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 reported[2] 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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1 Maurice-Williams RS, Harvey PK. Isolated palsy of the fourth cranial nerve caused by an intracavernous aneurysm. *J Neurol Neurosurg Psychiatry* 1989;52:679.
2 Rush JA, Younge BR. Paralysis of cranial nerve III, IV and VI. Cause and prognosis in 1000 cases. *Arch Ophthalmol* 1981;99:76–9.
3 Slavin ML. Isolated trochlear nerve palsy secondary to cavernous sinus meningioma. *Am J Ophthalmol* 1987;104:433–4.
4 Trobe JD, Glaser JS, Post JD. Meningiomas and aneurysms of the cavernous sinus. Neuro-ophthalmologic features. *Arch Ophthalmol* 1978;96:457–67.

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 reproduceable 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, hyperteleorism 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