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Improvement in nerve condition after plasma exchange for Guillain-Barré syndrome.

Sir,—Plasma exchange may produce clinical improvement^{1 2} in cases of acute post-infectious polyneuropathy (Guillain-Barré Syndrome). We have treated a patient with this syndrome in whom improvement in nerve conduction followed a single plasma exchange, and preceded clinically evident improvement.

A 37 year old woman developed paraesthesiae in the hands and feet 10 days after a vaginal infection, treated with amoxycillin and metronidazole. Three days later weakness began in the legs and spread over four days to involve the arms and bulbar muscles. Examination revealed severe weakness of the limbs, facial muscles and neck flexors and distal sensory loss. All the tendon reflexes were absent. The CSF was normal. The left ulnar motor nerve conduction velocity was 42 m/s; the F wave could not be elicited. An hour after one 4 litre plasma exchange the left ulnar motor nerve conduction velocity was 54 m/s. Much of this improvement was due to the appearance of a low amplitude early deflection seen after both proximal and distal nerve stimulation which had not been elicitable before the plasma exchange. In addition, an F-wave was now seen in response to 10% of the stimuli; the proximal conduction velocity, calculated from F-wave latency was 40 m/s. After

the second and third plasma exchange the left ulnar motor nerve conduction velocities were 54 m/s and 55 m/s respectively; the amplitude of the earliest component of the M response increased. F waves were seen after 20% and 25% of stimuli respectively and the proximal conduction velocities calculated from the F wave latencies were 50 m/s and 55 m/s respectively. Clinical improvement began six days after the first plasma exchange. Power in the limbs and respiratory function as measured by peak flow rates improved simultaneously.

The beneficial effects of plasma exchange in some patients with Guillain-Barré syndrome may be due to the removal of complement-dependent myelinotoxic antibodies.^{3 4} The improvement in the motor nerve conduction velocities and, of more importance, the appearance of components of the motor action potential which could not previously be elicited (an initial component of the M response and the F wave) suggest that this humoral immune disorder may lead to reversible conduction block. The return of conduction velocity to normal with plasma exchange may be a useful predictor of clinical response.

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Holmes-Adie Syndrome

SIR,—Abbruzzese *et al*¹ reported electrophysiological data on five patients with the Holmes-Adie syndrome. They were able to elicit a tonic vibratory reflex (TVR) from the soleus muscle in all patients, despite the absence of the Achilles' reflex.

From these findings, Abbruzzese *et al* argued that polysynaptic pathways are normal in the Holmes-Adie syndrome, but to explain the absence of the tendon reflex they postulated the existence of a lesion impairing the transmission of impulses from Ia presynaptic terminals to the alpha motoneurons.

In three typical cases of Holmes-Adie syndrome we elicited a TVR of the soleus muscle with 100 Hz vibration of the tendon belly, but no Achilles' reflex could be recorded. It is interesting to note that polysynaptic reflexes of flexion (RA II, RA III) of the short head of the biceps femoris muscle,² and a non-nociceptive extension reflex of the soleus muscle,^{3,4} were recorded normally in those patients. As compared with values from normal subjects, no change in latency, shape or amplitude of the flexion and extension reflexes of the lower limb was found. Each reflex was elicited by its specific stimulation.

The presence of TVR in all our patients confirms the findings of Abbruzzese *et al*; the normal behaviour of other polysynaptic reflexes in the lower limb can be considered as an additional proof of the hypothesis that polysynaptic pathways behave normally in the Holmes-Adie syndrome.

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