Leigh's disease is rather a nosological mess at the present time with cases being reported from various times of life, and with various bits of evidence incriminating different metabolic pathways in the energy generating system. Provided that we keep our eyes firmly fixed on the fact that acute energy deprivation seems to be at its root, and this may come about from various causes, both environmental and genetic, it becomes easier to think constructively about the fundamental nature of the problem.

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The syndrome of irreversible lithium effectuated neurotoxicity

SIR: I read with interest the case report by Tesio et al1 describing a cerebellar syndrome in lithium poisoning. There is a general lack of awareness about irreversible and untreatable complications of lithium treatment2 despite evidence to contrary.3-5 Till recently it has been maintained that the side-effects of lithium are not disabling.2 I identified 55 cases of persistent sequelae of lithium therapy after a review of published literature though earlier reviews had given a smaller number.3-5 Some of the cases of persistent sequelae of lithium therapy have been mistaken for neuroleptic malignant syndrome6 owing to a superficial resemblance. Moreover, some cases of longlasting sequelae of lithium therapy may occur without having acute poisoning, a fact which has not been appreciated in earlier reviews.3-4 Though most common sequelae are persistent cerebellar symptoms, other clinical manifestations have also been documented. In a typical presentation, acute lithium poisoning precedes the sequelae and the acute phase is generally without cerebellar symptoms.4 As consciousness returns the neurological sequelae become more apparent.4 In four cases cerebellar signs were present from the beginning of the acute phase in the cases I reviewed. Atypical presentations may include persistent papillodema, optic neuritis, isolated downhill dystagnus, peripheral neuropathy and myopathy. Those with atypical presentations are unlikely to have undergone an acute organic brain syndrome. In such cases symptoms develop insidiously while on long-term lithium therapy and persist after discontinuation for varying periods. Prognosis generally is good and in some cases of chronic lithium neurotoxicity the neurological signs may resolve in less than two months after discontinuation.7-10 These cases7-9 cannot be termed longlasting according to criteria laid down by Schou.4 In general spontaneous recovery may occur in varying degrees over a period of time. Some cases, however, may be unchanged and irreversible. Complete neurological recovery is uncommon but patients may respond to rehabilitative measures with significant functional gains and may return to previous living arrangement.11

I suggest that these persistent sequelae of lithium be called the syndrome of "Irreversible Lithium Effectuated Neurotoxicity." Extensive demyelination has been found by biopsy of peripheral nerves so involved. It is likely that toxic demyelination at various sites in the central nervous system especially in the cerebellum may be the mechanism involved in the aetiology of this syndrome.

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Matters arising

References

9 Saul RF, Hamburger HA, Schorst JB. Pseudotumour cerebri secondary to lithium carbonate. JAMA 1985;253:2689-70.

Tesio replies:

I agree with Dr Adityanjee's suggestion that we recognise the persistent sequelae of lithium poisoning as a syndrome to be named the syndrome of irreversible lithium effectuated neurotoxicity.

First, however, I think we should state more definitely the specific features of this syndrome and the minimal criteria for its diagnosis.

The case I and my coworkers described in a previous paper,1 the review of the literature2 and Dr Adityanjee's letter itself suggest that only persistent neurological deficits following acute intoxication could form specific syndrome pathognomic for lithium poisoning. Minimal
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criteria for diagnosis could be the following: (1) onset with acute organic brain disease, (2) anamnestic and/or laboratory evidence of lithium over dosage prior to the onset of acute organic brain disease, (3) simultaneous appearance of severe cerebellar signs and of mild peripheral neuropathy as consciousness recovers, (4) persistence of cerebellar signs six months after poisoning and discontinuation of lithium therapy.

This last point is supported by the evidence that substantial improvements of the neural deficits themselves cannot be expected after six months, though functional improvement may occur3 up to one year after poisoning.1 4

As Dr Adityanjee remarks, non-nervous acute and chronic sequelae, such as renal and/or cardiac failure, Grave's disease and myopathies may complicate the clinical picture. These sequelae may also be dramatic enough to point to some form of acute intoxication,1 and/or persist to various degrees of severity4; nevertheless, they seem to lack the specificity needed for a suspicion of lithium poisoning either in the acute or in the chronic phase.

Dr Adityanjee claims that neurological signs and symptoms may well follow chronic lithium intoxication, too; however, he also recalls that usually they are much more various and reversible than those following acute intoxication. Thus, I suppose he would agree that the identification of a specific and irreversible syndrome in chronic toxicity should be much more questionable.

Certainly, limiting the diagnosis of the syndrome to the cases fulfilling the above minimal criteria should make this syndrome a rather rare finding. At the same time, however, the syndrome itself could be identified and related to lithium poisoning with a higher degree of specificity.

References

Book reviews

Symptom Management in Multiple Sclerosis.

The editor of this small book is the Director of Rehabilitation at a multiple sclerosis centre and the Clinical Professor of Neurology at the University of Minnesota. He has assembled a team of twelve to describe the management programme developed at their rehabilitation unit.

For each symptom there is a brief explanation, a mention of drug treatment and a variety of rehabilitation therapies. The contributors are in the main nurses, occupational and physical therapists and it is these supportive ancillary professions that the main appeal of this book will find favour.

There is an excellent introductory summary of the CNS in two pages, a brief but equally lucid statement on "multiple sclerosis is...". Then chapters on tremor, spasticity, weakness, fatigue, sexuality, diet, pressure sores, bladder and bowel etc. Each is commendably brief, simple and clear. In fact most sections left one wanting to know more detail of what was done in Fairview Hospital. Although the neurologist will learn nothing new, it may be helpful for him to read about the way others handle his patients.

The editor also has in mind the multiple sclerosis patient, family and friends, who doubtless will find interesting and readable accounts which will aid their understanding of what are nowadays termed "therapeutic goals".

Simple plain writings often conceal considerable labours in their preparation. This small practical volume is welcomed and will benefit a wide range of readers.

JMS PEARCE


This, the 46th volume of the Advances in Neurology series, is only the second to be devoted wholly to diagnostic investigation. As many readers will agree, the issues in this series have varied considerably in their worth, and this current edition is only moderately useful. These multi-authored books usually are composed of formal reviews, and this volume is no exception. It is the publication of the proceedings of a conference on Intensive Neurodiagnostic Monitoring, held as a satellite to a meeting of the American Epilepsy Society. The book is therefore almost entirely concerned with monitoring in epilepsy, and is a largely American view (indeed, the style and imprimatur of the American Epilepsy Society is very obvious throughout). It comprises 18 chapters grouped into sections on: combined video and EEG recording, ambulatory EEG, intensive monitoring in specific problem areas, data reduction and a summary section. My main criticism of the work is that it covers an area already well reviewed, often by the very same authors—and indeed some chapters follow very closely previously published papers. Most of the chapters go over well trodden ground, and while many are well written, the whole has a pedestrian feel. The book must have been some time in conception, as few (any?) of the references post date 1985 (the date of the conference was not given). The best chapters are the most original or the most technical, and these include the two chapters on data reduction (by Gotman and Frost), and that on ambulatory ECG and EEG monitoring (by Blumhardt). Ives gives a simple but succinct review of video recording, and Kellaway an authoritative review of monitoring in children. The chapter by Engel and Crandell, is interesting in seeming to show a much more cautious approach to intracerebral recording than in their previous publications, and their indications for this procedure seem to have narrowed. Omissions from the book include any mention of intensive care monitoring or the monitoring of sleep. In 1985, Supplement 37 of the journal Electroencephalography and Clinical Neurophysiology was published as a hard back volume, to celebrate the 50th anniversary of the Montreal Neurological Institute, entitled Long Term Monitoring in Epilepsy (Ed Gotman, Ives and Gloor). This was a much more interesting and original, and more technically accomplished book.

SIMON SHORVON