

sufficient to correct this, although some immediate improvement resulted. With reference to the important question of section of the tendons of the wrist-joint, this example shows that it can be done with impunity, and even with success. Although no efforts were made to bring the severed ends into apposition, yet perfect union took place. One is aware how often it happens that if the tendons be severed by broken glass or any other accident non-union results. In this case the utmost care was taken to keep the wrist and fingers flexed for ten weeks after the second operation, so that by these means the uniting material became firm. Some experiments<sup>2</sup> I made on division of the tendo Achillis of rabbits convinced me of the necessity of great care in avoiding premature stretching of the uniting material of divided tendons. I have known it to continue to stretch for fourteen months after the date of operation. Therefore in this case the wrist was kept slightly flexed for a whole year after the second operation. To the fact, then, that the extensors were prevented from pulling upon the weak uniting material of the flexors at the wrist I ascribe in a large measure the complete restoration of the functions of the hand.

Finsbury-circus, E.C.

## A CASE OF PSEUDO-HYPERTROPHIC PARALYSIS WITH THE KNEE-JERKS PRESERVED.

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THE following case of pseudo-hypertrophic paralysis presents several unusual features and seems of sufficient interest to be placed on record.

A boy aged ten years was brought to me at the Hospital for Sick Children, Great Ormond-street, on Dec. 29th, 1894, with the following history. He had been quite well and strong, and as forward as other children, till he was five years old, when it was noticed that he was "weak on his legs" and that his calves were unduly large. This weakness continued to increase, but very slowly—so slowly that he was active and could run, play football, &c.—until a year before he came under observation, when he had a severe attack of scarlet fever, and after this he became rapidly weaker, and his football performances were confined to keeping goal. The arms were now noticed to be getting weaker, so that he had difficulty in holding the brush when blacking his boots. He was born at the full term and was a perfectly healthy infant; he walked at sixteen months and could talk when two years old. Beyond the attack of scarlet fever mentioned above he never had any illnesses. His head was "funnily shaped" from birth. The family history revealed nothing important. A brother twelve years old had broken his left arm on two occasions and his right leg once, but presented no other signs of disease. On examination the patient was found to be a rather thin but active-looking boy. There was an obvious prominence on the forehead along the line of the frontal suture, and the skull presented a well-marked scaphoid shape. His intelligence and degree of education were fully up to the standard. He walked with an awkward and rather high-stepping gait. When his feet were off the ground they showed a well-marked "pes cavus" deformity, which disappeared when he stood erect. There was commencing slight contracture of the gastrocnemii on both sides. On examination of the muscles there was great enlargement with weakness of the gastrocnemii and of the vasti externi—the crureus and vastus internus being but little affected on each side. The glutei were very little enlarged. The muscles in front of the leg were little affected. An electrical examination showed over-excitability of the gastrocnemii and vasti externi to stimulation by a faradaic current, a current too weak to produce contraction in a healthy child at once causing a contraction in these muscles, and the reaction was greater on the left side, on which the hypertrophy was also most marked. The reaction of the other muscles to the faradaic current was normal. To the constant current all muscles responded normally. In the upper limb there was considerable weakness of movements

about the shoulder. The deltoids and triceps were enlarged, and notably the infra-spinati on each side. There was some wasting of the biceps, pectorales, latissimus dorsi, and of the muscles of the thumb. The muscles all responded readily to both forms of electrical stimulation. Sensation to all forms of stimuli was normal in all parts. The knee-jerks when he was first seen were extremely active, but after he had been under observation six weeks they became much more moderate. Ankle clonus was not elicited at any time. During the last few months he has had slight difficulty in controlling micturition, and had wetted the bed on several occasions. He had retained control over the sphincter ani, but had to obey any calls very promptly, any hot drink producing a rather loose action of the bowels almost immediately. In all other respects he appeared to be quite healthy.

The chief points of interest are the small number of muscles affected although the disease had been recognised for five years, the retention of the knee-jerks, and the electrical reactions. The preservation of the knee-jerks at so late a period of the disease is so unusual a condition that I have been unable to meet with any recorded case. In this case there seem two possible explanations. The first—to which I incline—is that it was owing to the fact that, although the vastus externus was extremely enlarged, no change could be observed in the vastus internus, whose electrical reactions also corresponded to those of unaffected muscles. It has been shown by Professor Sherrington<sup>1</sup> that the vastus internus is the essential muscle for the production of the knee-jerk. The nerves to the other constituents of the quadriceps may be cut in an animal, but if the nerve to the vastus internus is uninjured the knee-jerk will be elicited as before. In the case of my patient I attribute the retention of the jerk to the escape of the vastus internus, and expect that as soon as that muscle becomes invaded the knee-jerk will disappear. The other possible alternative is that there may be some coexistent gross spinal cord disease; but of this not the slightest evidence could be discovered except the very doubtful evidence from the weakness of the sphincter muscles. The curious over-excitability of the enlarged muscles to faradism is a phenomenon that I have observed before in at least one case of pseudo-hypertrophic paralysis, and presumably is due to an abnormal excitability of unaffected fibres in the muscle, and may indicate some change in the muscle fibres immediately preceding the atrophic process.

Wimpole-street, W.

## A CASE OF PULSATING EXOPHTHALMOS.

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THE patient, a married woman aged thirty-six years, saw me on Oct. 23rd, 1894. For five weeks she had had severe "neuralgic pains" in the right side of the head, more marked near the temple. Four weeks ago (one week after the commencement of the pains) she heard a pulsating noise in the right side of the head, accompanied by severe throbbing pains; and she had also noticed that the right eye had become prominent. The pain, noises, and protrusion of the eye were at first very slight and had gradually become worse up to one week before I saw her. During the last week they had remained stationary. When she was excited or bent the head the noises and protrusion became more marked. The patient was a pale, weakly woman and looked fifty years old. The right upper lid drooped, was swollen and tense, with thick veins, and covered the eyeball. The lid could not be lifted up. A thick fold of dark-red conjunctiva protruded between the lids and was larger on the inner side. The lower lid was also swollen. The eyeball protruded forwards and outwards, was divergent, and could not be moved. No pulsation of the eyeball could be seen or felt when the patient was quiet. As soon as the head was bent a slight pulsation was noticed. The eyeball could be pressed back into the orbit to some extent. The conjunctiva bulbi was thick and red and contained many dark veins which converged towards the cornea. On the inner side of the eye there was a soft tumour which seemed to press

<sup>2</sup> Guy's Hospital Reports, 1891.

<sup>1</sup> Journal of Physiology, vol. xiii.