BELL'S PALSY AND HERPES ZOSTER

BY

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Clinical evidence has long led to the belief that the commonest aetiological agent in Bell's palsy is exposure to a cold draught. But its mode of action is obscure, and whether cold alone is adequate or whether it exerts its effect upon an already susceptible nerve has been the subject of much debate. Were cold *per se* the deciding factor one would expect a greater incidence, both total and seasonal. Seasonal figures, however, show no great variation, although it is a fact that one new case is commonly followed by several others at certain seasons of the year (Walshe, 1940). Instances of bilateral, recurrent, hereditary, or even familial peripheral facial paralysis seem to support the idea of a predisposing factor (anatomical or otherwise). On the other hand, the facial nerve seems to be just as susceptible to toxic and infective conditions as to the common draught. Such conditions may be general—for example, polyneuritis—or local, such as tetanus.

When Ramsay Hunt (1907) described herpetic involvement of the geniculate ganglion of the facial nerve he focused attention on the virus of herpes zoster as a possible cause of Bell's palsy. In the syndrome which now bears his name we find the combination of peripheral facial paralysis and a herpetic eruption in and around the external auditory meatus of the same side. Vesicles may also be present on the pillars of the fauces and the anterior two-thirds of the tongue on that side. The subject usually complains of pain in the ear, herpetic vesicles make their appearance, and facial paralysis follows in a few days. This sequence is not constant, and the condition may be ushered in with a painless facial paralysis. The small size and the transient nature of many vesicles, and their frequently hidden position in the meatus, make geniculate zoster a condition easily missed. The eruption may precede the paralysis by a week or more, and many cases must then be labelled Bell's palsy. The obvious question that arises is whether Bell's palsy is ever due to invasion of the geniculate ganglion by the zoster virus. It is not difficult to imagine geniculate zoster without the cutaneous eruption. Aitken and Brain (1933) were able to demonstrate by complement-fixation tests that in the blood serum of patients with geniculate zoster there were antibodies to the zoster virus. The sera of twenty-two cases of Bell's palsy were found to contain zoster antibodies in only four instances. Serological evidence therefore points to zoster infection as a probable though uncommon cause of Bell's palsy. It is thus of interest to record a recent clinical experience which supports this view.

In March, 1940, a number of cases of herpes zoster were encountered. They occurred in crops of two, three, or four at a time, presented no unusual features (with one exception), and ceased to appear at the end of the month. In all there were perhaps twenty cases; probably others occurred but were not seen. That these cases represented a small epidemic is supported by their rarity during other months of that year. Many men were but mildly affected and did not take to bed. A few, however, showed general disturbance, with a profuse painful eruption, and were admitted to hospital. In one ward, at one time, there were five such cases.

Case Reports

Case 1. Herpes Zoster involving Th. 9 Segment on Left Side.—The illness was ushered in with headache, malaise, and

a bout of sickness four days before admission. Burning pricking pain in the skin of the affected segment followed, and a typical herpetiform rash subsequently developed. During the acute stage there was pyrexia of 101° , but this subsided to normal in three days. There was no glandular enlargement: a white cell count was normal (8,700 cells per c.mm.), but there was a slight lymphocytic reaction in the cerebrospinal fluid (15 lymphocytes per c.mm.); the protein content was normal (25 mg. per 100 c.cm.). Pituitrin injections were ineffective. Recovery was uneventful.

Case 2. Herpes Zoster affecting Th. 7 and 8 Segments on Left Side.—Anorexia, lassitude, and pyrexia (100°) preceded the onset of neuralgia and the appearance of the rash by three or four days. The latter was profuse, and much pain was experienced for a time. Lumbar puncture was not done. There were no enlarged glands. Pituitrin was without effect.

Case 3. Herpes Zoster involving L. 1 and 2 Segments on Right Side.—In this case the rash was rather late in appearing. The patient experienced severe pains and dysaesthesiae for a week before its arrival. A diagnosis was made of meralgia paraesthetica before the vesicles were detected. There was a curious frequency of micturition, with pyrexia (99°), shivering attacks, and discomfort over the bladder. The urine was normal on several examinations, however, and cultures were negative. The remote possibility of vesical herpes zoster was considered. At first the rash was practically confined to the first lumbar segment, but later it spread to involve the adjacent area below. A normal cerebrospinal fluid was found and there were a few painless glands in the inguinal regions. Pituitrin was not beneficial.

Case 4. Herpes Zoster involving Th. 6 Segment on Left Side.—This patient, who came from the same unit as Case 5, developed pain, pyrexia (99°), and a rash one day before admission. The vesicles were sparse, but there was much neuralgia and cutaneous hyperaesthesia. Three days after the rash had appeared it would have been impossible to recognize the true nature of the eruption, which could well have been missed and the condition labelled "intercostal neuralgia." Lumbar puncture revealed a normal fluid. Pituitrin apparently reduced the pain and discomfort.

Case 5. Herpes Zoster of the Trigeminal Nerve with Ipsilateral Facial Paralysis.—This patient, aged 24, was admitted to hospital with a history of severe pain in the left side of the face for three or four days. A profuse herpetiform rash developed during the night before admission. Within twenty-four hours numerous large coalescing blisters were clustered over the lower half of the left side of the face. There was much redness, swelling, and oedema of the tissues, with a brisk general reaction. The temperature remained at 100° for forty-eight hours and malaise and anorexia were marked. The vesicles were distributed over the areas supplied by the maxillary and mandibular divisions of the trigeminal nerve: a few were clustered together on the lower eyelid, and others on the chin just crossed the midline. There were no vesicles or other form of eruption in the auricular zone. The tympanic membrane, external auditory canal and meatus, concha, tragus, antitragus, helix and fossa, and posterior surface of the lobe of the ear were entirely normal. There was no earache throughout the course of the illness. In the area of the ophthalmic division no eruption appeared, but tactile and painful sensations were diminished. This was especially marked in the supra-orbital and nasociliary areas. Owing to the profuse rash it was impossible to be certain whether there was any sensory change over the areas subserved by the second and third divisions of the fifth nerve. Later in the illness tactile and painful sensations were diminished. The corneal reflex was almost absent, but a feeble conjunctival response persisted. The mucous membranes of the cheek, lips, gums, tongue, and fauces were normal. The sensation of taste was normal and there was no weakness of masseter or temporal muscles.

On the morning of the fifth day in hospital, when pain had subsided and the rash was drying up, the patient awoke with a complete peripheral facial paralysis on the left side. There had been no earache and the ear was normal. The mucous membranes of the mouth were still healthy. The sense of taste, however, was almost completely absent over the anterior half of the tongue on the left side. The patient was unaware

of this hemiageusia. Blunting of sensation over the left side of the forehead and cheek was still present. He remained in hospital for several weeks and made a complete recovery. The sensory changes persisted for a few more days: the hemiageusia disappeared between the nineteenth and twenty-third days of the illness, while the facial paralysis had practically gone three weeks after its appearance.

Case 6.—While the above five cases of herpes zoster were in hospital I saw a case of Bell's palsy in the out-patient department. It was a typical peripheral facial paralysis in all respects and there had been no pain or rash or general reaction. The man made a good recovery within a month. He was employed in the same unit as the first patient (Case 1), and I learned that there had been two or three other instances of herpetic eruption there. Cases 2, 3, and 4 came from separate units, but their quarters were adjacent. I saw several other cases of herpes zoster from the same unit as Case 5. The observation of Case 6 during the height of the epidemic becomes significant when it is stated that only two instances of Bell's palsy have been seen at this hospital during the past twelve months. The second occurred in September, 1940.

Commentary

The first interesting feature of these cases was the development in one of them, on the eighth day, of an ipsilateral facial paralysis. The herpetic eruption was confined to the second and third divisions of the trigeminal nerve, but there was diminished sensation to touch and pain over the first division. *No auricular zoster developed.* Undoubtedly, then, the herpes virus had invaded both the Gasserian and geniculate ganglia. The former resulted in the facial eruption and the latter in the facial paralysis. Whether these manifestations appeared in order of the involvement of these structures can only be surmised. We do not know what interval elapses between the time of infection of the ganglion cells and the appearance of the rash, but it is quite possible the virus attacked the geniculate from the Gasserian ganglion by way of the superior superficial petrosal nerve. It was not until the infection reached the cell stations in the geniculate ganglion of the fibres of the chorda tympani nerve that taste was affected.

The absence of auricular herpes is especially interesting. It indicates that herpetic invasion of the geniculate ganglion can take place without any cutaneous manifestation in the distribution of its sensory fibres. Facial palsy may be the only sign. Hemiageusia would probably be a more frequent sign in facial paralysis due to geniculate ganglionitis than in that due to a lesion arising at the stylomastoid foramen.

Another interesting feature was the isolated occurrence in the same unit as one of the herpes zoster patients of a typical Bell's palsy; this suggests a common aetiological agent. The possibility of coincidence cannot, of course, be denied, but it remains that this case of Bell's palsy was the only one observed at the hospital for a period of nine months. When considered in the light of Case 5—in which zoster and facial paralysis were present simultaneously—the suggestion of a virus origin of Bell's palsy becomes highly probable.

Such findings suggest the possibility of analogous lesions in other sensory nerves. The abortive nature of the rash in Case 4 brought to mind the condition of intercostal neuralgia, and the lumbar neuritis in Case 3 suggested a diagnosis of meralgia paraesthetica during the week preceding the rash. Some cases of facial neuralgia may be due to herpetic Gasserian ganglionitis.

Summary

In a small outbreak of herpes zoster one case of combined trigeminal and geniculate zoster was encountered. It was characterized by facial herpes and an ipsilateral facial palsy. The former preceded the latter by five days. No auricular or

faucial herpes developed. Facial paralysis and hemiageusia were the only signs of geniculate involvement. During this zoster epidemic there occurred the only instance of Bell's palsy seen at the hospital for a period of nine months. The patient came from the same unit as one of the zoster cases.

This evidence supports the view that some cases of Bell's palsy are due to herpetic infection of the geniculate ganglion.

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AN OUTBREAK OF TRICHINIASIS IN WOLVERHAMPTON AND DISTRICT

A CLINICAL ACCOUNT OF SEVEN CASES

BY

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Acute trichiniasis is produced by the embryos of *Trichinella spiralis* during their migrations from the human intestine to the voluntary muscles, and, though extremely rare in Great Britain, the disease is not uncommon in America and Germany. The life history of the parasite is adequately described in the standard textbooks, and, as is well known to medical practitioners and students, infection in man is acquired by eating raw or undercooked pork. The clinical features of the cases described in this article were distinctly unusual and caused some difficulty in diagnosis. Nephritis, pneumonia, renal damage, influenza, pyrexia of unknown origin, suspected enteric fever, and pyelitis were the conclusions arrived at before admission of the patients to New Cross Hospital.

The predominating signs and symptoms consisted of oedema of the eyelids, headaches, conjunctival haemorrhage or injection, mental torpor with lethargy, drowsiness, confusion, and slowed cerebration, heaviness in the limbs, generalized aches and pains, muscular tenderness and stiffness, disturbances of reflex action, paresis, and severe constipation. Vomiting occurred at the outset in two cases and colic in one. My colleagues and I were aware that we were confronted with a distinct clinical entity which was hitherto unknown to us. Blood urea investigations were carried out in the early cases and yielded results which fell well within normal limits. Examination of the blood was next decided upon, and the presence of eosinophilia was discovered. The patients were then seen in consultation with Dr. J. H. Sheldon, consulting physician to New Cross Hospital, who put forward a tentative diagnosis of trichiniasis.* Clinically this disease is generally described in three stages in accordance with the life history of the parasite. Carmichael Low and Hamilton Fairley (1933) state that for the first week during the invasion period gastro-intestinal symptoms develop, with nausea, vomiting, colic, and diarrhoea with perhaps blood and mucus; then when migration of larvae begins myositis of the tongue, laryngeal, and intercostal muscles, and the diaphragm occurs, giving rise to difficulty of swallowing, speech, and respiration. The muscles of the jaws, arms, legs, and abdomen may also be involved, with stiffness and pain; the affected areas are extremely tender and hard to the touch. Oedema, especially of the face, urticaria, leucocytosis with high eosinophilia, prolonged remittent fever (102° to 104° F.), and sweating are said to be characteristic. Cachexia and subcutaneous oedema

* A preliminary account of the present epidemic is given by Dr. Sheldon in the current issue of the *Lancet* (February 15).