

history in the individual, even if we find cases of nervous disease and of mental disease in the antecedents or even at times in the individual himself, we can sometimes hold forth great hope. We can assure that patient that there is no likelihood of an occurrence or recurrence of nervous or mental diseases in himself, or that, if he marries, there is no likelihood of such an occurrence in his descendants. That is often difficult to decide, but in certain cases he thought we could speak with confidence, and the absolute pessimism of the French teaching on heredity leaves out any element of hope. Dr. Knapp said he laid much stress upon the whole life history of the individual, the importance of faulty training, of faulty nutrition and of example and environment in the production of a neurotic, unstable disposition. He believed, however, that the important thing we must consider is the physical status of the ancestors. In spite of what Dr. Clark and Dr. McCarthy have said, it is by no means susceptible of proof that an acquired neurosis in the parent will be followed by a similar neurosis or any other form of functional neurosis in the child, but if the parent has a defective physical condition leading to pathological alterations of the germ plasma, that may produce a weakling physically in every way, and that weakling may develop neurotic disease. Dr. Dercum's reference to myxedema leads to a specific application. If a parent has myxedema, and after the development of that myxedema, if that should be possible, gives birth to a child, that child might very naturally be expected to be a weakling on account of pathological alterations in the germ plasma from the toxic processes involved in the myxedema, but if after the child is born the parent develops myxedema, especially if we could prove it was myxedema resulting from some local injury to the thyroid gland, the question of myxedema as a factor in heredity would be absolutely thrown out of court. We must make a detailed study of the individual and his family, taking into consideration all infections, all diseases, and all the results of education, of nutrition, of feeding, of the whole life and the special forms of disease which develop in the family, before we can come to definite scientific conclusions as to the importance of hereditary factors.

(To be continued.)

NEW YORK NEUROLOGICAL SOCIETY.

April 2, 1907.

The President, DR. CHARLES L. DANA, in the Chair.

PRESENTATION OF FOUR CASES OF CONGENITAL CEREBELLAR ATAXIA.

By Dr. Millicent B. Hopkins.

A mother, aged forty-four years, and three children, all girls, made up this group. The father of the children was forty-one years of age and apparently perfectly healthy; his personal and family history were negative, save that a brother died of heart disease. As to the mother's family history, her mother died of cancer of the uterus and her father suffered from rheumatism and was an alcoholic. The mother attributed her condition to the fact that her mother, during her pregnancy, received a severe

blow over the abdomen; otherwise her family history was absolutely negative. Her personal history had no points of interest, excepting that she did not walk until her eighth year. She had had six children and no abortions. One child, a girl, similarly afflicted, died at three years of age of scarlet fever, while one girl of nine was a normal child, and a boy of four years was also entirely healthy. Her deliveries were all easy and normal. The personal history of the children was negative, excepting that they did not walk until very late.

Dr. B. Onuf said the patients shown by Dr. Hopkins, at least the eldest girl, seemed to present some choreiform movements of the face or some ataxia of expression.

Dr. George W. Jacoby, referring to the mother of the children, who was forty-four years old, said he had never seen a case of congenital cerebellar ataxia at such an advanced age.

Dr. Arthur C. Brush said he had one case under his observation at present in a man about forty-six years old. The patient had been in the hospital for ten years, and was employed in one of the wards. The case was a very marked example of cerebellar ataxia.

The President, Dr. Dana, referred to a case of cerebellar ataxia of a family type observed by him. In that instance, the disease had developed in three generations. One member of the family, in whom the ataxia did not develop until she was about forty, was now nearly seventy years of age. There was no history of any mental defect in these cases. In all cases the ataxia developed late.

SCHLOSSER'S ALCOHOL INJECTION INTO THE FORAMEN OVALE FOR RECURRENT TRIGEMINAL NEURALGIA, AFTER EXTIRPATION OF THE GASSERIAN GANGLION.

By Dr. Otto G. T. Kiliani.

The patient was a man, seventy-three years old, a cook by occupation, and a native of Germany. His family and personal history was negative, with the exception of the fact that he had been suffering from facial neuralgia for the past forty-eight years. He attributed his affection to an injury which he received when he was twenty-five years old. In 1878 his pain became so intense that the second branch of the trigeminus was resected. He submitted to further operations in 1881, 1884 and 1885, and after the latter operation he was free from pain for four years. The pain subsequently recurred, and in 1898 Gasserectomy was performed, which gave him relief from pain for a year. In 1904 another peripheral operation was performed, which relieved him for two months. When he was admitted to the German Hospital, on Dec. 20, 1906, he was having about one hundred attacks a day. After an ineffectual attempt to find the infra-orbital nerve, Dr. Kiliani made an injection of alcohol, according to Schlösser's method, into the third branch, without any result. Accordingly, on Jan. 19, 1907, he made his first injection of two c.c. of alcohol (80%) into the foramen ovale, after which the patient was free from pain for three days. Similar injections were made on Jan. 26, and on Feb. 7 and 14. Since these injections he had remained entirely free from pain.

Dr. Kiliani said that these injections of alcohol into the foramen ovale were done without narcosis, although narcosis had been given the first time, as he was not positive whether he could rely upon the patient to re-