PARALYSIS OF THE FACIAL NERVE WITH HERPES ZOSTER.

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THE facial nerve becomes paralysed more frequently than any other single nerve in the body, and yet the cause of this paralysis is in most cases unknown. In a certain small percentage it is due to inflammation of the wall of its canal in the bone, following on disease in the middle ear; or to trauma, operative or otherwise. Rarely it is caused by a central lesion, or is part of a polyneuritis, being then often bilateral. Again, it may accompany or follow a herpes of the face or neck, and is then generally supposed to be due to the inflammation of zona affecting the geniculate ganglion. But in the majority of cases no definite cause can be found; and, in default of a better explanation of the onset of this paralysis, exposure to cold is blamed. In view of the frequency with which this factor occurs in everyday life, it must always be considered unsatisfactory except as a merely contributory cause. From the nature of the case, material for pathological examination can be obtained but rarely. Inquiry must be made chiefly by clinical means.

It is the object of this article to show that herpes zoster affecting the geniculate ganglion is a much more frequent cause of facial paralysis than is commonly supposed, and that many if not all the cases where exposure to cold is blamed are due in reality to that micro-organism, at present unknown, which, attacking the posterior root ganglia of the spinal nerves, and the homologous ganglia of the cranial nerves, produces the interesting train of symptoms called herpes zoster.

Doucet (*Thèse de Paris*, 1936) collected 26 cases of herpes associated with facial paralysis. Analysis of these cases shows that the eruption appeared in the following situations: face, 27 per cent., neck 19 per cent., auricle 4 per cent., tongue and palate 19 per cent., face and neck 27 per cent., face, neck, and mouth 4 per cent.

Hearing was affected in 4 cases out of the 26—twice when the eruption was on the face only, once when on face and neck, once when on neck alone. Taste was diminished in 2 cases—associated once with herpes of the face and neck, once with herpes of face only. In 3 cases paralysis of one side of palate was noticed—once each with herpes of face, neck, and mouth. Three cases of herpes of the auricle are included, but in none of them was hearing affected.

No single one of these cases collected by Doucet showed all the symptoms which may be present in a case of the complete "syndrome of the geniculate ganglion." These are—as described by Ramsay Hunt in the following year—herpes auricularis with facial palsy and affection of the auditory nerve.

It is evident from the consideration of these cases, and indeed from everyday clinical experience, that the symptoms which have been mentioned—herpes zoster of face, neck, ear, or mouth, paralysis of facial nerve, auditory nerve affection, diminution of taste—may occur grouped together in almost every possible combination. Facial paralysis may be associated with one or all of these other symptoms; no one of them is essential, neither herpes of ear, herpes of face or neck, nor affection of the eighth nerve.

If facial paralysis associated with herpes zoster is due to the inflammation of zona affecting the geniculate ganglion, those fibres whose affection causes the appearance upon the ear of the characteristic herpetic vesicles present in the complete syndrome of the geniculate ganglion often, indeed usually, escape. Thus in only 3 of the 26 cases collected by Doucet was the auricle affected. If this is so when the Gasserian ganglion is attacked together with one or other of its homologous ganglia, the Gasserian or posterior root ganglia of the second and third cervical nerves, why should it not be so when it occurs as an isolated lesion—that is, why should not facial paralysis be the sole symptom of an inflammation of the geniculate ganglion caused by the same virus which produces typical herpes zoster elsewhere?

Such an explanation fits in with the course and symptoms of a case of "idiopathic" facial paralysis. As in herpes zoster, there is often preliminary malaise; in children sometimes fever. Commonly, too, there is prodromal pain. In herpes zoster an enlargement and tenderness of the lymphatic glands draining the area of skin on which the eruption later

appears, but preceding that eruption, are almost invariable. I have not found such enlargement reported as occurring in facial paralysis, but it was present in a recent case of mine. The history of this case is as follows:

The patient, a male aged 21, previously healthy, on October 24th began to have pains behind the right ear and down the neck posteriorly on right side. He did not feel well; his throat was sore. On October 29th he felt a little tender lump in front of ear. October 30th: He woke up to find that his face was one-sided. When examined the same day he said he felt out of sorts; his ear felt "dull" and his tongue rough; he felt giddy. There was complete right facial palsy of the lower neurone type. One tender lymphatic gland in front of tragus of ear and one beneath lobule. No other glands palpable. Skin normal; auditory meatus and membrana tympani normal. Fauces slightly reddened, but no vesicles seen. No deafness to whisper or to acoustometer, but a low-toned tuning-fork was heard longer on affected side than on right. Taste normal; no nystagmus or Rombergism.

During the next few days his general condition returned to normal, and auditory and sensory symptoms disappeared. Facial paralysis persisted, showing only slight improvement after two months. The affections of taste and hearing in this case were so slight that a less observant patient might not have remarked on them.

It seems probable that in many cases of apparently uncomplicated facial paralysis there will on inquiry be found some slight symptoms pointing to an affection of the geniculate ganglion, and leading one to suppose that the paralysis is a neuritis due not to exposure to cold but rather to inflammation in the geniculate ganglion, the facial fibres suffering severely, while those which carry the sensation of taste—the pars intermedia—and those of the adjacent eighth nerve have almost, if not completely, escaped.

DYSTOCIA DUE TO GIGANTIC FEMALE FOETUS.

BY

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A WOMAN, aged 22, was admitted to the Louise Margaret Hospital, Aldershot, on March 22nd, 1922, with oedema of the legs and face, and a'buminuria. Her periods had been regular up to the last one, which began on July 25th and ended July 30th, 1921. Quickening was felt about the first week in December. She had had one normal confinement two years before, when a healthy female child weighing 10 lb. was born. The mother and father are both six feet in height.

On admission the patient had marked oedema of face, lower abdomen, vulva, and legs. The abdominal enlargement was much greater than usual, and suggested multiple pregnancy and hydramnios. On palpation the uterus was so tense that the only foetal part distinguishable was the vertex, and even this was difficult to define. Only one foetal heart was heard, on the right side, between the umbilicus and anterior superior spine. The fundus reached to the ensiform cartilage. A tentative diagnosis of hydramnios and multiple pregnancy was made.

The pelvic measurements were as follows: Intercrestal 12 in., interspinous 10½ in., external conjugate 8½ in., inter-tuber ischii 4½ in. By vaginal examination the promontory could not be reached. The blood pressure was 160 mm. of mercury on admission, but after three days it fell to 130 mm.

The urine contained a heavy cloud of albumin, but no pus cells or renal casts; ammonia nitrogen 0.23 gram, urea nitrogen 0.7 gram, a slight trace of acetone and diacetic acid. On culture it yielded a coliform organism.

The urinary condition and oedema cleared up satisfactorily under treatment with vapour baths, saline aperients, and a course of potassium citrate, followed by urotropine and acid sodium phosphate (given separately), combined with light diet.

The patient was kept under observation in hospital, and on April 26th, at 8.15 p.m., the membranes ruptured. Pains began at 9 p.m. The vertex was felt presenting in the R.O.A. position. Pains continued regularly, but not very strong, and at 3 a.m. on April 27th the os was fully dilated. After one and a half hours in the second stage, as there was no advance of the head, which was engaged in the brim, and as the foetal heart sounds were still heard, axis traction forceps were applied under anaesthesia, and the head brought to the outlet after considerable difficulty. The delivery of the head through the outlet between the pains was very difficult owing to its large size. A loop of cord feebly pulsating could be felt above the head.

Obstruction to the further birth of the child occurred at this stage owing to the shoulders being wedged across the brim of the pelvis in the right oblique diameter. Attempts at digital traction with the finger in the posterior axilla failed. A blunt hook was therefore used, with the result that the shoulders were brought sufficiently low down for the arms to be disengaged and delivered alternately. The child was stillborn. The perineum was ruptured, but the tear did not extend through the sphincter ani. The