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Reoperation related to graft complication following anterior cervical fusion

At our department anterior cervical decompression and fusion is usually performed using the Cloward or the Smith Robinson method; for the severely spondylitic cervical spine we occasionally employ the technique of partial multiple vertebrectomy with the insertion of a block graft,¹ a procedure we refer to as a "Trench". It is our standard practice to obtain a lateral cervical spine radiograph on the first postoperative day. Some surgeons find the radiograph useful to confirm that the correct level has been fused. In addition, the radiograph will provide some information about the degree of retropharyngeal swelling due to haematoma. However, we interpret the significance of postoperative cervical radiographs in conjunction with the clinical findings. If the patient is symptomatic and the radiograph shows a graft abnormality an early re-exploration may be undertaken. In an asymptomatic patient, partial anterior slippage of the graft or minor degree of collapse are considered acceptable and the patient is followed up. If the patient develops symptoms or signs then a late re-exploration may be performed.

Between 1979-89, 822 patients had anterior decompression and fusion at our department. Fifty one of these had further anterior decompression, 27 at a different level and 24 at the previously operated level. In 12 of the latter group, re-exploration was related to collapse or extrusion of the bone graft. The other 12 patients had a re-exploration to remove retained osteophytes or disc fragments. Other patients not counted in this group included one who was re-explored for infection following a cervical discectomy without a fusion and three who had reoperation to evacuate a postoperative haematoma in the neck. Preoperative radiographs were carried out in all cases to identify the correct level before and therefore none of our patients had a fusion at the wrong level. We carried out a retrospective study of the 12 patients that required re-exploration because of a graft complication. The postoperative clinical features and the early check cervical spine radiograph were analysed to find out if the clinical features alone are a reliable predictor of the patients with a graft complication that may necessitate a re-exploration. In this group of patients four had a one level Cloward operation, two had a two level Cloward operation, three had a one level Smith-Robinson procedure, two had a three level "Trench" procedure and one had a four level "Trench" procedure.

The graft had collapsed in six cases, slipped anteriorly in five and slipped posteriorly in one case. The early check radiographs

showed a serious abnormality which necessitated an urgent re-exploration in four cases, each of whom was symptomatic at the time. In four other cases, the early radiograph showed a minor abnormality which was initially managed conservatively. Re-exploration was performed between two to four months postoperatively when the complication worsened radiologically and was accompanied by symptoms and signs. The final four cases had a satisfactory early check radiograph and the complication became obvious between one week to five months later. They all had clinical symptoms that prompted us to repeat the radiographs.

It was clear that each of the 12 patients had an obvious clinical problem at the time of re-exploration whether it was early or late. These were: severe brachialgia affecting the asymptomatic side in two cases, severe dysphagia in four cases, recurrence and/or worsening of myelopathy in four cases and recurrence of brachialgia in one case. In one other case there was no postoperative improvement and the patient's myelopathy slowly deteriorated. In our study we did not encounter a single case that was re-explored on the basis of a graft abnormality on the plain cervical radiograph alone.

Our rate of reoperation related to a graft complication following anterior cervical fusion was 1.45%. It is most likely that of the 822 patients that had anterior cervical fusion at our unit, a number of them had a minor degree of graft abnormality on the postoperative radiograph. These were managed conservatively and did not develop symptoms and signs to necessitate a reoperation. Lunsford² reported a reoperation rate of 4% following anterior cervical discectomy and fusion and the reoperation rate reported by Williams³ was 5.1%.

In our experience a significant graft abnormality that necessitated a reoperation was always associated with a definite clinical problem. Abnormalities on the cervical spine radiographs in asymptomatic patient could be managed conservatively.⁴ It is possible therefore to conclude that an early radiograph after a cervical fusion is unnecessary in a patient who is asymptomatic or improving. A radiograph is needed only if the patient complains of dysphagia, persistence or worsening of their radiculopathy or myelopathy. Late recurrence of symptoms or signs is an indication for further radiographs to assess the condition of the bone graft.

While we acknowledge that there are many reasons for an early postoperative radiograph, we do not feel that these reasons are sufficiently convincing in an asymptomatic patient. A patient who had a wrong level fused will continue to have symptoms. An experienced spinal surgeon will know the adequacy of his spinal grafting at the end of the operation and is thus able to select the high risk asymptomatic patients who need to be carefully followed up with postoperative radiography. This decision will take into account the pathology (for example, rheumatoid arthritis), the preoperative assessment (for example, cervical instability) and the technical aspect of the operation (for example, osteoporotic bone graft and unsatisfactory positioning).

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MATTERS ARISING

Akathisia following traumatic brain injury

Akathisia following traumatic injury involving right parietooccipital and orbitofrontal areas may be due to blockade of dopamine¹ lateralised to the right hemisphere.² Fluoxetine-induced akathisia in women with obsessive-compulsive disorder³ whose right hemisphere is at a higher metabolic rate⁴ suggests that inhibition of dopamine, which is mediated by serotonin in obsessive-compulsive disorders, may occur as a result of post-traumatic alteration of metabolism in the right hemisphere. This lends further support to the role of decreased dopaminergic activity in the prefrontal cortex in akathisia.¹

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Isolated palsy of the fourth cranial nerve caused by an intracavernous aneurysm

An interesting case of isolated trochlear palsy due to an intracavernous carotid aneurysm was recently reported by Maurice-Williams and Harvey.¹ Two points of contention arise in this report: 1) the need for performing carotid angiography early in the course in an older patient with an isolated fourth nerve palsy, and 2) has a cause and effect relationship between intracavernous aneurysm and fourth nerve palsy been conclusively shown in this case?

As they acknowledge in their report, isolated trochlear palsies are most commonly caused by trauma and vascular disease.² In patients over the age of 50 years, an ischaemic

infarction of the peripheral trochlear nerve presumably occurs. As with other isolated cranial mononeuropathies in this age group, ipsilateral fronto-temporal headache and browache may be associated. Invariably, spontaneous recovery of double vision occurs in the ensuing months. In younger patients, a congenital trochlear palsy with decompensation should be ruled out by carefully measuring vertical fusional amplitudes in primary gaze. Ocular myasthenia may simulate any pupil sparing, painless ophthalmoplegia and should be investigated with intravenous edrophonium testing when suspected. Only rare cases of solitary fourth nerve palsy due to compressive causes or aneurysms have been reported. In one such instance,³ a fourth nerve palsy in a 51 year old was seen to progress over a 2 year period. Investigation revealed an intracavernous meningioma as the cause. Neuro-imaging (CT, MRI) should be reserved for those cases of fourth nerve palsy which are not truly isolated, which do not improve after several months of observation, or are shown to be progressive in nature. Carotid angiography seems warranted only in those rare situations in which an associated subarachnoid haemorrhage (such as due to basilar artery aneurysm) has been shown by lumbar puncture or computed tomography.

Intracavernous aneurysms that cause cranial neuropathy are often large and easily demonstrable on CT or MRI. Pain is often associated and remission of symptoms and signs is unusual.⁴ Invariably the oculomotor nerve is involved although an isolated abducens nerve palsy may have been seen. As stated by Maurice-Williams and Harvey, only one case of intracavernous aneurysm producing solitary palsy has been previously reported² and in that case cranial nerves III and VI were later involved. Based on the existing data, I feel therefore that the fourth nerve palsy in their case was most likely ischaemic in origin and that the small aneurysm seen on angiography was probably coincidental. This interpretation would be more consistent with the spontaneous remission which occurred.

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BOOK REVIEWS

Chronopharmacology in Therapy of the Epilepsies. Edited by FE DREIFUSS, H MEINARDI, AND H STEFAN. (Pp 187; Price: \$110.50). New York, Raven Press. 1990. ISBN 0-88167-626-8.

Chronopharmacology, the study of the influences of biological rhythms on the kinetics and effects of drugs and conversely the effect of drugs on these rhythms, is emerging as an important concept which as yet has not made significant breakthrough into clinical practice, apart from the timing of drug administration according to its pharmacokinetics. The effect of meals, for example, the delay in absorption of VPA given during or after meals which occurs within specific time intervals may be more important than hitherto recognised as may be the effect of sleep or wake, night or day.

In fact there are at least five factors which have to be taken into account; the nature of the galenic preparation, the circadian time of treatment, the timing, quality and quantity of meals in relation to drug timing, the age and gender of the patient and the differences in people's genetic characteristics. One can anticipate that in the future a specific drug may be required to be taken at a specific time of day (or night) in relation to a specific dose and a specific uniform meal so as to obtain constant and reproducible therapeutic drug levels. The mind boggles at the introduction of these variables into drug trials!

Regarding this volume, I find the chapters by Smolensky and Renberg on medical chronobiology with special reference to temporal patterns in epileptic seizures and by Newmark and Dubinsky on the significance of seizure clustering most informative from the clinical point of view. The volume is not particularly well produced, the variable print settings of the different chapters being somewhat disconcerting. This book is of interest to neuropharmacologists, and not particularly at present to most clinical neurologists.

GAB DAVIES-JONES

The Genetics of Neurological Disorders 2nd Edition Series: Oxford Monographs on Medical Genetics—18. By M BARAITSER. (Pp 733; Price £30.00). Oxford University Press, 1990. ISBN 0-19-261813-X(pbk).

Neurologists reared on the late RTC Pratt's seminal and studious work will welcome Baraitser's 2nd edition. The expansion of knowledge has not discouraged him, nor caused him to view Pratt's seminal book as a *damnosa hereditas*. Though many diseases considered are unique or rare, the range is now so wide that clinical neurologists are commonly asked to provide both diagnostic and genetic counsel in routine clinics.

In most common diseases: epilepsy, migraine, MS and Parkinson's disease this is

established practice. But, the author chastens clinicians, suggesting that genetic counselling is badly done. Whether or not this justifies the subject as a new clinical specialty in every centre is more arguable, since counselling rests above all on unerring diagnosis—an area in which the neurologist should be better accomplished than the geneticist whose territory is far wider, spanning from abetalipoproteinaemia to Zellweger's syndrome.

Baraitser's first edition was a *tour de force*. This second one reflects the rapid expansion of categories of both rare and common problems with hereditary components. As a comprehensive source of reference it is unrivalled. Each section provides a succinct digest of the salient clinical features of the disorder, its genetics, and anticipated risks to other members of the family. The list is truly encyclopaedic, as testified to by the 209 pages of bibliography—an invaluable and necessary part of such a work.

Where it falls down is that explanation, mechanisms and pathogenesis are often neglected so that we end up with a rather lacklustre compilation. The inclusion of even a brief statement would provide a more intelligent and considerably enlivened text; but, presumably the objection is that this would expand an already bulky book. Readers may have the feeling that the enormous number and diversity of very rare syndromes would be better assembled at some central computer base with suitably convenient access for syndrome hunters. Indeed such is the scale of this laborious volume that one suspects much of its contents may already exist on hard disks. It might be better in future editions to include subsections in compendium form, with smaller typeface and line spacing, for the more esoteric syndromes, for example: those with microcephaly, hypertelorism and funny noses; or, the rarest types of lipid, glycogen and ganglioside storage diseases; this might afford more space for short discussion of why, or how, the protean manifestations of these and other diseases are produced.

This criticism is of a peccadillo in an otherwise unique compilation of great importance which sets out clearly all the current information about genetic elements, markers and calculations of risk. No neurologist can afford to be without a copy close to hand.

JMS PEARCE

Coronary and Cerebral Vascular Disease. A Practical Guide. By LA ROLAK AND R ROKEY. (Pp 381; Price: \$52.00.) New York: Futura Publishing Co, 1990. ISBN 0-87993-353-4

This book is designed to be a practical guide to management of patients with atherosclerotic disease of the heart and brain (sic); it is admirable, puzzling but a little dull. Admirable because it reviews many topics which would be of considerable interest to the physician (and the occasional neurologist) who takes an interest in cerebrovascular disease. The book is divided into three sections. The first deals with the pathogenesis, clinical features and epidemiology of coronary and cerebral vascular disease. The second covers "the patient with concomitant stroke and myocardial infarction" the third and perhaps most useful, covers "the management of the