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MATTERS ARISING

Non-vascular aetiology of lacunar syndromes

We have read with interest the paper by Anzalone and Landi¹ on non-vascular causes of lacunar syndromes. From 31 August 1985 to 31 October 1989, we studied prospectively our patients from the stroke clinic that included several cases of lacunar infarctions and lacunar syndromes. We have found several cases of lacunar syndromes due to non-vascular aetiology. The main cause of lacunar syndrome in our patients was neurocysticercosis.

Some of these cases have been reported elsewhere.²⁻⁵ Our series include 12 patients aged 18 to 57 years, mean 34.5 years. During the last four years 733 patients attended our stroke clinic and from these we found 114 cases with lacunar syndrome. The twelve cases due to neurocysticercosis are 8.3% of lacunar syndromes.

In two patients the lacunar syndrome (ataxic hemiparesis in both) were produced by parenchymal brain cysticercosis,² in the remaining patients the lacunar syndrome was produced by cerebral infarction (lacunar infarction). In most cases it was located in the capsular area and in only two patients was the lesion located in the subcortical area. These infarctions were associated with subarachnoid cysticerci. It has been well recognised that subarachnoid cysticerci usually induced subacute, chronic or recurrent meningitis with abnormal thickening of leptomeninges at the base of the skull and inflammatory

entrapment of blood vessels around the circle of Willis. The histopathological findings in these vessels usually include fibrosis of the media and endothelial hyperplasia and occasionally complete occlusion.

As in the series of Anzalone and Landi¹ a past history of arterial hypertension was uncommon in our series.

The lacunar syndromes in the present series included sensorimotor syndrome in five patients, pure motor hemiparesis in four and in three patients ataxic-hemiparesis. Two cases had cysticerci in brain parenchyma, suprasellar in five, one interpeduncular, one insular, one meningeal and diffuse arachnoiditis in two cases.

Cerebral angiography was performed in five patients, and evidence of vasculitis was found in three patients.

Cysticercosis is not usually considered in the differential diagnosis of lacunar syndromes, this becomes important in areas of the world where cysticercosis is endemic.

We agree with Anzalone and Landi¹ with the concept that early CT scanning must be performed in patients with lacunar syndrome, particularly if the patient is young, normotensive or resident in countries where neurocysticercosis is a frequent health problem. In the latter we would strongly recommend the routine examination of cerebrospinal fluid including immune reactions for cysticercosis.

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Crying and laughing after brain damage

We doubt that Allman's suggestion¹ will find much support, yet the problem he considers is a real one. It is not only a question of facilitating communication. An important aspect is the need for a term that, like most other medical terms, can be used in various parts of speech without recourse to cumbersome circumlocution. The indispensable need, however, is for a term that recognises the essential feature of the phenomenon, namely that it is a disorder of the mechanisms of expression of emotion, and not of emotion or affect itself. This is what is wrong with the suggestion "emotionalism".

Perhaps the solution for this problem of nomenclature demands a lateral flight of fancy. The vocabulary of medicine is predominantly Greek. On the whole, this poses no problems, indeed it helps to avoid the contamination of meaning that often attends the use of English expressions through their vernacular connotations. The traditional symbol of the expression of emotion is the ancient Greek diptych of the theatrical laughing and crying masks, known

as *prosopion*. This suggests *dysprosopiea*, allowing the equally simple adjective *dysprosopieic*. Where Sigmund Freud has trod, we surely dare to go.

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BOOK REVIEWS

Therapy of Parkinson's Disease. Neurologic Disease and Therapy Series 5). Edited by WC KOLLER AND G PAULSON (Pp 583 Illustrated; Price \$125.00 (US and Canada), \$150.00 (All other countries.) New York: Marcel Dekker Inc. 1990. ISBN 0-8247-8219-4

Having enjoyed reading and commending Koller's *Handbook of Parkinson's Disease* last year, I looked forward to learning more about therapy from this new book edited jointly with Paulson. Their preface reminds us, perhaps a little unkindly, that few of the contributing authors are old enough to remember patients with end-stage Parkinson's disease "who lay in sawdust" . . . or, those given levodopa in whom we "witnessed dramatic changes as patients in Stages III or IV became completely functional". Very true, but I am not sure we would all agree with Koller's introductory remark that "the types of therapy have dramatically increased *pari passu* . . . due in large part to the discovery of MPTP model of parkinsonism." The contents show 69 contributors in serried ranks, amongst whom are included almost all of the USA doyens as well as a selection of Europeans.

The five main sections cover Assessment and measurement of symptoms and signs, Pharmacologic agents, Surgical approaches, Diet in therapy, and Other therapeutic approaches.

The result is a very useful and comprehensive survey of treatment which is up-to-date, well referenced and authoritative. Modern techniques range from apomorphine, a rebirth of stereotaxic thalamotomies, appraisal of neurotransplantation, levodopa infusions and, to be right up to the minute the DATATOP trial of selegiline and tocerolol; all are reviewed in detail.

There are however faults. In a fact laden text, good writing and thereby easy reading are just as important as in a more discursive series of essays. And here the book disappoints, despite the publisher's attractive printing format illuminated by clear illustrations and diagrams. Many contributors have careened the edifice of rational, reflective prose in favour of an almost obsessive devo-