There are also practical and concise chapters on operative positioning and perioperative management, and on cranial nerve function monitoring.

The scope of the book is therefore very wide, but—no criticism intended—those seeking information on vascular lesions or intra-axial tumours should look elsewhere. The text is invariably practical and authoritative, and the illustrations uniformly good. I could not fault the index.

This book is probably a must for departmental libraries, and will be welcomed by all neurosurgeons undertaking surgery in the posterior fossa, and perhaps particularly by those preparing to sit specialty Fellowship.

AJ STRONG

Cerebral Small Artery Disease. (Advances in Neurology, Vol 62). Edited by PM PULLICINO, LR CAPLAN and M HOMMEL (Pp 241, Illustrated; Price: $123.50). 1993 New York, Raven Press. ISBN 0-7817-0051-5 (Order Code 2591)

In the Middle Ages a favoured topic for debate among theologians concerned how many angels could rest on the point of a needle; latter-day neurologists debate with equal vigour (but no more success) the doctrine of the lacune. These perplexing holes were first well described by French pathologists as small cavities within the substance of the brain and thought by most to be ischaemic in origin. Other pathological conditions resembling but distinct from lacunes were recognised and the term came to be used to describe small circumscribed deep-seated infarcts. Early attempts to correlate individual lacunes with clinical symptoms and signs were hampered by the fact that there was no way of locating with certainty the site of lesion during life and when the patient eventually came to autopsy the lacunes were usually multiple. Pierre Marie however recognised the clinical correlate of longstanding *état lacunaire* as a pseudobulbar palsy, spastic quadriparesis and dementia.

In the 1960s Fisher using a classical clinico-pathological approach defined a group of clinical syndromes (pure motor stroke, ataxic hemiparesis, dysarthria-clumsy hand syndrome) caused by small deep infarcts in specific locations. It soon became clear that the same syndrome could result from a lacune in a number of different sites, that a clinical lacunar syndrome did not always signify a small deep-seated lesion and that pathological lacunes could be found in the absence of any symptoms.

In recent years modern imaging has opened up the possibility of clinico-pathological correlation during life. However radiolorgical changes may be deceptive—not all regions of signal change are infarcts, the lesions are often multiple and the size of the lesion on imaging (an important diagnostic criterion) may differ greatly from its pathological size.

We end up with three definitions of lacunes—pathologists mean a small deep infarct in certain sites, clinicians mean one of a number of neurological syndromes and radiologists mean a region of signal change of a specified size and location. The possibilities for argument and misunderstanding are endless.

As if this were not enough, the disagreement about the nature of a lacune has developed into a further argument about its cause. Modern views on this have again been much influenced by Fisher who regarded a lacune as a small deep infarct exclusively in the territory of a penetrating artery. In most cases the penetrating artery is occluded by lipohyalinosis, a consequence of chronic hypertension, but in a few occlusion results from atheroma or embolism. Furthermore the artery is not always completely blocked and it now appears that small deeply placed infarcts in distal field territories can be caused by haemodynamic events such as systemic hypotension in patients with occlusions of large extracranial arteries.

This well-produced book deals extensively with these controversies and leaves the reader (like FE Smith's judge) possibly none the the wiser but certainly better informed. There are excellent reviews of cerebrovascular anatomy, microvascular territories, arterial pathology, radiology and clinical syndromes and much speculation on pathogenesis, notably a well-balanced and informative chapter from Dr Pullicino, one of the editors. The illustrations are also unusually good and the chapters are fully referenced. All in all this is a book to be recommended to all those interested in cerebral vascular disease and one which shines some welcome light into dark places and small holes.

R ROSS RUSSELL

The Biology of the Autistic Syndromes/2nd Edition. By C GILLBERG and M COLEMAN. (Pp 317, Illustrated; Price $37.50). 1992 Cambridge University Press. ISBN 0-521-43228-6.

This is a remarkable book. No one who deals with autism will ever be the same after reading this book which is a fresh edition of the original title by Coleman and Gilberg published by Praeger New York in 1985. In scope and detail it is as rich as an old-fashioned Christmas cake, not to mention the hidden charms which may cause injury if not noticed. In so far as the book is aimed not only at psychiatrists and neurologists but also at psychologists special educators and even parents of children with autism, it is inevitable that which components are indigestible will vary from one readership group to another.

Originally there was Kanner's infantile autism but that has not been a helpful concept nor has the idea of "pervasive developmental disorder": autism is a specific neurological symptom complex not nearly as pervasive as in less politically correct times was called mental deficiency or mental retardation. Having teased that out the authors review the clinical, epidemiological, genetic, biochemical, endocrine, immunological, electrophysiological, brain imaging, neuropathological and neuropsychological literature with extensive references.

I have concerns that child psychiatrists who read this book may do the recommended neurological examination and the recommended investigations without the intermediary of a paediatric neurologist, a difficulty being that the authors are not neurologists themselves. My substitution for the second part of table 25.1 (neuropsychiatric assessment check list for autism) would be first to get a first quality paediatric neurologist to conduct the examination (for example one doesn't just look for Lisch nodules as confirmation of type I Neurofibromatosis—one arranges for a slit-lamp by the ophthalmologist). You may investigate using the philosophy of testing outlined in Handbook of Neurological Investigations in Children. This table ends with a novel inverted definition of the milk-maid sign.

There are lucky charms in this book as for example in the section on epilepsy. There one finds for example a fine reference to "autisme convulsi?" to emphasise that not only do epilepsy and autism go together but that autism might be a symptom of the epileptic process, the manifestation of one type of minor epileptic status. This is an autism treatable by anti-epileptic medication. There is uncontrolled evidence that vigabatrin when it abolishes infantile spasms may reduce subsequent autism liability.

J B P STEPHENSON

SHORT NOTICES

Electrical and Magnetic Stimulation of the Brain and Spinal Cord (Advances in Neurology, Vol. 63). Edited by O DEVINSKY, A BERIC AND MICHAEL DOGALI. (Pp 323; Price: $139.00) 1993. New York, Raven Press. ISBN 0-7817-0066-3.

Anatomic Localization for Needle Electromyography. By STEVE R REIRINGER. (Pp 154 Illustrated; Price: $28.95) 1994. Philadelphia, Hanley & Belfus Inc. ISBN 1-56053-068-5.

CORRECTION

Currier RD, Haerer AF, Meydrech, EF. Low dose oral methotrexate treatment of multiple sclerosis: a pilot study. *J Neurol Neurosurg Psychiatry* 1993;**56**:1217–18. The final sentence on p.1217 should read "An exacerbation was defined as a focal neurological worsening lasting at least two weeks followed by incomplete or complete recovery and so judged by an experienced neurologist."