Letters

Dopamine Receptor Enhancement

SIR.—The report of Dr Godwin-Austen and his colleagues¹ 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

- 1 Godwin-Austen RB, Twomey JA, Hanks G, Higgins J. Oxyphenylamine in Parkinson's Disease. *J Neurol Neurosurg Psychiatry* 1980; **43**: 360-4.

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

dysaesthesia 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.¹ 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, lacrimotor 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.¹ 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.¹ Reduced taste sensation on the tip of the tongue is a common feature of the syndrome, but is not usually accompanied by vesiculation.² There is evidence for a somatic component in the chorda tympani.³ 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 neuronitis of the

seventh nerve may be seen in geniculate herpes¹ 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

- 1 Hunt JR. The symptom-complex of the acute posterior poliomyelitis of the geniculate, auditory, glossopharyngeal and pneumogastric ganglia. *Arch Internal Medicine* 1910; **5**:631-75.
- 2 Esslen E. *The Acute Facial Palsies*. Berlin: Springer Verlag, 1977:92-3.
- 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.