To our knowledge there are as so far no reports on follow up nerve conduction studies of patients with hyperkalaemic paralyse presenting with findings otherwise typical of Guillain-Barré syndrome. Our electrophysiological data reflect the progressive inexcitability of nerve fibres due to extracellular hyperkalaemia leading to multiple functional conduction delays and blocks and thus mimicking acute Guillain-Barré syndrome.

The fact that the sensory nerve action potential (tibial nerve) was initially absent whereas the EMG showed normal electrical activity with insertion activity of muscles suggests that muscle fibres were much less influenced by hyperkalaemia than were nerve fibres. The predominance of medium size and larger motor units on EMG argues against a selective affection by hyperkalaemia of the largest and fastest conducting nerve fibres. Thus a homogeneous slowing of conduction velocities in all sizes of the fibre may be the most likely cause of slowed conduction velocities seen here. Also, conduction failure was present in a substantial proportion of fibres on initial examination. Distal segments of the nerves seemed only marginally more affected than proximal sites. This suggests that conduction defects were diffuse rather than focal.

Recently, a case with hypokalaemia mimicking Guillain-Barré syndrome was reported.1 Electrophysiological in this case showed a predominant decrease of compound muscle action potentials whereas sensory nerve action potentials were preserved. Thus by contrast with the situation in hyperkalaemia, muscle membranes in hypokalaemia seemed to be more severely affected than nerve fibres.

Correspondence to: Dr Markus Naumann, Neurologische Universitätsklinik, Justus-Schneider-Strasse 11, 97080 Würzburg, Germany

Measures of medial temporal lobe atrophy in Alzheimer's disease

Diagnosis of Alzheimer's disease in life is made on clinical grounds, and currently employed criteria are burdened with considerable subjective judgement. In view of the increasing possibility of treatment, an objective and early stage sensitive indicator of the disease could prove extremely valuable. Jobst et al. proposed that a simple CT measurement of the medial temporal lobe might improve in vivo diagnosis of Alzheimer's disease. Patients had pathologically confirmed Alzheimer's disease but dementia was of severe degree (mean mini mental state examination (MMSE) 9-3) and scanning occurred as late as one year before death. Work by Scheltens et al. on other measures of medial temporal lobe atrophy focusing more precisely on the hippocampus (hippocampal height, width of the choroid fissure, and width of the temporal horn), has shown greater atrophy in moderately demented Alzheimer's disease patients than in controls.

At present, there are no data as to which of these indicators of temporal lobe atrophy is more useful in the detection of Alzheimer's disease in its early phase. Therefore, we assessed the sensitivity of the measures obtained with MRI in patients with clinically defined mild to moderate Alzheimer's disease.

Twenty-six consecutive patients with clinically defined Alzheimer's disease (age 53 to 87, mean 71.0 (SD 9.1) years; MMSE 12 to 27, mean 18.5 (4.6) and 21 normal controls (age 53 to 86, mean 70.2 (9.9) years; MMSE 23 to 30, mean 28.9 (1.9)) were recruited in the study. Patients with Alzheimer's disease underwent extensive neuropsychological testing, as previously described. Cases and controls underwent MRI of the brain with a 1.5 tesla MRI system. A three dimensional technique was employed for image acquisition, allowing reconstruction of 1 mm thick slices. Minimum thickness of the medial temporal lobe was measured on axial temporal lobe oriented images 20° caudal to the orbitomeatal line. Hippocampal height, width of the choroid fissure, and width of the temporal horn were measured in the coronal plane according to Scheltens et al. All measurements were made by a single observer, blind to clinical diagnosis, on T1 weighted images. Only the right or left measurement indicating greater atrophy was considered for each subject.

Minimum thickness of the medial temporal lobe for all controls fell between the 5th and 95th centiles of normal values (figure). On average, all measures indicated greater atrophy in patients with Alzheimer's disease (12.0 (2.2) vs 13.7 (1.8) mm in controls; r = 2.97; p = 0.005 for minimum thickness of the medial temporal lobe, 12.6 (2.2) vs 14.2 (1.4) mm; r = 3.07; p = 0.004 for hippocampal height, 4.9 (1.6) vs 3.0 (1.4) mm; r = 4.26; p = 0.0005 for width of the choroid fissure, 6.8 (2.0) vs 3.8 (1.4) mm; r = 6.30; p < 0.0005 for width of the temporal horn). Overlapping was considerable for the first measure, however, and less pronounced for the other measures (figure).

To take into account the effect of age on atrophy, measurements were transformed into multiples of the median (MoM); observed/expected value as computed with linear regression on controls. The best value of MoM discriminating patients with Alzheimer's disease from controls and the relative expected sensitivity were then computed by fitting a gaussian model to patients with Alzheimer's disease and controls with specificity set to 95%. Jobst et al. have shown that in their sample a cut-off of 0.79 MoM for minimum thickness of the medial temporal lobe gave an expected sensitivity of 92%. In our less severely demented patients, we found a similar cut-off of 0.80 MoM for 95% specificity, but the expected sensitivity was only 30%. Expected sensitivity was higher for hippocampal height (39%, cut off 0.84 MoM), width of the choroid fissure (40%, cut off 1.71 MoM), and width of the temporal horn (72%, cut

Correspondence to: Dr K Bushara, Department of Neurology, University of Wisconsin Hospital and Clinics, 600 Highland Avenue, Madison, Wisconsin 53792, USA.

1 Molgo J, Comella JX, Angaut-Petit D, et al. Preemptive actions of botulinum neurotoxins at vertebrate neuro muscular junctions. J Physiol (Lond) 1991;14:1266-

2 Ambache N. A further survey of the action of clostridium botulinum toxin upon different types of autonomic nerve fibre. J Physiol (Lond) 1951;113:1-17.


4 Vita G, Girlanda P, Puglisi, Marabotto L. Neuromuscular reflex testing and single fibre electromyography in botu