LA PARAPLÉGIE SPASMODIQUE FAMILIALE ET LA SCLÉROSE EN PLAQUES FAMILIALE (Hereditary Spasmodic Paraplegia and Hereditary Disseminated Sclerosis). MM. R. Cestan and G. Guillain (Revue de Méd., Vol. xx, Oct., 1900, p. 814).

Amongst nervous diseases there is a group termed hereditary diplegia by some, and hereditary spasmodic paraplegia or disseminated sclerosis by others. Under these designations different maladies are confused, as in the two following instances, which are strikingly different: (1) A boy, aged fifteen years, came under observation in May, 1900. His father and sister showed precisely similar symptoms. He had been quite healthy, but at eight he had difficulty in walking. He walked on the points of his toes, and had a constant feeling of weight on his legs. He remained in this condition until twelve. Movements of the arm were then executed slowly and with difficulty, and the head inclined towards the right side. In May, 1900, the boy's gait was obviously spasmodic, but there were no signs of cerebellar trouble. The patellar and Achilles reflexes were exaggerated and there was distinct spinal trepidation. Babinski's sign was present. Intelligence and speech were unimpaired. The author regarded the case as one of organic spasmodic paraplegia with spasmodic torticollis, the lesion being situated in the pyramidal tracts. From a practical standpoint the condition requires to be distinguished from Little's disease. In the latter, however, there is usually a history of premature birth, obstetric traumatism, or infantile meningitis. The spasmodic phenomena, moreover, begin early, and remain stationary, or ameliorate slightly, and there may be strabismus, cranial asymmetry, defective intelligence, convulsions, or arrested development.

(2) This group differs from the preceding in the addition of other symptoms. The parents were healthy, and had ten children, of whom one died in infancy of meningitis, two are epileptics, and two others have special symptoms as described below. A boy, aged sixteen, came under observation in November, 1899. He had been healthy up to the age of three, and easily learned to walk. Subsequently he walked badly, and frequently fell, but there was no pain or sense of fatigue. Between two and seven he had frequent attacks of laryngismus stridulus. He had always been mentally dull, but as he grew up, walking, though not quite normal, was accomplished without difficulty or fatigue. In April, 1899, he began to articulate badly and his sentences were sometimes unintelligible. In January, 1900, there was sudden blindness of the left eye, followed by equally sudden recovery. When examined in February, 1900, he walked slowly with short steps with deviation from the right. If asked to turn around suddenly as he walked, he had a sense of losing his equilibrium, and as he moved there was frequent vertigo. The patellar and Achilles reflexes were exaggerated, and the foot with the toes *en grappe* resembled the condition found in Friedreich's disease. Speech was slow and scanned, as in disseminated sclerosis. There was marked nystagmus. Writing was accomplished without difficulty.

The sister of this patient only developed difficulty in walking at twenty. At twenty-two there were voice troubles. In February, 1900, she was thirty-two. Walking was difficult and spasmodic, not "cerebello-spasmodic," as in her brother's case. There was great rigidity of muscles, and the feet showed the characters of Friedreich's disease. The patellar and Achilles reflexes were exaggerated. Ba-

binski's sign was elicited, and there was marked nystagmus, with slow, scanned speech.

The writer regards both these cases as a family form of disseminated sclerosis. Exaggeration of reflexes, spasmodic gait, the speech and the absence of scoliosis and choreiform movements, excluded Friedreich's disease. The first family was affected by pure hereditary spasmodic paraplegia; the affection in the second was of a different type and belonged to a hereditary type of disseminated sclerosis.

BONAR.

THERAPY.

REMARKS ON FACIAL PARALYSIS, WITH SPECIAL REFERENCE TO TREATMENT. A. Short (Birmingham Medical Review, April, 1900).

The author in his paper says he intends only to deal with the form of neuritis which occurs either in the Fallopiian canal or below it, unaccompanied by any disease of the bone. He has collected forty-one cases of this form of facial paralysis, and has full records of twenty-five. Formerly it was thought that this form of paralysis was due to swelling of an inflammatory nature in the sheath of the nerve, and was ascribed to cold and referred to generally as rheumatic neuritis. Later researches, however, show that in some cases the lesion is a real parenchymatous inflammation of the nerve-structure itself, and not due to pressure alone, the inflammation extending along the nerve outside the skull and even invading the divisions on the face, as well as being present inside the bony canal. Alcohol, lead, and syphilis have all been ascribed as causes of the affection, but the last of these is the only one which seems to be probable.

The treatment is local, general, and electrical. As regards local treatment, counter-irritation behind the ear by means of a blister is of great service in an early condition of the affection, as it often relieves the pain. Should blisters from any cause be inadvisable, a stimulating liniment may be used to rub in over the mastoid process. With respect to the general treatment, iodide of potassium in five-grain doses three times daily is usually given, as in the treatment of other forms of neuritis; but in a considerable number of cases treated without it the author did not think they were any worse for the lack of it. At the end of the first fortnight strychnia should be given, and continued until the muscular contraction is almost as good as on the sound side, and then stopped. By far the most valuable treatment, however, is the electrical treatment by stimulating the muscular fibers with galvanism; in this way their nutrition is kept up until voluntary action returns. The author is of opinion that, as a rule, galvanism is not applied early enough. No doubt a few mild cases will recover without the use of galvanism, but recovery in such cases rarely takes place before three or four weeks have elapsed, and if the case happens to be a severe one the best time for applying the treatment has passed if we wait until the end of this period. Prognosis based upon the presence or absence of the reaction of degeneration is not always to be depended upon, for in one of Dr. Short's cases it was well marked on the fifth day, and the man was quite well in a month's time. The author does not think that the third day of the paralysis is any too soon for the application of galvanism, and the weakest current that will cause contraction of the muscles should be used. Faradism is not absolutely necessary, and when the nerves on account of their irritable condition, respond less readily to it than to galvanism, it should be avoided. Later on, when reaction to faradism is normal, and the voluntary action of the muscles is weak, a