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 three or more cervical spine levels. Occasionally we employ the technique of partial multiple vertebralbody with the insertion of a block graft, a procedure we refer to as a "Trench". It is our standard practice to obtain a 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 is considered acceptable and the patient is followed up. If the patient develops symptoms or signs then a late re-exploitation may be performed.

Between 1979-89, 822 patients had anterior cervical 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-exploitation was related to the removal of a block of bone from the bone graft. The other 12 patients had a re-exploitation to remove retained osteophytes or disc fragments. Other patients not counted in this group included one who was re-exploited 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 ensure 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-exploitation 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 secondary re-exploitation. In this group of patients four had a total Cloward operation, two had a total Cloward operation, three had a total 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-exploitation in four cases, each of whom was symptomatic at the time of operation. In four other cases, the early radiograph showed a minor abnormality which was initially managed conservatively. Re-exploitation was performed between two and 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 re-explore the radiographs.

It was clear that each of the 12 patients had an obvious clinical abnormality at the time of re-exploitation 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 case that was re-exploited on the basis of a graft abnormality on the plain cervical radiograph alone.

Our rate of reoperation related to a graft complication in 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 but were managed conservatively and did not develop symptoms and signs necessitating reoperation. Lunsford reported a reoperation rate of 4% following anterior cervical discectomy and fusion and the operation rate reported by Williams 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. It is possible to conclude that an early radiograph after a cervical fusion is unnecessary in a patient who is asymptomatic and 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 sufficient 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 the decompression 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).

MATTERS ARISING

Akathisia following traumatic brain injury

Akathisia following traumatic brain injury involving right parietooccipital and orbitofrontal areas may be due to blockade of dopamine 1 lateralised to the right hemisphere. Fluoxetine-induced akathisia in patients with obsessive-compulsive disorder whose right hemisphere is at a higher metabolic rate 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.

REFERENCES


Chromopharmacology, 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.

GAR DAVIES-JONES


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 damnosum 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 counselling.

In most common diseases: epilepsy, migraine, MS and Parkinson's disease this is established practice. But, the author chastes clinicians, suggesting that the genetics of epilepsy 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 abeta-lipoproteinemia to Zellweger's syndrome.

Baraitser's first edition was a tour de force. This second one reflects the greater recognition 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 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 lacunae-strewn presentation. For example, in a brief statement would be provided a more intelligent and considerably enlivened text; but, presumably the objection is that this would expand an already bulky book. Readers may wonder the feeling that a large 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 in future editions to include subsections in a compendium form, with smaller typeface and line spacing, for the more esoteric syndromes, for example: those with microcephaly, hypotelorism 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 protein 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 at hand.

JMS PEARCE


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 clinical response to myocardial infarction" the third and perhaps most useful, covers "the management of the