Letters

Dopamine Receptor Enhancement

SIR.—The report of Dr Godwin-Austen and his colleagues on a phosphodiesterase to enhance the activity of one type of dopamine receptor is of intrinsic interest. It also raises a general problem which the increasingly precise possibilities of neuropharmacological analysis of cerebral function is uncovering. Is a measurable reduction in a given cerebral enzyme cause or effect of an observed cerebral dysfunction? Clearly if the receptor cell is dead or severely damaged an effector enzyme cannot enhance its activity. Moreover the decreased cell population may itself simply be reflected in a reduced enzyme presence. The point has been pursued by Davison and his colleagues at the National Hospital Queen Sq, amongst others, in Alzheimer’s disease over the reduction of choline acetyl transferase. Anomalies of response to replacement therapy in other neurological conditions could clearly have a similar basis. While it is desirable to try the effect of increasing a reduced enzyme, if this can be done without disadvantage to the patient, it is also going to be increasingly important to try and assess the quantity of target neurones still available for activation. This is but one aspect of the substantial re-writing of the mechanisms of cerebral function that neuropharmacology is providing.

Reference


CWM WHITTY

Cranial herpes zoster: a case report and a hypothesis

SIR.—We wish to describe a patient with so-called geniculate herpes zoster. Vesicles seen on the anterior tongue and the posterior palate suggested the possibility that transaxonal spread had occurred within the palatine and lingual nerves.

A 35 year old woman first noticed impairment of taste, numbness, and dyseaesthesia of the anterior left side of her tongue, and over the next four days developed a complete left facial palsy. Two days later herpetic vesicles were seen in the “geniculate” zone of the external ear. In addition, there were vesicles on the tip of the tongue on the left with extensive ulceration of the left posterior palate. The uvula and tonsils and the posterior tongue were spared, as was the anterior palate. Additional features were right-beating nystagmus, vertigo, and hyperacusis and reduced lacrimation on the left.

In this patient herpes zoster infection involved gustatory, lacrimal and somatic motor fibres of the seventh, and the vestibular division of the eighth cranial nerve. These structures are associated in the region of the geniculate ganglion. Eruptions in the external ear were confined to the geniculate zone proposed by Hunt. The vesicles observed on the tongue and palate are less easily explained. Vesiculation of both these areas has been reported in the Ramsay Hunt syndrome, either with or without impairment of taste. Reduced taste sensation on the tip of the tongue is a common feature of the syndrome, but is not usually accompanied by vesiculation. There is evidence for a somatic component in the chorda tympani. A similar anomalous relay of somatic fibres from the posterior palate through the greater petrosal nerve is conceivable but has not been reported. We propose an alternative hypothesis. An inflammatory neuritis of the seventh nerve may be seen in geniculate herpes and it seems probable that close to the site of primary infection of sensory nerves neighbouring nerves may become involved by direct extension. The clinical features of the present patient suggest a severe infection with progressive involvement of contiguous neurones of the seventh and eighth nerves. We propose that similar lateral transfer of infection could have occurred at more distal sites. In the palatine and lingual nerves the somatic sensory fibres distributing to the posterior palate and the anterior tongue from the trigeminal ganglion are in contact with taste fibres serving these areas from the geniculate ganglion (fig). Following primary involvement of taste fibres from the geniculate ganglion, somatic fibres of the trigeminal nerve could have become secondarily infected. A similar process of inter-fibre spread could occur in primary trigeminal herpes, so that the anatomy and relations of the branches of the fifth nerve could be a determinant of many apparently unrelated clinical appearances.

References

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3 Costen JB. The transmission of pain

Figure Diagram of somatic sensory (-----) and special sensory (-----) nerves related to observed clinical features. Shading (-----) indicates areas of vesiculation.


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

Sir,—Plasma exchange may produce clinical improvement 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 may be due to the removal of complement-dependent myelinotoxic antibodies. 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.

References


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Holmes-Adie Syndrome

Sir,—Abbruzzese *et al* 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 LA 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, and a non-nociceptive extension reflex of the soleus muscle, 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.

References


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