Nerve conduction studies before and after normalisation of hyperkalaemia

	Sural nerve		*Tibial nerve (motor)*			*Median nerve (motor)*		
	Amplitude (μV)	*Velocity (m/s) (34° C)*	*Amplitude (mV) (proximal/distal)*	*Velocity (m/s) (34° C)*	*Latency (ms) (proximal/distal)*	*Amplitude (mV) (proximal/distal)*	*Velocity (m/s) (34° C)*	*Latency (ms) (proximal/distal)*
Day 1: K⁺ (8·4 mmol/l)	No response	No response	0·16/0·3	18·0	44·4/22·4	2·7/6·2	20·0	25·0/9·4
Day 10: K⁺ (4·0 mmol/l)	23·0	37·0	16·9/19·6	38·0	16·8/6·2	13·2/13.1	41·0	11·0/4·6
Normal findings	>10·0	>40·6	>2·9 (distal)	>41	<15·1/6·0	>3·5 (distal)	>48·0	<8·8/4·6

abnormalities except slightly slowed conduction velocities (table). Needle EMG of hypothenar and quadriceps muscles showed normal recruitment, duration, and amplitude of motor unit action potentials. An EEG and ECG were in the normal range.

The clinical presentation of our patient with rapidly progressive tetraparesis, areflexia, and mild sensory symptoms would have been entirely consistent with the diagnosis of Guillain-Barré syndrome. This suggestion seemed confirmed by nerve conduction studies showing extremely delayed distal latencies, conduction block (median nerve), and delayed motor and sensory conduction velocities. Only the history of concurrent convulsions was suspicious, but this could have been explained by coincidental hypertensive encephalopathy due to chronic renal disease.

Although slowly rising serum potassium concentrations are usually well tolerated, an acute increase in serum potassium can cause life threatening cardiac complications. Only rarely are pronounced neuromuscular symptoms an initial feature of hyperkalaemia.[1] Neurological symptoms that usually develop at serum concentrations higher than 8 mmol/l[2] include dysaesthesia and weakness, sometimes presenting as Guillain-Barré syndrome.[3,4] The respiratory musculature seems to be relatively well preserved[3] as in our patient.

To our knowledge there are so far no reports on follow up nerve conduction studies of patients with hyperkalaemic paresis 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 (sural nerve) was initially absent whereas the EMG showed normal electrical insertional 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 in fibres of all sizes is the most likely cause of the 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.[5] Electrophysiology 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.

M NAUMANN
K REINERS
B SCHALKE
C SCHNEIDER
Department of Neurology, University of Würzburg, Würzburg, Germany

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

1 Pollok M, Baldamus CA. Störungen des Kaliumhaushaltes. In: Classen M, Diehl V, Kochsiek K, eds. *Innere Medizin*. München: Urban und Schwarzenberg, 1991:1271–4.
2 Sterns RH, Narins RG. Disorders of potassium balance. In: Stein JH, eds. *Internal medicine*. Boston: Little, Brown and Co, 1990:853–6.
3 Radó JP. Successful treatment of hyperkalemic quadriplegia associated with spironolactone. *Int J Clin Pharmacol Ther Toxicol* 1988;26:339–45.
4 Knochel JP. Neuromuscular manifestations of electrolyte disorders. *Am J Med* 1982;72: 521–35.
5 Warner TT, Mossman S, Murray NMF. Hypokalaemia mimicking Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 1993;56:1134–5.

Botulinum toxin and sweating

Botulinum toxin A is widely used in the treatment of dystonic disorders. It acts by inhibiting the release of acetylcholine at the presynaptic level.[1] The neuromuscular blockade it produces is irreversible and recovery occurs over about three months by resprouting of the axons and formation of new acetylcholine receptors.[1] Both motor nerves to skeletal muscles and autonomic cholinergic fibres seem to be similarly blocked by the toxin.[2] In addition to skeletal muscle paralysis, autonomic dysfunction is the rule in botulism, and objective autonomic function tests are abnormal, indicating both sympathetic and parasympathetic dysautonomia.[3] Similar but minor abnormalities in autonomic tests have been reported after local injections of the toxin.[4]

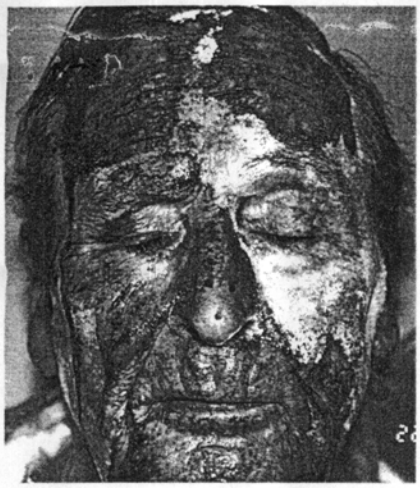
Botulinum toxin has long been known to block postganglionic sympathetic cholinergic fibres to sweat glands in animals.[2] Although it might be expected that the toxin would have a similar effect on sudomotor fibres in humans, objective tests of sweating seem not to have been previously reported in patients with botulism or in those receiving local injections. We studied facial sweating in patients treated with botulinum toxin A injections for hemifacial spasm.

Three patients (one woman and two men aged 69, 76, and 78 respectively) attending the botulinum toxin clinic for one, 1·5, and seven years respectively for treatment of hemifacial spasm agreed to participate in the study. All were receiving intermittent treatment with botulinum toxin A injections (60–120 mouse units; Dysport–Porton Products) around the orbit on the side of hemispasm. None of the patients was receiving anticholinergic drugs. In two of the patients, facial sweating was tested one week after the last injection session. In one patient the sweat test was performed three months after the previous injections and repeated one week after the last session of injections. Sweating was tested with Ponceau red dye and starch mixture. This is a pink powder that changes colour to bright red when wet.

The powder was dusted on to the face with cotton wool. Thermoregulatory sweating was induced by keeping the subjects with the trunk and limbs enveloped in polythene sheeting in a warm humid room. The room temperature was maintained at 32–35°C with an electric fan heater and humidity was maintained by boiling an electric kettle. Heating continued until facial sweating, as detected by the change of colour of the dye, was evident. This took 30 to 50 minutes. The distribution of sweating on the face was charted and photographed.

Facial sensation, pupil size, and reaction were normal in all three patients. Two of the patients had developed a mild ptosis as a side effect of previous injections. In all three patients there was an area of anhidrosis around the orbit including the upper part of the cheek and the side of the nose, the temple, the eyebrow, and the lower part of the forehead, on the side of the injections. The shape and size of the anhidrotic area was similar in all three patients (figure). In the one patient, the result of the sweat test was similar three months after previous treatment.

This brief study seems to provide strong evidence that sudomotor efferents are affected by botulinum toxin A. As in cholinergic neuromuscular transmission, diffusion of the toxin and inhibition of acetylcholine release from the presynaptic terminals of the sudomotor nerves seems to be the underlying mechanism producing anhidrosis after botulinum toxin A injections. A similar anhidrotic effect of the injected toxin was shown by Ambache in



Localised anhidrosis around the left orbit on the side of botulinum toxin injections.

the footpads of kittens.[2] The area of anhidrosis does not correspond to any particular peripheral nerve distribution. In the absence of sensory disturbance it is unlikely that anhidrosis is the result of inadvertent direct nerve injury from the injecting needle.

Localised facial anhidrosis is clearly of no clinical significance as a side effect of botulinum toxin injections. These results suggest a further therapeutic use for the toxin, however. Botulinum toxin type A might be useful as a treatment for patients with severe focal hyperhidrosis. The treatment may be of particular help in axillary hyperhidrosis, a socially and emotionally disturbing condition in which the hyperhidrotic area is usually localised to the central part of the axilla where eccrine glands are heavily concentrated producing 70–80% of axillary sweat secretion.[5] If medical treatment proves ineffective or produces unacceptable side effects, surgical excision of the axillary sweat glands is the other current option.[5] In this group of patients, treatment with botulinum toxin type A might be a worthwhile alternative. The dosage and the required interval between injections remain to be determined, as the recovery time for sudomotor terminals is unknown. This study indicates that the effect is likely to persist for at least three months.

KHALAFALLA O BUSHARA
DAVID M PARK
Department of Neurology, Southend Hospital, Essex SS0 0RY, UK

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.* Presynaptic actions of botulinal neurotoxins at vertebrate neuromuscular junctions. *Journl de Physiologie (Paris)* 1990;**84**:152–66.
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.
3 Girlanda P, Vita G, Nicolosi C, Milone S, Messina C. Botulinum toxin therapy: distant effects on neuromuscular transmission and autonomic nervous system. *J Neurol Neurosurg Psychiatry* 1992;**55**:844–5.
4 Vita G, Girlanda P, Puglisi, Marabello L, Messina C. Cardiovascular reflex testing and single fibre electromyography in botulism. *Arch Neurol* 1987;**44**:202–6.
5 Hurley HJ, Shelley WB. A simple surgical approach to the management of axillary hyperhidrosis. *JAMA* 1963;**186**:109–15.

Measures of medial temporal lobe atrophy in Alzheimer's disease

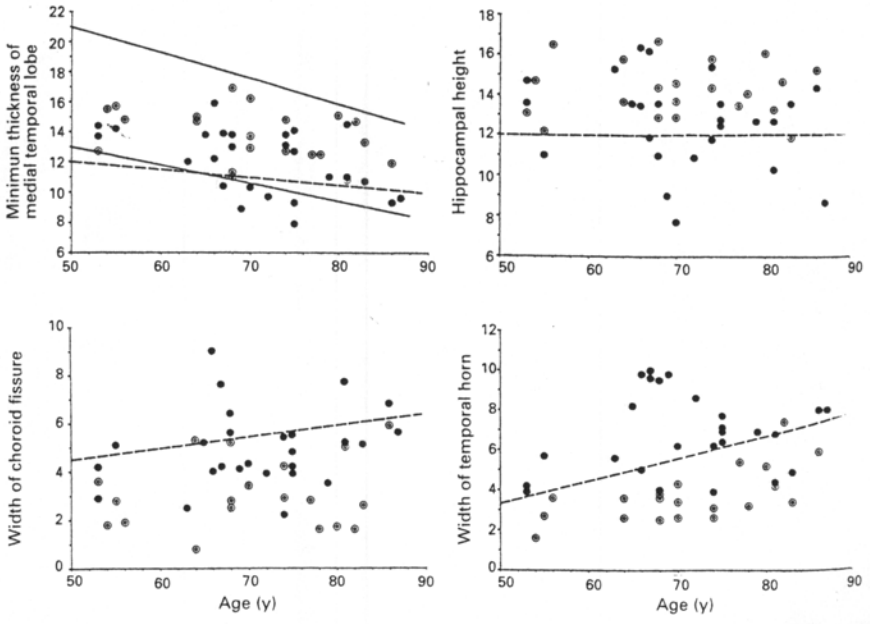
Diagnosis of Alzheimer's disease in life is made on clinical grounds,[1] and currently employed criteria are burdened with considerable subjective judgement.[2] In view of the increasing possibility of treatment, an objective and early stage sensitive indicator of the disease could prove extremely valuable. Jobst *et al*[3] have 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*[4] 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[1] (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.[5] 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.[3] Hippocampal height, width of the choroid fissure, and width of the temporal horn were measured in the coronal plane according to Scheltens *et al*.[4] 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) *v* 13·7 (1·8) mm in controls; $t = 2{\cdot}97$; $p = 0{\cdot}005$ for minimum thickness of the medial temporal lobe, 12·6 (2·2) *v* 14·2 (1·4) mm; $t = 3{\cdot}07$; $p = 0{\cdot}004$ for hippocampal height, 4·9 (1·6) *v* 3·0 (1·4) mm; $t = 4{\cdot}26$; $p < 0{\cdot}0005$ for width of the choroid fissure, 6·8 (2·0) *v* 3·8 (1·4) mm; $t = 6{\cdot}50$; $p < 0{\cdot}0005$ 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.[3] 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*[3] 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



Measures of medial temporal lobe atrophy in relation to age. Full circles: patients with Alzheimer's disease (n = 26); open circles: controls (n = 21). Dotted lines = cut off values for 95% specificity. Solid lines = 5th to 95th centile distribution of minimum thickness of the medial temporal lobe for normal controls according to Jobst, et al.[3]