Whatever the explanation, the facts are clear. Nasal vaccination can induce immunity to respiratory infection, but more vaccine is required, and it must be given more often, than by intramuscular injection. This is certainly of theoretical interest, and efforts may still be made to induce immunity in this way in special cases—for instance, when parenteral injection would be harmful, as in the vaccination of infants with respiratory syncytial virus. It is more likely to stimulate a further search for acceptable living intranasal vaccines and improved parenteral ones.

## Trigeminal Sensory Neuropathy

In 1935 Wilfred Harris[1] described a disorder characterized by transient disturbance of sensation in the distribution of one or more divisions of the fifth cranial nerve, and he pointed out that in some cases the resolution of sensory loss was followed by the development of trigeminal neuralgia. T. R. Hill[2] noted two similar cases but with the motor division of the affected trigeminal nerve also involved and an associated Horner's syndrome on the same side. B. Hughes[3] reported a further three cases of trigeminal neuropathy in which the onset of facial sensory loss was heralded by pain of a similar distribution. Then J. D. Spillane and C. E. C. Wells[4] described a series of 16 cases of isolated trigeminal neuropathy. In one of these patients sensation gradually recovered over a few weeks or months, but in others numbness and objective sensory loss persisted, though no other signs of neurological disease developed.

Now J. N. Blau, M. Harris, and S. Kennett[5] report a series of ten patients each of whom complained of painless numbness on one side of the face. The sensory disturbance was usually noticed on awaking or while shaving or applying lipstick, and many patients described the sensation as being like that resulting from dental anaesthesia. The symptoms were confined to the territory of the second division of the trigeminal nerve in one case, to the third division in three cases, and both the second and third simultaneously in five. All three divisions were affected in only one patient. In no case was the motor division of the nerve involved, and the corneal reflex was invariably retained. Three patients with disturbance of the third division noted impairment of taste on the affected side of the tongue. On examination there was blunting of the appreciation of both pinprick and light touch in the affected area. In no case was there any other evidence of neurological abnormality.

Five of the ten patients recovered completely over periods varying from a few weeks to a few months, but in the remaining five the symptoms persisted over many months of observation. Radiological studies of the sinuses and base of skull were normal in all cases and serological tests for syphilis were negative, while the erythrocyte sedimentation rate was also normal. The postnasal space was examined with negative results in seven cases, and in two patients the cerebrospinal fluid was examined and was found to be normal in all respects.

The cause of this syndrome is obscure. In each of the three cases with a painful onset described by Hughes[3] sensory impairment was inexorably progressive, and arachnoiditis round the trigeminal sensory root was found at operation. While it is certainly possible that some patients could have an early neuroma of the Gasserian ganglion,[6] such tumours are rare. M. H. E. Seward,[7] who described four cases, all ending in complete or partial recovery, suggested an analogy between this condition and Bell's palsy. Blau and his colleagues[5] point out that transient paresis of one sixth cranial nerve, giving diplopia on lateral gaze to the affected side, is relatively common. When, as is usually the case, this develops in elderly or atherosclerotic persons, it is usually attributed to atherosclerosis.

D. L. Knox and colleagues[8] have recently described ten children under the age of 15 each of whom suffered a transient sixth-nerve palsy of three to six weeks' duration. They postulated a virus as the cause of this syndrome. To Blau and his colleagues this is an attractive hypothesis, and they raise the possibility that the trigeminal neuropathy which they describe, as well as the more common transient palsies of the abducens and facial nerves, could possibly have a similar aetiology. But no evidence to support this hypothesis is available.

In this context a remarkable case, also reported by Wilfred Harris,[9] is of some importance. He described the case of a woman aged 47 who developed complete right-sided facial sensory loss with loss of taste on the affected side at the age of 21. These sensory defects cleared up after one year, to be followed three years later by typical paroxysmal trigeminal neuralgia on the same side of the face and 14 years later by other symptoms and signs of multiple sclerosis. Though this case is probably unique,[10] it is well to remember that transient trigeminal sensory loss may sometimes be due to a demyelinating lesion.

Despite the unknown aetiology of this interesting syndrome Blau and his colleagues[5] give some useful practical advice on its investigation. They suggest that, if one or more sensory divisions of the fifth cranial nerve are affected and the corneal reflex is preserved, the postnasal space should be carefully examined, the erythrocyte sedimentation rate should be performed, syphilis should be excluded serologically, and radiographs of the paranasal sinuses and skull base should be taken to exclude other causes of trigeminal nerve disease. While the prognosis is in general good, particularly when the sensory disturbance is confined to the third division of the trigeminal nerve, if the motor division is also involved full neurological investigation in hospital with contrast radiography is indicated. Only in a small proportion of cases of trigeminal sensory neuropathy are other neurological symptoms to be expected, but long-term follow-up of these cases is likely to throw new light on the aetiology of the syndrome.


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