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.

L LEEL-OSSY

Departments of Neuropathology, Neurology,
Neurosurgery, and Psychiatry, University Hospital
Tatabánya and Miskolc, Hungary

- 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.

R F GLEDHILL

Department of Neurology, MEDUNSA and
Ga-Rankuwa Hospital, South Africa

Correspondence to: Professor R F Gledhill, Department of Neurology, PO Box 108, Medunsa 0204, South Africa. Telephone 0027 12 521 4136/4209; fax 0027 12 521 4758/560 0086.

- 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.

S EVERS

Department of Neurology, University of Münster,
Albert Schweitzer Strasse 33, D 48129 Münster,
Germany

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.

PETER L SILBERT

Department of Neurology, Royal Perth Hospital, Perth,
Western Australia

ROD MOORE

Sports Medicine, St John of God Hospital, Murdoch,
Perth, Western Australia

BRIAN DAWSON

Department of Human Movement, University of
Western Australia, Perth, Western Australia

Correspondence to: Dr P Silbert, Department of Neurology, Royal Perth Hospital, GPO Box X2213, Perth, Western Australia. Telephone 0061 8 9224 2593; fax 0061 8 93817848; email: psilbert@opera.iinet.net.au

- 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.

LUCA PADUA

PIETRO TONALI
1st Neurologia

ROBERTO PADUA
1st Ortopedia, Università Cattolica

AFaR Osp Fatebenefratelli, Isola Tiberina, Roma, Italy

- 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.