

to a fortnight later he had difficulty in swallowing liquids, which regurgitated through his nose (paralysis of palate). About two weeks later still, that is, about the middle of September, he noticed his legs and then his hands began to grow weak. This developed into the condition present at the time of my examination. He never had syphilis.

The subsequent course of the case may be briefly told. The legs began, during the following weeks, to improve, but the hands to grow worse, the ataxia being replaced by a paretic condition of both sensation and motion, so that, for example, he had great difficulty in buttoning his clothes. Grasp of hands, dynamometer L. 60, R. 70. He persisted, however, in keeping about his work, and as long as he did so his hands failed to improve. Finally he consented to take absolute rest in bed, and as soon as he did so he began to improve, and about the middle of December he was practically well. At that time the knee-jerks were still absent, but a few days ago he wrote me that they were "as good as they ever were," whatever that may mean.

The story of Case II. may be more briefly told. It was a child four and a half years old. Ten weeks previously he had had diphtheria. Two or three weeks after recovery the mother had noticed a nasal character to the voice. Nothing else was noticed until October 7th, when the boy complained of pain in right ankle, and on the next day the condition present at the time of my first examination (October 19) developed suddenly, according to the mother, namely, *paralysis of soft palate, absence of knee-jerks, and great ataxia of legs*. He could walk only with great difficulty, owing to inability to control his legs. The legs tested separately seemed to be strong enough to carry him, though there was undoubtedly some paresis. *No anaesthesia*, such as would be elicited by the prick of a pin or coarse touch. The age of the patient precluded finer tests. Some awkwardness in use of left arm, whether due to ataxia or paresis difficult to say. No tenderness of muscles or nerves. No atrophy. Head tends to fall forward, and he can only turn it slightly towards median line to right (paralysis) but easily to left. Pupils react very sluggishly and incompletely to artificial light; in strong daylight well, but less than right, and rhythmical oscillation of pupils can be made out under latter conditions. Muscles of legs respond well to faradic current.

In the course of the following week the boy grew worse, paralysis rapidly developing, obscuring the ataxia and making it almost impossible for him to stand or walk. As he became too weak to attend the out-patient clinic, the case was lost sight of.

Curiously enough, both cases came under observation at the same time.

The points of interest in the first case are: the paralysis of the palate, without primary inflammation of the throat, showing that this is not due to the local action of the poison, as is commonly assumed (Leyden); the infection following an autopsy wound; and the accidental association of the disease with original absence of knee-jerks, the latter in itself rare. These cases also suggest the probability that many of the cases of locomotor ataxia reported as cured may be of

this kind—a pseudo-ataxy following unrecognized attacks of diphtheria. When occurring in children this probability becomes almost a certainty.

APPARENT PLACENTA PRÆVIA.

BY WM. H. LATHROP, M.D., LOWELL, MASS.

I ATTENDED an obstetric case February 6th which had a feature of special interest. The woman was confined prematurely—at seven months—the miscarriage being caused probably by the separation of the placenta.

I felt the placenta when the os had just commenced to dilate, and supposed that I had a case of placenta prævia, but was surprised that she had no hæmorrhage. I soon found that the placenta was entirely loose. It had become detached from its original position, and found its way to the os, lying across the opening. It thus gave every appearance of being a placenta prævia, until it was observed to be detached, while its manipulation also was seen to cause no bleeding.

The patient was quite pale. She lost some blood when the placenta separated, and this passed in the form of one large, hard clot, after the delivery of the child.

This clot was unusually firm, and the serum from it had discolored the amniotic fluid. She had no hæmorrhage to speak of, excepting this.

Reports of Societies.

SUFFOLK DISTRICT MEDICAL SOCIETY.

H. F. VICKERY, M.D., SECRETARY.

THE annual meeting of the Suffolk District Medical Society was held April 27, 1889, the President, DR. JOHN HOMANS, in the chair. Besides other incidental business, the following officers were elected for the ensuing year:—

President, George W. Gay; vice-president, Edward N. Whittier; secretary, H. F. Vickery; treasurer, E. M. Buckingham; librarian, B. J. Jeffries; commissioner of trials, C. W. Swan; member of the nominating committee, Massachusetts Medical Society, Francis Minot; committee of supervision, F. Minot, R. M. Hodges.

Dr. Homans thereupon vacated the chair in favor of the President-elect, Dr. G. W. GAY. The president of the Massachusetts Medical Society, Dr. D. W. CHEEVER, then made the following remarks:—

Mr. President and Fellows of the Suffolk District Medical Society: I thought it was proper to make a few remarks at this meeting so nearly preceding the annual meeting of the State society, and therefore I ask permission to take up a few moments of your time with regard to the affairs of the society in the whole State.

I have visited fifteen out of the eighteen districts of the society throughout the State, and it seems to me very proper that I should make a semi-official visit among my friends here of the Suffolk District, because, although perhaps you may not realize the fact, the Suffolk District comprises one-fourth of the entire State society. Two other districts are, however, of enormous size,—the Middlesex South has quite two hundred members and the Worcester District has one hundred and twenty or thirty. It