CORRESPONDENCE

Corpora amylacea in hippocampal sclerosis

I read with pleasure the short report of Van Paesschen *et al.*¹ This finding perhaps may raise some interest again in corpora amylacea as well as in the pathology of temporal lobe epilepsy. As I studied corpora amylacea for about 25 years I call the attention of the authors to the first—but important—finding of Ramsey who published in her article in 1965² a case of a 35 year old man who had intractable temporal lobe epilepsy. Surgical intervention was performed with temporal lobe resection including 3 cm of hippocampus. The pathologist's report stated that the tissue seemed histologically normal except for a few areas of pyknosis and shrinkage of neurons and a decrease in the number of neurons in the hippocampus. The presence of corpora amylacea without gliosis was noted and was especially prominent in the hippocampus. The diagnosis was "mild presenile cortical atrophy of the temporal lobe with corpora amylacea in the hippocampus."

I studied the occurrence of corpora amylacea in 1407 cases of various diseases with special reference to the so called predilection sites of corpora amylacea, and I sometimes found large numbers of corpora amylacea in the hippocampal area and other regions with and without special pathology, even in young people. The conditions which favour the development of corpora amylacea vary greatly (aging, chronic vascular—hypoxic—diseases, ALS, multiple sclerosis, dementias, etc) as well as various pathogenetic mechanisms promoting formation, which included chronic hypoxia, neuronal degeneration, external hydrocephalus—as a consequence of local cortical atrophy—diabetes mellitus, and other processes which induce stress states expressed by strong HSP 60 positivity in our investigations (see³⁻⁷ for appropriate references). So the corpora amylacea formation really is an epiphenomenon in different diseases, as Van Paesschen *et al* state.

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- 1 Van Paesschen W, Révész T, Duncan JS. Corpora amylacea in hippocampal sclerosis. *J Neurol Neurosurg Psychiatry* 1997;63:513–15.
- 2 Ramsey HJ. Ultrastructure of corpora amylacea. *J Neuropathol Exp Neurol* 1965;24:25–39.
- 3 Leel-Ossy L. The origin and the pathological significance of the corpus amylaceum. *Acta Neuropathologica (Berl)* Suppl II, 1981:396–99.
- 4 Leel-Ossy L. The structure and the pathological significance of the corpus amylaceum. (Hung.) In: Leel-Ossy L, ed. *Selected chapters in neuropsychiatry*. Budapest: Statistics Publishing Co, 1989:189–202.
- 5 Leel-Ossy L. Pathological significance and characteristics of corpus amylaceum. *Neuropathology (Japan)* 1991;11:105–14.
- 6 Leel-Ossy L. The occurrence of corpus amylaceum (polyglucosan body) in diabetes mellitus. *Neuropathology (Japan)* 1995;15:108–11.
- 7 Leel-Ossy L. Statistical evaluation and pathological significance of the incidence of corpus amylaceum. *Clinical Neuroscience (Hung)* 1997;50:90–102.

Secondary hyperkalaemic paralysis

In their report describing a case of hyperkalaemic paralysis associated with renal failure and use of spironolactone, Evers *et al*¹ provide an illustration of the results obtained from conduction studies performed on the patient's median sensory nerve. Utilising the calibration markers, the average amplitude of the sensory action potential (SAP) measures 10.6 μ V before haemodialysis and 27.2 μ V afterward. However, these measurements are recorded in table 1 as being 20.0 μ V and 24.0 μ V, respectively.

An increase in SAP amplitude after normalisation of serum potassium concentration would offer additional support the authors' suggestion that the pathological process causing weakness in secondary hyperkalaemic paralysis probably originates at the level of nerve rather than muscle.

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- 1 Evers S, Engelen A, Karsch V, *et al*. Secondary hyperkalaemic paralysis. *J Neurol Neurosurg Psychiatry* 1998;64:249–52.

Evers replies:

The figures in table 1 were indeed incorrect and the correct figures from the original data are 8 μ V before haemodialysis and 24 μ V after haemodialysis.

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Traumatic distal femoral neuropathy

We read with interest the recent report by Padua *et al*¹ on a body building champion with an isolated mononeuropathy of a distal branch of the femoral nerve, having recently seen a similar patient, with a different cause.

A 39 year old sports physiologist underwent a resting muscle biopsy using the Bergstrom technique, for research purposes. He had previously undergone eight of these biopsies on the other leg without adverse effect, and this biopsy was performed by an experienced operator. Under lignocaine anaesthesia the biopsy needle was inserted. As manual suction was applied followed immediately by closing the biopsy needle, the subject had an intense feeling of cramp (that had not been noticed with previous biopsies). The site of the biopsy was 5 cm lateral to the mid-point between the patella and the anterior superior iliac spine. Over subsequent months wasting of the distal lateral vastus lateralis muscle was noted. An EMG 3 months after injury showed increased insertional activity, fibrillation potentials, and positive sharp waves with no activation of motor unit potentials in the distal fibres of the vastus lateralis muscle. A further EMG 6 months after the biopsy showed evidence of reinnervation.

In our patient, the distal mononeuropathy was traumatic, and clearly related to the needle biopsy. A distal motor branch of the femoral nerve as identified in the anatomical studies of Padua *et al*¹ was traumatised by the biopsy. It does raise the possibility of an alternative mechanism for the patient of Padua *et*

al which was not considered in the article, and can be difficult to verify by clinical history alone. The use of anabolic steroids is very prevalent among body builders with the lateral thigh being a common site of administration. Direct injection could have traumatised the nerve, and may also explain the lack of any improvement with time. It is also noted in the illustration of the unaffected leg, that a small dimple is present in the skin at a site where the distal motor branch may be vulnerable, although this is significantly more distal than the biopsy site in our patient. This may, however, be an indicator of previous injections to the lateral thigh.

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- 1 Padua L, D'Aloia E, Lo Monaco M, *et al*. Mononeuropathy of a distal branch of the femoral nerve in a body building champion. *J Neurol Neurosurg Psychiatry* 1997;63:669–71.

The authors reply:

We are grateful for the response of Silbert *et al*. In our paper¹ we described an isolated mononeuropathy of a distal branch of the femoral nerve and hypothesised that stretching and compression of the nerve had probably occurred during strenuous exercise.

We agree with the possibility of a traumatic nerve lesion due to needle injection and we have knowledge of some cases of this kind of nerve injury in body builders, but in the case reported in the article, we specifically asked the patient if drug injections had been carried out in the thigh. He replied that he had never used this kind of drug administration. Moreover, our patient did not refer to any pain in the thigh, whereas in the case of Silbert *et al* an "intense cramp" was felt by the subject. Concerning the figure, detailed clinical examination of both thighs had been performed and no suspected "dumping" was seen. We think that the effect in the figure could rather be due to the hypertrophy of a nearby muscle and as noted it is distal to the site of the femoral nerve branch.

For these reasons, the hypothesis of needle injury was not considered in the article, but we agree that in the case of nerve lesion in body builders, a needle injury (for anabolic drug injection) must always be suspected.

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- 1 Padua L, D'Aloia E, Lo Monaco M, *et al*. Mononeuropathy of a distal branch of the femoral nerve in a body building champion. *J Neurol Neurosurg Psychiatry* 1997;63:669–71.

BOOK REVIEWS

Radiosurgery. Volume Two. Edited by DOUGLAS KONZOLKA. (Pp 268, Sw Fr 258). Published by S Karger AG, Switzerland, 1998. ISBN 3-8055-6547-X.

It has sometimes been said, perhaps unfairly, that radiosurgery has been a treatment looking for a successful application. This volume, the second in the series on radiosurgery, attempts, in some parts more successfully than others, to refute that suggestion. The technique was first established by Leksell, using a collimated cobalt source ("the gamma knife"). In the United Kingdom, the Stereotactic Radiosurgery Centre in Sheffield has been treating patients since 1985. The published results for treatment of arteriovenous malformations have been good and its place in the non-operative management of cerebral arteriovenous malformations is now secure. Radiosurgery has now become the treatment of choice for appropriately sized and located malformations. However, increasingly refined techniques of endarterial obliteration for vascular lesions have also been developing apace in parallel, and it seems likely that the combined use of both techniques will in future make it increasingly rare for us to have to undertake surgery for these often demanding lesions.

The main drawback of the "gamma knife" technique of radiosurgery has been the enormous capital outlay required for the equipment. In the United Kingdom there is only one machine and this has perhaps inhibited a wider application of the technique in the management of intracranial malignancy. The greater availability of machines in the United States, and the commercial pressures to retain the appropriate return on investment, has led to a wider range of applications in the United States and the results of some of these are published in this book. The development of fractionated stereotactic radiotherapy using relocatable stereotactic frame and a

linear accelerator has shown that it is possible to achieve good results with this technique and some of these are also published here. In the United Kingdom we are beginning to identify a much wider range of applications for this technique and, in the not too distant future, we should expect a whole range of reports of its use in malignant brain tumours, recurrences, melanomas, and other metastatic lesions. However, for the moment the literature is rather sparse and it remains to be seen whether the technique in future will also become the treatment of choice for these rather dismal conditions.

The place of radiotherapy in general, and stereotactic techniques in particular, for benign lesions, such as acoustic neuromas and meningiomas, in my view remains uncertain despite some rather encouraging results published here. At this stage it is perhaps sensible to proceed with caution until the long term effects are clearer. With regard to the future, the rapid advances in imaging techniques using MRI, the various new variants of CT, and the PET machine, offer the tantalising possibility of being able to identify both the anatomical and functional pathology to be targeted for treatment. With the merging of these exciting new technologies it may at last be possible to make some real progress in improving the outcomes for CNS tumours.

DAVID HARDY

Handbook of Neurologic Rating Scales. Edited by ROBERT M HERNDON (Pp 276, US\$125). Published by Demos Vermande, New York, 1997. ISBN 1-888799-07-2.

This well produced book joins a small group of books which actually show some of the measures now used in clinical research. There are many, many measures, and each book can only show a selection. This book would be helpful to anyone who is interested in learning about measures of disease severity and outcome, although it might not be the best in any particular circumstance.

Everyone should read the introduction to this book, written by Dr Herndon. It is full of common sense. It also emphasises the most

important point of all, and I repeat it here: "... the most important question in designing or choosing a scale is how well it is suited to the task at hand in terms of validity, efficiency, sensitivity and specificity ...". If only every researcher would read, remember, and act on this one statement then neurological research would advance greatly.

The book is then primarily structured around diseases. This is useful to anyone interested in a specific disease, but means that many non-specific areas of measurement are not covered. The chapters cover most of the common and important neurological diseases: multiple sclerosis; amyotrophic lateral sclerosis; head injury; stroke; movement disorders including Parkinson's disease; epilepsy; and dementia. Many of the chapters follow a similar layout, but there are exceptions. Most seem to be written by people who have direct experience of the scales, and indeed there is much original and useful data relating to the assessment of progression in motor neuron disease in that chapter. There are two more general chapters. The first covers paediatric developmental scales, and I think that this is the only similar book to cover this area. The second covers the measurement of the outcome from rehabilitation. The best chapters are those covering multiple sclerosis, amyotrophic lateral sclerosis, movement disorders, and dementia. The chapter on stroke is much less comprehensive, and contains some errors in references (for example, to the Hamrin ctivity Index), and the chapter on head injury stands out as containing much less useful information.

The book gives considerable useful information about many of the scales mentioned, and certainly would help anyone who was unfamiliar with the field. The references are reasonable, although sometimes inaccurate and quite often statements are made without reference, which can be irritating especially as references do exist to support most statements. Overall this book is good, and complements the other books available well. It will be especially helpful to those interested in the current "hot" diseases, multiple sclerosis, amyotrophic lateral sclerosis, and Parkinson's disease.

DERICK WHITE