pathologically as suffering from the late infantile form of Batten's disease. Utilizing discontinuous density gradient centrifugation, the autofluroescent granules were localized at the 1.4-1.6 M sucrose interface (density 1.2). After pronase digestion of this fraction, EM revealed a highly pure fraction of curvilinear profiles. The fluorescent material was insoluble in common organic solvents. All lipid phosphorus was removable by chloroform-methanol extraction. Chemical studies were performed on the highly autofluorescent lipid-free material. Infra-red spectroscopy revealed the presence of amide, NH, OH, C=C, CH, Ch_2 and CH_3 groups consistent with the elemental analysis. Fatty acid, carboxylate, phosphate, sulfate, aromatic, pyrimidine or ether groups were not found as also indicated by direct probe-pyrolysis mass spectrometry. Strong base treatment cleaved the material and yielded fluorescent products readily extractable by chloroform with infra-red spectra showing intense NH absorptions with retention of the C=C, CH, CH_2 and CH_3 groups. Strong acid destroyed the fluorescence. The purified autofluorescent component of curvilinear bodies is not lipid in nature, unlikely directly derived from lipid peroxidation and has properties like unsaturated polyamide polymers.

Acute Convergent Strabismus in Children

R. C. Polomeno, T. H. Kirkham, Montreal

The sudden onset of a convergent strabismus with horizontal diplopia in a child is usually caused by a sixth nerve paresis or by divergence paralysis and, rarely, by convergence spasm. The more frequent type of convergent strabismus which is caused either by an accommodative factor or is precipitated by physical or emotional stress is characteristically intermittent and horizontal diplopia is rare.

Five patients with accommodative and non-accommodative convergent strabismus and horizontal diplopia whose onset was sudden but who did not have any clinical signs of a neurological disorder are described. There was no limitation of the lateral rectus and saccadic velocities were normal on electroculography. The horizontal diplopia and convergent strabismus were eliminated with glasses in two cases and surgically in three cases.

The physiology of single binocular vision as well as the causes and management of accommodative and nonaccommodative convergent strabismus is discussed.

Periodic Breathing, Unresponsiveness and Automatisms

J. Lafleur, J. Reiher, Sherbrooke

Periodic breathing is a respiratory pattern in which hyperpnea regularly alternaties with apnea. Patients with Cheyne-Stokes respiration (CSR), the most common type of periodic breathing, generally exhibit clinical signs of bilateral lesions deep in the cerebral hemispheres, or of metabolic impairement of presumably similar cerebral regions.

Over the past five years, three children — with a normal neurological examination at rest — exhibited with hyperventilation periodic attacks characterized by stereotyped respiratory, electroencephalographical and clinical manifestations. These sequential attacks, we have termed pseudo-absences.

From an analysis of available polygraphic recordings, it can be stated that:

1. The periodic hyperpnea of pseudo-absences consists of regular excursions of respiratory movements, without the crescendo-decrescendo sequence typical of CSR.
2. The EEG concomitants of pseudo-absences resemble but a mere accentuation of the EEG slow waves induced by hyperventilation, or by apnea following hyperventilation; they are of higher voltage however, more widely distributed and distinctly slower.
3. Concomitantly with the appearance of the slow waves, apnea, unresponsiveness, and automatisms are observed. Thereafter, as the electroencephalogram reverts gradually to a more normal appearance, the child resumes spontaneously to hyperventilate again. The same sequence of respiratory, EEG and clinical events repeats itself — over and over — the second and subsequent pseudo-absences being precipitated by shorter bouts of hyperventilation than the initial one.

Although the number of patients is small, the following conclusions would appear justified:

1. Although alterations of consciousness induced by hyperventilation have been mentioned occasionally, a clear definition of attacks which we have singled out under the name of pseudo-absences has not been reported heretofore.
2. Pseudo-absences are probably more common than suspected.
3. Pseudo-absences must be differentiated from typical and atypical absences. Children with pseudo-absences should not be catalogued as epileptics.
4. The precise mechanism of sequential pseudo-absences probably rests on cyclic modifications of PO_2, PCO_2, and cerebral blood flow, but needs to be investigated further.

Adrenoleukodystrophy

G. G. Hinton, G. B. Young, London

Adrenoleukodystrophy is a sex-linked recessive disorder consisting of primary adrenal failure and progressive neurological symptoms and signs, secondary to C.N.S. demyelination.

Two boys recently presented with a clinical picture, compatible with adrenoleukodystrophy. One boy, age fourteen, had a two year history of behaviour problem and poor school performance, with a decline of full scale IQ from 92 to 71, over two years. He presented clinically with acute adrenal failure after a viral infection. Neurological examination was normal apart from mild intellectual impairment. The second boy, age eleven, had a two year history of behaviour problem and poor school performance with a six month history of spastic-ataxic gait and generalized seizures. Neurological findings included bilateral optic atrophy, bilateral spasticity and left-sided ataxia.

Both patients had melanoderma with gingival pigmentation and abnormally low serum cortisol levels. ACTH stimulation tests showed no response, confirming the diagnosis of primary adrenal insufficiency.

EMG and Nerve Conduction Studies in each case were normal. Bilateral sural nerve biopsies showed a normal density and normal spectrum of size of myelinated axons, and a normal population of unmyelinated axons. On electron microscopic study, Schwann cells did not show the characteristic lamellar bodies described in the literature.

Clinical and Pathological Review of Eight Cases of Globoid Leukodystrophy

H. Darwish, L. Becker, D. Armstrong, C. Fitz, J. A. Lowden, Toronto

Globoid leukodystrophy is usually described to start acutely after age 4-6 months. Various reports have emphasized the presence of normal developmental grains before the onset of the disease. We reviewed eight cases seen at The Hospital for Sick Children with confirmation of the diagnosis by histology or enzyme studies. Three patients were abnormal from birth or in the first six weeks. Another three came to medical attention for relevant symptoms before age four months. Only two patients had normal development followed by regression sometime after age six months. The majority of our patients were male. In addition to the usually reported symdrome and neurologic signs, half our patients had flat or asymmetric facies early in the course.

The presence of subdural effusion was suspected on neuroradiologic procedures in two cases, thereby delaying the appropriate diagnosis. In two more patients, subdural hematoma were found at autopsy. This high incidence could be related to the irritability and consequent inapparent abuse. Cerebral atrophy or an irritative chemical etiology will be discussed as other possible causes.

Folate Responsiveness Homocystinuria

J. A. R. Tibbles, Halifax

An eleven year old boy initially presented with features suggesting minimal brain dysfunction. Over the next nine months he developed seizures, dementia, long tract signs, girdle weakness and peripheral

neuropathy. Investigations revealed a moderately elevated plasma homocysteine with a normal methionine level, homocystinuria, an unidentified urinary mucopolysaccharide and a low serum folate. Methionine load studies indicated normal synthesis of cystathionine (Dr. S. H. Mudd). Fibroblast cultures demonstrated a deficiency of methylene tetrahydrofolate reductase to 18 and 25% of normal (Dr. R. W. Erbe). Treatment by folic acid has reversed the dementia and neuropathy and greatly improved the long tract signs.

While only the fourth described case of this condition, it has important general applications. (1) The neurologic picture seen may be a model of nervous system involvement due to folate depletion. (2) Schizophrenia like behavior as described in one other case, with its more widespread metabolic implications, does not appear typical. (3) Concurrently administered drugs may precipitate the condition as found in two of the four recorded cases. (4) Folate dependent homocystinuria must be included among the causes of combined central and peripheral nervous system disease and apparent degenerative disease in the second decade.

Familial Agenesis of the Corpus Callosum With Sensori-motor Neuronopathy: A New Autosomal Recessive Syndrome Originating in Charlevoix County

E. Andermann, F. Andermann, S. Carpenter, G. Karpati, A. Eisen, D. Melancon, J. Bergeron, Montreal

In recent years, we have recognized a syndrome in patients originating from Charlevoix County who present with psychomotor retardation and slowly progressive flaccid quadriparesis, most marked in the lower extremities. Other features of the symdrome include brachycephaly, bilateral ptosis, strabismus, asymmetrical facies, hypoplastic maxilla, large angle of the mandible, high-arched palate, kyphoscoliosis, pigeon-chest deformities, and various digital anomalies.

These patients have been proven to have complete agenesis of the corpus callosum, at times associated with heterotopias. The electrophysiological findings indicated evidence for anterior horn cell disease. There was also a total absence of sensory evoked potentials, which was considered to reflect involvement of the dorsal root ganglion. Gastroonemius muscle biopsies showed denervation atrophy. By phase microscopy on semithin epon sections, sural nerves showed absence of almost all large myelinated fibers with preservation of small myelinated fibers and unmyelinated fibers. Wallerian degeneration was absent.

This combination of central and peripheral abnormalities is inherited as an autosomal recessive disorder, with remarkable intra and interfamilial similarities. The syndrome has been ascertained in 42 patients from 21 sibships, and 18 patients in 10 sibships have been examined to date. All the families originated from settlements between Baie St. Paul and La Malbaie, and most of the sibships have now been traced to a common ancestral couple who married in Quebec City in 1657.

Because of its high incidence, this syndrome represents a major public health problem in Charlevoix County, as well as in the areas to which these families have migrated. Since carrier detection is not yet feasible by biochemical or morphological means, genetic counselling with avoidance of consanguineous marriages is the only effective form of prevention.

Although there have been a few isolated case reports of familial agenesis of the corpus callosum in the world literature, the above syndrom has not been previously described to our knowledge.

The Rigid Spine Syndrome (Dubowitz)

H. G. Dunn, Vancouver

Dubowitz (1970, 1973) described a syndrome consisting of limitation of spinal flexion associated with widespread but relatively non-progressive myopathy. His 4 patients were all males who mostly had little weakness but were liable to develop spinal deformity. They had a moderately elevated level of serum creatine phosphokinase (SCPK) and a "myopathic" EMG pattern. Muscle biopsies showed myopathic changes and fibrosis without evidence of polymyositis.

Three cases of this syndrome have been encountered in two Canadian families. The first was a boy who suffered a fracture-dislocation of the cervical spine at 8 years. Two years later he tended to walk on his toes and had increasing difficulty in stooping and also in extending his elbows. On examination at 12 years he was of borderline small stature with excessive lumbar lordosis and had hardly any spinal flexion, restricted extension of elbows, heelcord contractures, and weakness of several muscle groups. He also had "minimal brain dysfunction" with hyperkinesis. SCPK level ranged from 503 to 643 I.U. Glucose tolerance was normal. ECG suggested left ventricular hypertrophy. EMG showed mild "myopathic" changes in tibalis anterior and deltoid and more severe "myopathic" changes with fibrillation potentials in paraspinal muscles. Nerve conduction was normal. Biopsy of deltoid demonstrated a myopathy.

This boy's mother was quite well, but had an SCPK level of 201 I.U. The maternal grandmother was first diagnosed as having muscular dystrophy before the age of 20 years. She developed a rigid spine and required heelcord lengthening. By the age of 50 she was wheelchair-bound and grossly weak, with marked limitation of spinal flexion. At 53, she had normal nerve conduction and severe "myopathic" changes on EMG, with occasional high-frequency discharges in deltoid and paraspinal muscles. Autopsy in the same year showed muscular dystrophy with more severe involvement of proximal muscles, myocardial fibrosis and relative sparing of sternomastoids.

The third case was a girl who had first been noted to have spinal stiffness at 8 years. She gradually developed a fixed lordosis with tight paraspinal muscles, also some restriction of hip movements, heelcord contractures and pes cavus. On examination at 14 years she was also significantly small for her age and had absent tendon reflexes and mildly impaired vibration and touch sensation in fingertips and toes. Glucose tolerance curve was "diabetic". Nerve conduction studies demonstrated a sensori-motor polyneuropathy with conduction block in the lateral popliteal nerve. EMG showed severe "myopathic" changes, with high-frequency discharges, noted best after needle movement. SCPK level ranged from 180 to 284 I.U. Biopsy of peronei showed severe "neuromyopathy" with fatty and fibrous changes, while sections of sural nerve demonstrated some loss of myelinated fibres.

The pathogenesis and possible treatment of the syndrome will be discussed.

Acute Multiple Sclerosis Simulating Brain Tumour

J. Murphy, F. Dominique, Winnipeg

An 18 year old female first noted weakness of the left lower limb on 13th July 1975. On admission to hospital on 18th July, this had progressed to complete left hemiparesis. Neurological deterioration continued and by 12th August the patient was moribund.

Brain Scan showed multiple discrete filling defects in the hemispheres. Angiography was negative. Viral Studies were negative. Brain biopsy was subject to light and electron microscopy, and suggested acute Multiple Sclerosis (slides available to show).

Therapy with A.C.T.H. produced dramatic improvement with clearing of Brain Scan. Subsequently the patient suffered a relapse with optic neuritis and appendicular ataxia following minor trauma.

Global Paresis of Saccadic Eye Movements in Spinocerebellar Degeneration

J. A. Sharpe, Toronto

Quantitative analysis of ocular motor function by infrared reflection oculography showed paresis of all fast eye movements in a syndrome of spinocerebellar ataxia similar to that described by Madia and Swami (Brain 94: 359, 1971). Macular degeneration and dementia were variable manifestations of this disorder in a black man and his son and in a white girl of Greek descent.

Fast eye movements include voluntary and reflex saccades and nystagmus fast phases. In those patients saccadic paresis in all directions was manifested by reduced peak velocities and multiple-step hypometric saccades. Optokinetic and caloric nystagmus fast phases were decreased and slowed. The pursuit subsystem was preserved with attentive smooth tracking at 30°/second. Refixation was facilitated by vestibular slow eye movements activated by hand thrusts. Saccades require a pulse step of ocular motor neuron innervation. Main sequence curves that define the relationship between peak velocity and amplitude were utilized to determine abnormality of the pulse that generates fast eye movements. Full gaze excursions and normal position holding in these patients signified sparing of the neural integrators presumed to mediate the required stop of motor neuron innervation. Bilateral in-

volvement of the fronto-tegmental pathways subserving saccades with consequent defective recruitment of brain stem saccadic pulse generators is considered responsible for this global saccadic paresis.

Computerized Axial Tomography As a Diagnostic Tool in Wilson's Disease

Z. Grahovac, D. A. Guzman, D. Howse, R. F. Nelson

Computerized axial tomography has proven of immeasurable value in delineating structural lesions of the brain and is particularly useful in the diagnosis of tumor, hematoma and infarction. Less attention has been given to its diagnostic potential in metabolic disease.

Hepatolenticular degeneration while primarily a disorder of copper transport mechanisms does produce, in advanced cases, structural brain disease which was well described in S. A. Kinnear Wilson's original article in 1912. We have carried out computerized axial tomography in 7 proven cases of Wilson's disease. In 2 cases we demonstrated typical atrophy of the putamen and, to a lesser extent, the globus pallidus in addition to cortical atrophy. A good clinical correlation is noted between central nervous system involvement and x-ray findings in that the cases with positive x-rays ahd prominent neurological signs and symptoms while those with normal x-rays had primarily hepatic involvement. In 2 cases with negative x-rays central nervous system symptoms virtually disappeared with treatment. This suggests that computerized axial tomography may also be of considerable prognostic value with the implication that symptoms may be reversible as long as structural change is not evident. Since electroencephalography is usually normal unless there is encephalopathy due to liver disease and pneumoencephalography only shows nonspecific diffuse cerebral atrophy the computerized axial tomography is virtually the only way of demonstrating the degree of basal ganglion destruction. This is believed to be the first report of computerized axial tomography findings in Wilson's disease.

Long-Loop Motor Reflexes in Spastic Hemiplegia and in Patients With Cortical Sensory Loss

R. G. Lee, W. G. Tatton, F. E. LeBlanc, R. H. A. Haslam, Calgary

In normal humans sudden displacements of the wrist joint result in a series of discrete EMG responses from the forearm muscles resisting the change. These EMG components occur with reproducible onset latencies of 28-32 msec. (M1 response), 55-60 msec. (M2 response) and 85-90 msec. (M3 response). The M1 response is appropriately timed to be a monosynaptic spinal reflex. Evidence to date suggests that the M2 and M3 components result from feedback in supraspinal "long-loop" reflex pathways which possibly include somatosensory and motor cortex.

In a previous study (1) it was reported that the M2 and M3 components were markedly accentuated in Parkinsonian patients, and it was suggested that increased feedback over long-loop reflex pathways might be one of the mechanisms responsible for Parkinsonian rigidity.

These techniques have now been used to investigate two other groups of patients — spastic hemiplegia and patients with focal lesions involving the post-central sensory cortex. In the hemiplegics the M1 component was markedly enlarged, a finding consistent with the increased tendon reflexes observed clinically. Changes in the M2 and M3 components were less consistent — in one case these responses were moderately accentuated whereas in two other cases they did not differ greatly from responses recorded from the clinically normal arm.

In one patient with a solitary metastatic tumor in the left parietal lobe there was selective attenuation of the M2 response in the contralateral upper extremity. The second patient with a cortical lesion was a young boy with intractable focal epilepsy who underwent surgical resection of the arm area of somatosensory cortex. Long-loop reflexes were recorded before and following surgery, and in the post-operative study the M2 component was missing in the response from the opposite arm.

These results provide further support for the hypothesis that the M2 component is mediated by a transcortical pathway. The relative ease with which these studies can be performed suggests that they could be developed into a useful clinical tool for investigating patients with motor system disorders.

Neurofibrillary Tangles and the Reversible Dementia of Normal-Pressure Hydrocephalus

M. J. Ball, London

The clinical syndrome of "low-pressure" hydrocephalus has yet to be shown to have a constant pathological basis. Those few patients with NPH studies at autopsy (Heinz et al., 1970; De Land et al., 1972; Sypert et al., Sohn et al., Coblentz et al., 1973; Lorenzo et al., Earnest et al., 1974) have had hypertensive encephalopathy, leptomeningeal fibrosis, or Alzheimer's disease. We report the neuropathological findings on four patients shunted for this condition. The degree of neurofibrillary tangle formation in serial histological sections of both hippocampi of each brain was determined by quantitative morphometry, using a semiautomated scanning microscope (Ball, Can. J. Neurol. Sci., 1975). The number of tangle-bearing neurons per cu. mm. of hippocampal cortex correlated highly with (a) the length of clinical dementia before shunting; and (b) the total duration of dementia before death ($r_s = 0.99$, $p < 0.01$). The "tangle index" of the one patient who responded to shunting falls within the range of age-matched normal brains (from 19 mentally intact subjects). The density of tangles in the other three, who showed no improvement, is up to 20 times greater than in controls. The data suggest that the degree of neurofibrillary degeneration in mesial temporal cortex may account for the severity of dementia in NPH, and might explain the irreversible nature of the process in those patients with functioning shunts whose cellular changes have already become too pronounced.

Familial Dominent Fatal Parkinsonism

P. J. A. Bratty, Vancouver

In 1973 the author first reported a familial fatal dominant variety of Parkinsonism, characterized clinically by lethargy, abnormal respirations and gagging and in the single case examined biochemically by a low taurine in brain serum and spinal fluid. Further studies have been done on a fourth sibling with this disease, including a double blind trial of taurine and a trial of L-dopa with carbidopa. The results of these trials and chemical study of C.S.F. and serum on and off medication will be reported.

The Complications of Methysergide (Sansert) Therapy

T. J. Murray, J. W. Stewart, Halifax

Despite recent advances in the prophylactic therapy of migraine, methysergide continues to be used widely. Two recent serious complications with this drug emphasize the necessity of continuing caution when it is used, even with interrupted therapy.

A 48-year old schoolteacher took 6 to 8 mgms. of methysergide for over three years for intractable migraine. She presented with retroperitoneal fibrosis, hydronephrosis, bilateral renal artery stenosis, renal infarction, uremia, hypertension, pulmonary hypertension, mitral and tricuspid regurgitation and cardiac failure. When the drug was discontinued renal and cardiac function improved rapidly and has continued to improve over the next three years although cardiac murmurs are still present. At the present time she requires antihypertensive medication for continuing hypertension and renal defects are still present although renal function is now normal. A review of her investigations including cardiac and renal angiography will show the complications and progressive improvement following discontinuing the drug. This case represents the devastating effects that may occur on long term uninterrupted therapy.

A 47-year-old woman with familial myoclonus was given a trial of methysergide. After five months on therapy her myoclonus improved dramatically but she was admitted to hospital with pulseless, cold, cyanotic and painful arms and legs. These problems cleared within a week after discontinuing the drug. This case demonstrates that serious complications can occur with the relatively short term use of this medication as well.

Prevention and Suppression of Experimental Allergic Encephalitis by the Spinal Cord Protein, SCP

C. F. C. MacPherson, B. Yeung, London

Pretreatment of guinea pigs with three weekly injections of 100 μg of bovine SCP (J. Immunol. 116, 227, 1976) prevented them from develop-

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

ing clinical signs of experimental allergic encephalitis (EAE) when they were sensitized with excessive doses of purified bovine myelin basic protein (MyBP). The present report will describe experiments which showed that preventive pretreatment was effective when completed 12 to 28 days before sensitization. In contrast, 50% of animals were protected when pretreated with six weekly injections of BSCP and none were protected when pretreated with 10 weekly injections of BSCP.

EAE was suppressed when 500 μg of BSCP was injected in incomplete Freund's adjuvant on Days 4, 8, 12 and 16 after sensitization with purified MyBP. The suppressive property of BSCP purified from spinal cord and BSCP isolated from spinal roots will be contrasted and compared with the suppressive activities of bovine MyBP and bovine gamma globulin.

Evidence will be presented to indicate that the mechanism of the anti-encephalitogenic activity of SCP does not depend on anti-SCP antibody or on any immunochemical cross-reactivity between SCP and MyBP that can be detected by sensitive radioimmunological assays using either anti-SCP antisera or anti-MyBP antisera.

Angiographic Evidence for Basilar Artery Spasm

J. D. Brown, R. K. Coates, London

A woman, aged 46, with a remote history of common migraine was investigated because of 10 to 15 second episodes of opsoclonic eye movements, nausea, vertigo, tinnitus, left arm paresthesias and vertex pressure sensations which were occasionally followed by transient syncope. Examination revealed no neurological or cardiovascular abnormalities and she was not hypertensive. Selective vertebral and carotid angiography was performed with a red Kifa catheter passed through the right femoral artery. Studies of both carotid and left vertebral arteries were normal and without attendant spasm. Injection of 5-6 ccs. of dye into the right vertebral artery on two separate occasions caused the patient to complain of the above symptoms and then to be unresponsive for 10-15 seconds. Films taken at those times showed initial filling of the right vertebral artery and streaming of dye in the basilar artery followed by a hold up of the dye beyond the origin of the basilar artery. Spasm of the basilar artery as a possible mechanism to explain the prodrome of basilar migraine or for vertebrobasilar transient ischemic attacks is suggested by these findings.

Transfemoral Catheter Epidural Venography in Lumbar Disc Disease

M. A. Mangan, J. R. Bradshaw, N. A. Russell, St. John's

Despite long experience with myelography many neurological centres continue to encounter cases of suspected disc disease in which myelographic accuracy falls short of expectations. Spinal venography has been advocated for more accurate delineation of changes in the lumbar-sacral canal due to disc protrusions. The original intra-osseous technique fell into disrepute, mainly becuase of unpredictable filling of the internal vertebral venous plexus which rendered interpretation difficult. Recent reports of greatly improved venous filling facilitating highly accurate diagnosis using catheter techniques instead of the intra-osseous route has restored interest in this diagnostic procedure.

A five-year experience with the technique in fifty patients with normal or equivocal myelographic findings is reported. A description of the technique and the relevant anatomy is included, together with illustrative cases in which surgical findings are available.

Experience With ICP Monitoring

H. Schutz, F. A. Taylor, Toronto

The ventricular fluid pressure (VFP) was recorded in 146 neurosurgical patients over a three year period. The VFP was recorded an average of 4.4 days per patient with an infection rate of 1.4%. The principal patient groups studied were head injuries (41), brain tumours (25), intracranial haemorrhages (20), hydrocephalus (53) and miscellaneous (7).

Knowledge of the VFP affected daily management decisions in the following situations:

(a) elevation of VFP in clinical circumstances not suggestive of raised pressure.

b) normalcy of VFP in clinical circumstances suggestive of raised pressure.

c) provision of the only reliable sign during periods of controlled ventilation.

d) efficacy of dehydrating agents, ventilation and steriods in lowering VFP.

e) effectiveness of external and internal ventricular drainage in obstructive hydrocephalus.

f) assessment of compliance and volume pressure relationships.

Our experience allows us to assert that VFP monitoring has become an indispensable adjunct in the management of many neurosurgical patients.

Surgical Management of Craniopharyngioma in Childhood

H. J. Hoffman, Toronto

The treatment of craniopharyngioma in children has been a controversial topic ever since Matson stated that these tumours could and should be totally removed. Other surgeons have been far less successful in treating craniopharyngiomas and the statement frequently made in the literature is that complete excision of a craniopharyngioma is difficult and frequently impossible and carries an extremely high operative mortality. For this reason, the treatment of these lesions by simple cyst aspiration and radiotherapy has come into vogue, with this treatment finding strong support amongst radiotherapists and timid neurosurgeons.

During the years 1950-1975, 48 children with craniopharyngioma have ahd their primary surgical care at the Hospital for Sick Children in Toronto. Sixteen of these patients were treated initially by cyst aspiration. All of these patients required repeat craniotomies. Eight patients developed hydrocephalus with 5 requiring multiple shunt revisions. Ten patients received radiotherapy and 5 of these have died. In addition, 5 of the non-radiated group treated by aspiration have gone on to die, leaving only 6 survivors, 5 of whom have received radiotherapy.

Fifteen patients were treated by subtotal resection of their tumour. Two of these have died of tumour recurrence. Seven patients have never had recurrence of symptoms and have not been radiated. Six patients who did develop recurrent problems were radiated and all of these are alive.

Seventeen patients had a total excision of their tumour. None of these have required a repeat intracranial operation for tumour or hydrocephalus. None have been radiated. There have only been 2 deaths in this group, 1 occurring immediately postoperatively in 1955 prior to use of the operating room microscope: the other death occurred 2 years postoperatively in a well patient who developed hypoglycemia while having a metipyrone test carried out in another institution.

On the basis of an analysis of this series of cases, we have concluded that tumour adherence preventing total excision of a craniopharyngioma occurs between tumour and internal carotid artery in about 10-15% of cases. This would allow for total excision of a craniopharyngioma in upwards of 80% of patients with this lesion. The size of the tumour does not appear to be a factor in allowing for total excision nor does the age of the patient. The only deterring factor is previous manipulation of the tumour either surgically or by radiation which produces dense adhesions and makes curative surgery impossible. Radiotherapy does appear to slow down recurrence of cyst fluid but is certainly not curative and we feel should only be used if one is unable to totally remove the tumour and recurrent symptoms occur.

Modern neuroanesthesia and magnification allow for a total removal of these tumours without the risk that was incurred by surgeons in the past. Our series of craniopharyngiomas appears to reinforce Matson's thesis that these tumours can and should be totally excised.

Myelopathy Due to Cervical Spinal Stenosis

J. Stratford, Montreal

The term cervical spinal stenosis refers to those patients who have a congenitally small cervical canal, quite apart from any narrowing due to spondylotic ridges. The sagittal or A.P. diameter of the canal is the crucial measurement in these patients. The normal range is considerable with the average being approximately 17 mms. In spinal stenosis, a minimum A.P. diameter may be 12 mms. or less. Due to x-ray magnification, 12 mms. is equivalent to an actual measurement of 10.5 mms., very close to the 10 mms. A.P. dimension of the average cervical spinal cord.

In the series of ten patients to be reported, measurements were taken of the spinal canal at C4, C5 and C6 as seen on lateral x-rays. These measurements did not take into account any additional narrowing due to cervical spondylosis. Each of the ten patients had at least one sagittal or A.P. diameter of 12 mms. or less. Most patients had some additional narrowing due to spondylotic ridging and half had a definite history of trauma, either acute or chronic. All ten patients had myelopathy of varying degrees. Operation with multiple level laminectomy was done on nine of the ten patients and subsequently there was gradual improvement in the myelopathy.

The triad of congenital spinal stenosis, cervical spondylosis and trauma resulting in myelopathy is a significant entity.

Anterior Approach for Removal of a Cervical Intra-Dural Tumor: Case Report and Technical Note

J. C. Giroux, C. Nohra, Montreal

Anteriorly situated cervical intradural and extramedullary tumors are infrequent lesions.

Removal of these anterior lesions may be accompanied by a high morbidity.

We wish to report the case of a 61 year old patient with a history of progressive paraparesis. A myelogram demonstrated an anteriorly situated intra-dural, extra-medullary tumor at the level of C5.

A 2.5 cm. meningioma was removed through the anterior approach without any particular incident.

The patient recovered uneventfully and very quickly from his paraparesis.

We wish to discuss the surgical aspect and review of the literature on cervical tumors. To our knowledge there is no report yet in the literature on the use of this technique for cervical intra-dural tumors.

Temporal Bone Resection for Cancer of the Middle and External Ear

N. C. Hill, D. Fewer, F. Burrows, Winnipeg

In carefully selected cases, en block subtotal resection of the temporal bone is a useful procedure for the treatment of cancers of the middle or external ear which have invaded the temporal bone, particularly when these tumors do not respond to radiotherapy, or where painful radionecrosis of the temporal bone is to be avoided. A team approach is necessary, involving a neurosurgeon, a general surgeon and an otolaryngologist. If the external ear is involved a plastic surgeon should also participate.

The critical neurosurgical anatomy in this region involves an extradural posterior fossa approach to the internal auditory meatus, retracting the sigmoid sinus, and the location of the probable site of the internal carotid artery in the temporal bone before this bone is transected. These areas may be relatively unfamiliar to many neurosurgeons.

This paper describes the detailed surgical anatomy of this region, as well as the surgical experience with three successful sub-total en bloc resection of the temporal bone performed for two cases of carcinoma of the middle ear and one of carcinoma of the external ear, all of which involved the mastoid bone. The complications following these procedures are described as well as their management. Recommendations are made for improvement of the surgical technique, and an assessment is made of the results of treatment in this and other series.

Cervical Spinal Injuries

K. W. E. Paine, Saskatoon

Approximately 250 cervical spine injuries with and without spinal cord involvement initially treated at the University Hospital, Saskatoon, have been reviewed, Particular attention was paid to the actual method of management in relation to the bone injury, noting the advantages and disadvantages of each method and from this arises recommendations for the present day handling of such injuries. The paper will contain information regarding the methods used and the rationale of the recommendations made. This abstract summarizes the latter. The basic principles should be realignment of the cervical spine and appropriate methods of fixation so as to preserve spinal cord and nerve root function, to use such methods as will enable early rehabilitation and thus limit the patient's hospital stay and to allow recovery of maximum cervical spinal movement. These principles will apply whether the patient is quadriplegic or has a partial cord or root lesion or no neurological abnormality since even in the quadriplegic patient respiratory and urinary complications will be less the earlier mobilization can be obtained.

Realignment of the cervical spine can usually be obtained satisfactorily by skull traction.

Jefferson fractures are best treated by immobilization in a simple collar.

Fractures of the adontoid and the hangman's fracture should be treated by realignment with skull traction if necessary and then the application of halo traction. In young children wiring of the laminal arches between C1 and C2 is preferrable. Only if the halo traction fails to maintain satisfactory position should posterior wiring between C1 and C2 be considered in the adult but this will be followed by reduced cervical spinal movement.

Unilateral dislocations are best treated by open reduction and immobilization by wiring the two laminae together.

Rotational fracture dislocations will be reduced easily by skull traction and should be maintained by an anterior fusion of the Smith-Robinson type.

Bilateral fracture dislocations are realigned with skull traction and then fixed with an anterior fusion of the Smith-Robinson type.

Bilateral dislocations without fractures, if not reduced with moderate skull traction, should be reduced by open operation and the laminae wired.

In ankylosing spondylitis management has to be tailored to the individual.

In the explosion type fractures of the body there may sometime be reason for an anterior decompression followed by anterior fusion.

Simple compression fractures or fractures of the spinous process or the lateral masses without displacement can be managed in a simple collar, but should be followed frequently to detect increasing displacement or abnormal mobility.

Appropriate myelography and laminectomy should be performed when indicated but in general decompressive laminectomy is not helpful in the patient with a complete spinal lesion.

The Minerva cast probably should not be used nowadays as it does not give satisfactory stabilization and certainly can never be used in a patient who has sensory impairment over the trunk.

The halo traction should be used only for upper cervical injuries since it does not immobilize the lower cervical injury satisfactorily and, furthermore, can never be used in those patients with sensory loss on the trunk.

Posterior fusion with bone is unsatisfactory since it requires prolonged immobilization with continued skull traction until the graft takes and, therefore, delays rehabilitation.

Posterior wiring will never be satisfactory in fracture dislocations except of the adontoid and hangman's type since the stresses along the wire are not along the length of the wire. It can only be used in those instances where the holding of two laminal arches together will satisfactorily prevent further displacement.

Acrylic should never be used for internal fixation as it does not give satisfactory fixation and, furthermore, the number of vertebrae included in the fixation means that the end result is a patient with severe restriction of cervical spinal movements.

Pyogenic ("Hot") Psoas Abscess Secondary to Intervertebral Disc Space Infection

N. A. Russell, C. Heughan, St. John's

The term *Psoas Abscess* is traditionally applied to a manifestation of tuberculous osteomyelitis of the thoracic spine, in which a "*cold*" abscess presents in the femoral triangle. With the decline in the incidence of tuberculous disease occasional reports of acute pyogenic psoas abscess have appeared. These have arisen from bowel, from perinephric abscesses and from psoas muscle hematomas.

This report reviews three cases of pyogenic psoas abscess for which the only cause was previous lumbar disc surgery. The surgery had been performed a number of years prior to the presentation of the abscess and two had been complicated by early post-operative wound infection.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

The patients presented with clinical features that lead to an initial impression of disseminated malignancy. These presenting features are reviewed. The management of the cases is discussed. Fistulograms performed after drainage of the abscesses, showed the long sinus tracts running retroperitoneally from the femoral triangle up to the original source of infection in the vertebral bodies. Attention is directed to this diagnostic possibility in patients who present with debilitating illness after having previous lumbar disc surgery.

Detection of Early Hydrocephalus by Echoencephalography in Subarachnoid Hemorrhage

C. Luneau, J. Reiher, M. Heon, Sherbrooke

Hydrocephalus, as an early or late complication of subarachnoid hemorrhage (SAH) has been repeatedly documented. Often times however, its early occurrence is diagnosed only fortuitously after angiography has been performed to outline the source of bleeding.

The purpose of this study is to report on our experience with the detection of early hydrocephalus following SAH based on estimation of the 3rd ventricle size by echoencephalography.

In a series of 144 consecutive patients admitted with SAH, serial echoencephalograms were obtained, the initial one immediately upon admission in most instances.

44 patients were found to have at some time midline echo enlargement well beyond accepted values; in 21 of these, third ventricle distension was detected within the first 10 days following the onset of SAH.

Finally, echoencephalography can be performed at the patient's bedside without the risks and discomfort inherent to angiography or pneumoencephalography. It can be repeated at reasonable costs, much more often and much more rapidly than an EMI scan. Far from precluding more definitive studies, echoencephalography might help to decide more rationally on appropriate timing for these studies.

From an analysis of clinical, echoencephalographic and radiological data in these 21 patients, the following conclusions can be drawn:

1. To early hydrocephalus complicating SAH correspond rather uniform symptoms and signs, to be reckoned with in assessing patients status according to Botterell's grading system.
2. Clinical deterioration or improvement correlated well in most instances with an increase or decrease in size of the third ventricle, as estimated by serial echoencephalograms.
3. Early hydrocephalus represented a transient complication in 2 patients; temporary ventricular drainage or permanent shunting procedures resulted in clinical improvement in 11 of 12 patients.
4. Whence early hydrocephalus has been demonstrated, the earlier the drainage or the shunting, the more dramatic the clinical improvement seems to be.

https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

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https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press

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https://doi.org/10.1017/S0317167100025907 Published online by Cambridge University Press