chronic rheumatism, phthisis, and anæmia with cardiac dilatation. I took several tracings from each case day by day, with uniform results. The characteristics of the "double first sound pulse" were always brought out best by obliterating with the finger applied to the radial pulse on the distal side of the sphygmograph any effect which a recurrent pulse from the arteries of the hand might have on the tracing, but an exaggerated ventricle outflow remainder wave was distinct in all those taken in the ordinary way.

PARALYSIS OF THE SIXTH AND SEVENTH NERVES OCCURRING IN A PATIENT WITH WHOOPING-COUGH

By FREDK. A. CRAIG, M.B.R.U.I.,
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THE case first came under my notice on November 11th, 1895, the patient being a little girl aged 3 years and 4 months. There is no syphilitic or tuberculous history, and the only tendency to nerve disease traceable in the family is in the mother, who periodically complains of headache, probably migrainous. She has given birth to four children. She never had any miscarriages.

Fig. 1.—Showing face at rest.

The patient is the third of the family. When 9 months old she had a slight attack of bronchitis, which soon passed off. Save for this, she has always been healthy. During the second week of September whooping-cough appeared. The paroxysms of cough were not over-frequent during the day, but numerous and severe at night. On one occasion, during a paroxysm of coughing, blood came from the left nostril; this was the only time any visible hæmorrhage occurred.

About the end of the first week in October, the mother noticed the child's left eye squinting, and its face "peculiar," especially when she cried from toothache, due to a few carious teeth.

On November 11th, I was called in; that is, two months after the whooping-cough commenced, and about five weeks after the appearance of the paralysis.

The Facial Paralysis.—The characteristic physiognomy of facial paralysis was well marked. The lower part of the face appeared as if all the furrows were washed out, and it did not

take any part in the movements of the face, even in the movements produced in crying, which deeply furrowed and wrinkled the sound side. The angle of the mouth on the affected (left) side was on a lower level, and somewhat nearer the middle line; this was better marked when the child was sobbing, as the zygomatic muscles of the sound side pulled the mouth towards them. The lower lip and chin also appeared drawn over to the sound side, and the orifice of the mouth oblique. The mother informed me that she put her tongue out nearer the paralysed side of the mouth (she would not protrude her tongue for me). This was explained by the paralysed side of the mouth being drawn over towards the sound side, and the tongue thus appeared to be protruded towards the paralysed side of the mouth.

In the illustrations, in one of which the child was in a comparatively quiet mood (Fig. 1), the deformity produced by the facial paralysis is not great, whereas in the other the child was crying (Fig. 2), and the great contrast between the two sides of the face is well shown. When sleeping, the mother says the contrast between the two sides of the face is not marked. She could not firmly close the paralysed side of the mouth, some saliva more or less constantly dripping from the paralysed angle. Her mother stated that she could not blow straight in front of her, that she could not spit straight in

Fig. 2.—Showing facial paralysis when the child cried.

front of her, and that she chewed all her food with the non-paralysed side of the mouth. The orbicularis palpebrarum and frontalis muscles were very much weakened, though not absolutely paralysed. In ordinary winking the child did not shut the eye completely. In making an effort to shut her eye, she succeeded in closing it. When the eyes were shut forcibly the wrinkles around the paralysed eyelids were not so well marked as those around the non-paralysed eyelids. When she looked upwards the eyebrow on the paralysed side was not raised so high as on the non-paralysed side. Each inspiration during sobbing was accompanied by elevation of the eyebrow on the sound side only. The space between the eyelids on the paralysed side was greater than on the non-paralysed side, as is seen in the illustration. I believe sensation was unimpaired, and that taste, smell, and hearing were unaffected.

Eye Phenomena.—The left external rectus was completely paralysed, so much so, that in the resulting internal

strabismus the inner edge of the cornea was hidden beneath the internal canthus. The eye could not be moved outwards, and attempts to do so were accompanied by slight oscillations of the eyeball, probably due to the action of the oblique muscles. The movements of the sound (or right) eye are in no way impaired. When going about the house the child covered the left eye with its hand to prevent double vision. She had complained once or twice to her mother about seeing two objects instead of one. The father said that during sleep, when the eyelids were a little separated (on the left side), the squint was not so great; this was owing to relaxation of the internal rectus during sleep. The pupils were equal. The optic discs could not possibly be examined.

The heart sounds were normal; the urine was free from albumen and sugar. The lungs exhibited some signs of bronchial catarrh.

On December 1st the whooping-cough had cleared off. The paralysis had in no way improved.

On January 1st, 1896, the child was in perfect health, but the paralyses had not improved.

June 1st, the paralytic symptoms remain unchanged.

The history of the onset of the paralysis in this case is most unsatisfactory, neither mother nor father being able to state when they first observed it. They are not certain whether its onset was sudden or gradual, or whether the paralysis of the facial and sixth occurred at the same time. The father says that the eye squinted more on each day, and that it was a week before it reached its present state. The mother gives a history of a more sudden onset.

This case at first, I must admit, gave me trouble to classify, and I must thank Dr. Gowers for enabling me to put it down (I think unmistakably) as a case of infranuclear paralysis of the facial nerve. The want of absolute paralysis in the orbicularis palpebrarum and frontalis muscles may be explained by the lesion producing the paralysis not having to the same extent implicated the fibres for the orbicularis palpebrarum and frontalis as the fibres for the lower facial muscles. It may also be explained by the well-known fact that in recovery the power of winking and closing the eye is first regained; the fact of a little power being present in these muscles might indicate the first process of recovery, the paralysis having been in existence five weeks before I saw the child. If it be due to the latter, I am sorry to say the process is not advancing.

According to Dr. Gowers's classification, this case may be put down as a case in which the lesion is below the sixth nucleus, or as a case in which the lesion is in the sixth nucleus, but that the structure subserving the inward movement of the sound eye has escaped destruction.

The most prominent part of the eminentia teres in the floor of the fourth ventricle marks the position of the sixth nucleus; the facial nucleus is situated a little below and a little external to the sixth nucleus; the facial fibres curve round, while some actually go through the nucleus of the sixth. The diagram, which is copied from Ross's book, will

explain the close relation of the facial fibres to the sixth

nucleus. A lesion involving the nucleus of the sixth would undoubtedly affect the fibres of the facial.

Moreover, when paralysis of the sixth and facial exists alone, the disease is generally situated in the posterior part of the pons. If the lesion affected the facial nucleus, some writers say that the orbicularis oris would escape, but in this case it is paralysed. If the lesion of the facial were at the base of the brain, the auditory nerve would be implicated with it, but we have no reason to believe that hearing is in any way affected in this case. If the lesion were in the Fallopian canal, taste and hearing would probably be affected. If we admit, as Gowers himself admits, that even in a lesion affecting the sixth nucleus, the structure subserving the inward movement of the opposite eye may escape; if we admit that two lesions are less likely to be the cause than one, then we are driven to the conclusion that the lesion causing the paralysis of the sixth and facial is situated in the sixth nucleus. If we do not admit that the structure subserving the inward movement of the opposite eye could escape in lesions of the sixth nucleus, then we must say that two lesions are present, which is most unlikely. The most common causes which might give rise to this lesion within the pons are: hæmorrhage, embolism, thrombosis, cerebral abscess, tumours (including syphilitic growths), cerebral tuberculosis, and islets of disseminated sclerosis.

Disseminated sclerosis, cerebral tuberculosis, and cerebral abscess may, I think, in this case, be dismissed. The paralysis is not such as usually accompanies a tumour; it is not of slow origin; and it is not at all progressive. Epileptiform convulsions and cerebral vomiting are absent. Dr. Donaldson cannot make out whether optic neuritis (a symptom which is present in about 80 per cent. of the cases of intracranial tumours, at some time or other of their course) is present in this case or not, without chloroforming the child, a procedure which most parents are unwilling to submit to.

As to cerebral thrombosis this patient is a young child and free from syphilitic taint. I admit the possibility of a thrombus forming during the exhaustion produced by a paroxysm of coughing, but I think it is unlikely to be the cause in this case.

Embolism is usually due to mitral or aortic endocarditis, neither of which exists in this case. I have now dismissed all the likely causes of the lesion producing this affection of the facial and sixth nerves, except the commonest and most important, namely, hæmorrhage—hæmorrhages from the various mucous surfaces, such as the nose, the mouth, the pharynx, the larynx, under the conjunctiva, and even from excoriations of the skin are common in whooping-cough during severe paroxysms. And we are not without instances, verified by *post-mortem* examinations, of hæmorrhages into the brain occurring during a severe paroxysm of coughing. Gowers says that hæmorrhages into the substance of the brain, minute and massive, have occurred in young children during the paroxysms of whooping-cough.

The recorded instances, so far as I can trace, have always been accompanied by some signs of hemiplegia of arm or leg. Henoch reports a case of cerebral hæmorrhage producing left hemiplegia in a child 3 years old suffering from whooping-cough, the face escaping. Dr. Samuel West read notes of a case of right hemiplegia with aphasia occurring during a paroxysm of whooping-cough before the Clinical Society of London in 1887.

In the Section of Diseases of Children at the annual meeting of the British Medical Association in 1894 Dr. Hopkinson mentioned a case of cerebral hæmorrhage causing left hemiplegia in whooping-cough. At the same meeting Dr. Mansel Symptom remarked on the curious rarity of cerebral paralysis in whooping-cough. Dr. Barrier records a case of hæmorrhage between the dura mater and arachnoid as a result of a paroxysm.

If this case be due to hæmorrhage it must be small to cause so localised a paralysis; but the absence of other paralysis (such as of leg or arm) cannot be regarded as weighing much against this supposition, for examples of such localised paralysis due to small cerebral hæmorrhages and confirmed by *post-mortem* examinations are not wanting.

If this case be due to cerebral hæmorrhage the squint increasing for a week may be explained by the secondary

inflammation set up around it. It would also be interesting to know whether the hæmorrhage be venous, arterial, or capillary. Gowers says that healthy arteries, no matter how great the internal pressure to which they are subjected, seldom, perhaps never, give way, but that healthy veins may give way under extreme pressure. In this case we have no reason to believe that the arteries are in any way diseased.

The pressure to which arteries are subjected during a prolonged paroxysm of whooping-cough gradually diminishes after the first two or three expiratory efforts as the paroxysm proceeds, and in proportion as the arterial pressure diminishes the venous pressure increases, so that by the end of a severe paroxysm there is very little blood in the arteries, most of the blood being in the veins owing to the violent expiratory efforts retarding the flow of the venous blood in the *venæ cavæ*.

In whooping-cough, which is often accompanied (especially when bronchial symptoms have existed for some time) by enlarged bronchial glands, these still further impede the flow of blood in the large veins from the neck, and help to increase the venous pressure. This venous engorgement is seen during the paroxysm in the veins of the neck, face, and arms; it accounts for the hæmorrhages from the mucous surfaces, it accounts for the puffiness of the face about the eyelids, and when recurring constantly it causes some of the veins to remain dilated as those about the eyelids. From these remarks the low arterial pressure, the fact of a healthy artery seldom, perhaps never, giving way, we may reasonably exclude arterial hæmorrhage as the cause. On the other hand, from the high venous pressure, from the fact that veins have thinner walls than arteries, from the fact that healthy veins may under extreme pressure give way, and from the fact that the hæmorrhages which usually occur in whooping-cough are venous, we may reasonably assume that this hæmorrhage is venous, if it be a hæmorrhage, as I believe it to be.

A CASE OF HERNIA OF THE OVARY IN AN INFANT WITH TORSION OF THE PEDICLE.

By C. B. LOCKWOOD, F.R.C.S.,

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THE case which I am about to communicate is certainly very unusual. Mr. Pollard and others have described strangulation of herniated ovaries, but no record can be discovered of torsion of their pedicles. The details were as follows:

E. E. S. was a fat and healthy baby, 6 months old. On July 27th she refused the breast, was sick, and drew up her legs as if in pain. She was sick again upon July 28th, and upon July 29th her mother noticed for the first time a swelling in her right inguinal region. This swelling was tender but went away after some pressure. On July 30th the child slept quietly until the evening, when the lump returned. It was larger than it had been before, and the skin over it was red. Nothing was done to the swelling and the child was brought to the hospital. In the right inguinal region she had an oval swelling about 3 ins. long and $1\frac{1}{2}$ in. wide. Its upper end was at the internal abdominal ring, its lower end was lost in the right labium majus, which was inflamed and swollen. The skin over the swelling was also red and inflamed. The swelling was tense and had no impulse. Its size did not alter when the child cried, but it gradually increased until the operation.

The local characters of this swelling were those of an inflamed and strangulated inguinal hernia. This idea was supported by a history of constipation since July 28th, and of occasional vomiting, but after admission into the hospital flatus was passed, and a rectal examination was followed by a copious action of the bowels. This suggested that an operation was not urgently needed, but as the swelling did not subside and as the infant was in great pain, I performed an exploratory operation on August 2nd.

Chloroform having been given, an incision along the course of the inguinal canal exposed a bluish-red cyst with walls nearly an eighth of an inch thick. It was distended with about 2 drachms of dark brown, grumous fluid. When the sac was opened an ovoid mass, like a small damson, came

into view, and was thought for a moment to be a strangulated cæcum. But presently the deeply engorged, fimbriated end of the Fallopian tube was found peeping from beneath the lower part of this mass, which was now recognised to be an ovary full of blood. The ovary, Fallopian tube, and broad ligament lay free in the sac. About three-quarters of an inch from the internal abdominal ring their pedicle had made a half turn upon itself. This half turn was the cause of the vascular engorgement of the ovary, Fallopian tube, and broad ligament, because the part of the tube between the half twist and the internal abdominal ring was normal. Moreover, the internal abdominal ring was capacious, and seemed incapable of constricting its contents. When the ovary was pulled out of the sac the half turn disappeared.

The ovary and tube were clearly too much damaged to be returned into the abdomen. Therefore they were removed after the pedicle had been transfixed and tied. The sac was afterwards excised, and the stump fixed beneath the internal oblique and transversalis muscles, the inguinal canal was next sewn up, and the wound closed in the usual way with fishing gut.

The child made an uninterrupted recovery, and on August 10th the dressing was removed, the wound being healed by first intention.

The specimen merely shows the great engorgement of the ovary, Fallopian tube, and broad ligament with blood. The twist cannot be seen because it disappeared when the pedicle was divided. At the operation it was demonstrated to numerous spectators.

It is not at all easy to explain how the twist occurred. In the analogous case of torsion of the spermatic cord there seems little to indicate its causation. But some points seem quite clear. In the other specimens of hernia of the ovary which I have seen the herniated viscera had kept their relation to one another in spite of their displacement. Thus the Fallopian tube remains highest up in the hernial sac, with the ovary in front and below. This is, of course, different from the position of the ovary whilst within the abdomen; there it lies below the Fallopian tube but behind the broad ligament. But a moment's consideration shows that as the broad ligament with its contents turns the corner of the internal abdominal ring to get into the inguinal canal the position of the ovary is reversed. It keeps its position with regard to the Fallopian tube, but becomes anterior instead of posterior. In the present instance the ovary was the highest, having ascended in front of the Fallopian tube. The latter seemed to have fallen behind the ovary so as to be hidden from view except its fimbriated end, which peeped out below.

F T, Fallopian tube; O, ovary; P, pedicle (semidiagrammatic).

There is nothing to show why the ovary had ascended in this way, or how the Fallopian tube had got beneath it. But the end of the broad ligament in which they lay was quite loose within the hernial sac, so that considerable mobility must have been allowed.

A section had been made in the ovary, and shows that although full of blood it contains numerous ova. Under the microscope the ovarian tissues are normal, but the venules are distended with blood, and much blood is extravasated amongst the various structures.

[Since this case was read and published in extract, another of the same kind has been described by Mr. Edmund Owen.¹ In this the right side was also affected, and the pedicle had, I gather, undergone a similar twist with a similar result. Mr. Owen had also to excise the damaged ovary and tube.]

¹ *Lancet*, March 21st, 1896, p. 765.

SUCCESSFUL VACCINATION.—Dr. James Farquhar has received the Local Government Board award for successful vaccination in No. 3 district of the Pewsey Union for the second time.