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

placenta followed forty minutes later and appeared perfectly healthy, but weighed 4 lb.

The puerperium was uneventful. The patient was sitting up and well on the twelfth day.

Particulars of the Child.

Weight (verified by separate weighing machines, and observers) ...	24 lb. 2 oz.
Length—vertex to heel ...	35 inches.
Length of spine ...	15½ "
Bis-acromial ...	8½ "
Length of arm ...	13 "
Length of foot ...	5½ "
Circumference of thigh ...	10 "
Circumference of forearm ...	7 "
Bi-trochanteric ...	6 "
Head circumference ...	19 "
Bi-mastoid ...	4½ "
Bi-ma'ar ...	4½ "
Suboccipito-bregmatic ...	5½ "
Suboccipito-frontal ...	6½ "

The *post-mortem* examination was confined to examination of the brain. The bones of the vertex were harder and exhibited a greater degree of ossification than usual. The anterior fontanelle was smaller than usual. On opening the skull the dura was firmly adherent to the calvarium, and the brain was so soft and diffident that it could not be removed intact; it was put into formalin solution, but after twenty-four hours had fallen to pieces. A portion of the pituitary was saved and examined, but merely exhibited undue vascularity of the pars anterior.

The foetus is preserved in the Museum of the Royal Army Medical College, Millbank, London.

Induction of labour or Caesarean section would have been considered had it been possible to diagnose a foetus of such large size. Pelvimetry had shown that the maternal passages were abnormally large. The difficulty was that the uterus was so tense with excessive liquor amnii and the large foetus that its contents could not be palpated clearly. It was not reasonable to anticipate trouble with a pelvis of this size, especially as we had in mind the probability of hydramnios and twins, and knew that a 10 lb. baby had been born without trouble previously.

Cleidotomy was not performed because at the stage when it suggested itself as a means of overcoming the difficulty with the shoulders it was still hoped to get a living child.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

A CASE OF CONGENITAL PYLORIC STENOSIS.

THE occurrence of congenital pyloric stenosis in female children is, I believe, of sufficient rarity to be worthy of note.

A colleague called me into consultation upon a marasmic female infant aged 3 weeks. It was a second child, the first being a healthy boy, and labour had been normal. The mother appeared perfectly healthy and was feeding the child herself.

The child commenced to vomit its feeds when about 10 days old; at first this was so slight that it was considered to be a mere regurgitation caused by slight digestive disturbance, as frequently happens. During the next four days the vomiting increased and the child began to waste rapidly. Various dietetic changes were tried, but without success. In the third week the vomiting became really projectile in type, even the smallest feeds of albumin water being returned. These were the conditions when I was called in; in addition there were scattered râles over the bases of both lungs.

A feed was given to the infant in our presence, and within a minute well-marked slow peristaltic waves could be seen passing across the abdomen from left to right in the region of the stomach. This peristalsis was apparently painless. Shortly afterwards the vomiting began and was projectile in type. A thickening could be felt in the region of the pylorus. The stools up to this time had been normal, showing no signs of failure of digestion, but the apparent constipation now present was another point in the diagnosis.

Given these symptoms—namely, projectile vomiting, visible peristalsis in the gastric region, palpable thickening in the region of the pylorus, apparent constipation, and marked wasting—a diagnosis of congenital pyloric stenosis was made. I am indebted to Dr. Frederic Still for the following notes giving details of the treatment:

"The operation was performed by Mr. Tyrrell Gray, after repeated subcutaneous saline infusions had produced a slight rally. I was watching for the moment when it might be just possible to operate, for the child was in an almost desperate condition when I first saw it about eighteen hours before operation. It was not only extremely feeble and somewhat blue, but there were sharp râles all over the bases of both lungs.

"We took advantage of the temporary rally, and Mr. Gray did the Ramstedt operation—that is, a simple longitudinal incision through the muscular layer of the pylorus without cutting through

the mucosa. The child stood the operation well, and with further subcutaneous infusions rapidly gained strength, and has since kept down its breast-milk feeds well."

The infant has progressed satisfactorily, and has gained 1 lb. 2 oz. since the operation three weeks ago. This gain is perhaps less than might be expected, but is to be attributed chiefly to the deficiency of breast milk after the temporary weaning.

According to statistics, the condition is rare in female children, 80 per cent. of cases occurring in boys (Garrod, Batley, and Thursfield, *Diseases of Children*), and more commonly in first children.

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AUTOGENOUS VACCINE IN PUERPERAL FEVER.

I wish to bring before country practitioners the efficacy and great assistance of autogenous vaccines in the case of puerperal fever. Lately I had a case as follows:

The birth was by means of forceps delivery with small tear in the perineum. This was stitched. Four days later, after having been given castor oil, the patient's temperature rose and the lochia ceased. Immediately intrauterine douching (twice daily) was commenced and salol and hexamine were administered every four hours. This treatment went on for five days with little or no success. The pulse ran up to 120–130. The temperature was 105° in the morning and 101° in the evening on an average. On the sixth day 20 c.cm. of a well-known make of stock polyvalent anti-streptococcus serum was given, with no result. On the next day 10 c.cm., similarly with no result. The heart condition now began to show slight indications of commencing endocarditis.

I examined a loopful of the discharge from the cervix and identified coliform organisms and streptococci. I then sent a loopful of this discharge for the preparation of an autogenous vaccine.

As the action of the heart was becoming weak, heart stimulants were now used, chiefly digitalis, with caution, for fear of embolism, but even in moderately large doses the result was disappointing. Antipyretics were of little use, although small doses of aconite helped a little.

Four days after the dispatch of the specimens (with all due credit to the Royal College of Physicians' laboratory, Edinburgh) I received the autogenous vaccine, each cubic centimetre of which contained 50 million coliform organisms and 50 million streptococci. I gave 1/10 c.cm. immediately, and progressively doubled this dosage every twenty-four hours, till the patient was receiving approximately 1 c.cm. every twenty-four hours. The results were marvellous. The temperature fell as if by magic, and on the sixth day after the commencement of treatment by the autogenous vaccine the endocarditis had cleared up and the temperature was normal. Recovery was now well established. The vaccine was still continued for some days (1 c.cm. every twenty-four hours).

I wish to draw attention to the fact of the absolute failure of the stock serum in this case; in fact, after administration serum manifestations caused the patient's condition to become worse.

Cowdenbeath.

JOHN VEITCH, M.B., Ch.B.

ELECTRICAL TREATMENT IN TUBERCULOUS DISEASE.

IN the BRITISH MEDICAL JOURNAL of June 4th, 1921 (p. 812), I described experiments I had performed for the destruction of bacilli by electricity. Since that date I have treated an obstinate case of tuberculous colitis by galvanism with an excellent result.

The patient, aged 26, was sent to me by his doctor, who suggested that as I had destroyed staphylococci and *B. coli* by an electrical current it might be possible to kill the tubercle bacillus in the same way.

Nearly two years before I saw the patient he had mild diarrhoea and passed blood and mucus. He had previously had an empyema which had been operated upon and the wound healed. The diarrhoea was continuous and did not respond to drugs. Wasting was marked and the temperature high. An autogenous vaccine had been prepared and a course given with no apparent improvement in the symptoms. Later (nine months before I first saw him) appendicectomy was performed and daily the colon was washed out with saline, silver nitrate, and sometimes Dakin's solution, other antiseptic