

girdle and extends from the trunks to the muscles of the extremities (Werdnig, Hoffmann).

2. An infantile, bulbar, paralytic, facial type (Fazio, Londe).
3. A Duchenne-Aran type (Strümpell, Gowers).
4. A transitional form.

It seems at present that the influence of heredity is more clearly shown in the myopathic and neurotic forms than in the myelopathic form of muscular atrophy. Fibrillary tremor is the rule in the spinal form and the exception in the purely muscular form of atrophy, and yet it may be absent in the spinal form. Reaction of degeneration may occasionally be absent in spinal muscular atrophy and, in exceptional cases, be present in the purely muscular form. Neither simple nor degenerative atrophy of the muscular fibres is a fair criterion of the nature of the atrophy. SPILLER.

73. BEITRAG ZUR LEHRE VON DER PROGRESSIVEN NEURALEN MUSKEL-ATROPHIE (A Contribution to the Study of Progressive, Neural, Muscular Atrophy). By F. Egger (Archiv für Psychiatrie, 29, 1897, p. 400).

Egger reports two cases of progressive, neural muscular atrophy which occurred in brothers in a healthy family. The disease began in the younger patient when he was thirty-eight. It was first observed in the lower extremities in the peroneal muscles and on the right side of the body. Sensory disturbances were present in both cases. Muscular atrophy and paresis were soon visible in the upper limbs. Reaction of degeneration was also observed. The cases are reported on account of certain peculiar features. In both there was a possibility of poisoning from lead, and it may be that the effects of lead aided in the development of the disease. Vesical disturbance (frequent micturition and dribbling after urination) and swaying of the body on closure of the eyes were unusual symptoms in one patient. One of these cases shows that tabes must sometimes be considered in making a diagnosis of progressive, neural, muscular atrophy, for pain, loss of reflexes, Romberg's sign, girdle sensation and vesical disturbances are certainly very suggestive of tabes. Absence of pupillary rigidity does not necessarily exclude tabes, for an examination of over four hundred cases of tabes in Erb's clinic by Leimbach showed that changes in pupillary reaction were present only in 70.25 per cent., and in cases of one or two years' duration only in 63 per cent. The presence of the disease in two brothers and the early development of the muscular atrophy were contrary to the manifestation of tabes. Egger shares Oppenheim's opinion, viz., that progressive, neural, muscular atrophy is a chronic hereditary form of multiple neurosis; and in this aspect the possibility of lead poisoning in these two cases becomes of unusual interest. Egger thinks with Hoffmann that it is not probable that neuritis may be hereditary, but a slight power of resistance to disease may be transmitted, and it is well in treating a case of progressive neural, muscular atrophy to remove all injurious substances, lead, alcohol, etc., which are so deleterious in neuritis.

SPILLER.

74. BEITRAG ZUR NEURITISCHEN FORM DER PROGRESSIVEN MUSKEL-ATROPHIE (Contributions to the Neural Form of Progressive Muscular Atrophy). Siemerling (Neurologisches Centralblatt, 16, 1897, p. 568, Abst.).

The case described occurred in a young man of 20, whose mother died of tuberculosis. Up to his 5-7th year he developed normally, and then his hands and lower thighs began to atrophy. From his thir-

teenth year he was unable to walk. At first his psyche was normal, but later he became sullen and taciturn.

There was loss of pupillary light reaction, spasmodic twitchings in the left zygomatici, nasal speech with tremor of the tongue, marked atrophy of the hands and arms, main en griffe, the deltoid, pectoralis and biceps being the best preserved; no tendon phenomena, left leg extended, right contracted at the knee; marked atrophy of both sides, loss of active motion in both lower extremities; loss of pain sensations throughout the entire body, more markedly in the legs; fibrillary contractions in the intercostal muscles; marked diminution in the faradic and galvanic currents; hypochondria.

Autopsy showed a normal brain, macroscopic patches in the lateral and posterior columns; fatty degeneration of the lower thigh muscles. Microscopic examination of the cord showed degeneration of the posterior and lateral columns, particularly in the dorsal and lumbar region; atrophy of the anterior horn cells and of Clark's columns; posterior roots intact, anterior degenerated; degeneration of spinal ganglia, sympathetics intact; a widely spread degeneration of the sensory and mixed nerves; degeneration of the muscles in various places. The author believes the peripheral changes to have been the primary ones, the changes in the spinal cord being secondary.

JELLIFFE.

75. **BEITRAG ZUR HEMIATROPHIA FACIALIS PROGRESSIVA** (Contributions to Progressive Facial Hemiatrophy). J. Donath (Wien. klin. Woch., x., 1897).

The patient described was a man 26 years of age, who ten years previously had received a wound on the right side of the face, which left a scar upon his chin. Three years later the patient had a tooth pulled, and in the operation a portion of the lower jaw was also taken with the tooth. The clinical picture began at that time; the skin was thinned, pale, furrowed; there was an atrophy of the muscles and of the bones, suppression of the perspiration and diminution or suppression of the growth of hair. The sensibility of the right half of the face was diminished and the electrical reactions of the right facialis increased. There also was some atrophy of the right half of the tongue. There was no sensation of pain in the wound, but there was slight pain produced by movements of the lower jaw. The author was of the opinion that the pathological process was due to the infection of the wound at the time when the tooth was pulled, and that a diffuse nerve and vessel inflammation was produced, resulting in the clinical picture described.

VOGEL.

THERAPY.

76. **MYOPATHIE PROGRESSIVE AMÉLIORÉE PAR LA MÉDICATION THYROÏDIENNE.** (Progressive Muscular Atrophy Improved by Thyroid Therapy). Lepine (Lyon. Medical, 82, 1896, p. 35).

In a case of juvenile muscular atrophy, where the patient was unable to walk or even to stand erect for any length of time, the author gave weekly doses of thyroid gland—120 gms.—with the result that the patient was enabled to stand erect, and finally to go to work. There were no objective changes in the muscles. The author's explanation was that the thyroid secretions might have some influence over the energy of muscle contractility. Suggestion, he believes, was ruled out.

JELLIFFE.