sugars. Dr. Cadwalader had looked upon this case as a possible one of syphilitic disturbance of the pituitary gland.

Dr. F. X. Dercum said he had placed a case on record of pituitary tumor in a case of tabes. The case was one of acromegaly with typical tabes.

A CASE OF ATROPHY OF THE LEFT SIDE OF THE FACE, THE LEFT ARM AND LEG, FOLLOWING AN INJURY TO THE RIGHT PARIETAL AND MOTOR REGIONS

By John H. W. Rhein, M.D.

Charles Jordy, aged 24, elevator man, applied to the Philadelphia Polyclinic Hospital, February 28, 1916, complaining of pain along the distribution of the anterior crural nerve, following an operation performed on January 24 for keloid, by Dr. Speese.

On examination it was discovered that the leg and to a less degree the arm on the left side were smaller than on the right. This wasting, he stated, followed a gun-shot wound of the cranium in March, 1905. At that time he was accidentally shot while examining a gun.

He was admitted to the University Hospital, and on examination a wound was found over the parietal and motor regions on the right side. The left arm and leg were paralyzed and astereognosis was found on the same side. The face also was palsied.

He was operated on a week after admission, the wound was explored, and two or three fragments of bullets and many hairs were removed. Craniotomy was done, and an electrode applied, but without response. The wound was reopened for drainage, and two weeks after this it was opened more deeply to permit granulating from the bottom of the wound.

On May 25 he was discharged with some weakness in the left leg, exaggerated patellar reflex, but the astereognosis in the left arm had disappeared. He began to work again in six months, after having been in bed for four months.

On April 22, 1907, at the University Hospital, examination showed no sensory disturbances or spasticity. The patient walked with a limp and some toe-drop. The peroneus group of muscles was distinctly weakened, and the anterior tibial also. The patellar reflex was more active on the left side.

The reflexes in the upper extremities were not exaggerated.

On September 9, 1912, the second toe of the left foot was amputated on account of contraction.

Dr. A. P. C. Ashhurst did a Stoffel operation, that is, he divided many of the branches of the internal popliteal nerve supplying the gastrocnemius and soleus flexors of the toes, and the tibialis posticus.

On examination at the Philadelphia Polyclinic Hospital on February 28, 1916, it was found that the left arm and leg were distinctly smaller than the right arm and leg. He was only able to move the foot at the ankle joint slightly in flexion and extension. The knee jerks were equal and slightly exaggerated. A left-sided Babinski and Oppenheim were present. On testing his ankle clonus none was obtained, but there followed the test an irregular tremor of the thigh. There was an irregular coarse tremor of the left leg, when the muscles were put on tension. Sensation was normal.

	Left Circum	Right Circum
Calf	31.5 cm.	34 cm.
Thigh	39.5 cm.	46 cm.
Arm	25 cm.	26.5 cm.
Forearm	25 cm.	26.5 cm.

On April 26, 1916, an examination showed wasting of the left side of face. The left arm was smaller by 1.5 cm. in circumference, and the left leg at the thigh was 3 cm. smaller in circumference than the right, and the calf 1.5 cm. smaller.

The gluteal muscles were distinctly wasted. There was still lowered power of extension and flexion of the foot at the ankle. No sensory change to pain, touch, heat or cold, or astereognosis was present.

The X-ray plates locate the bullet now in the posterior portion of the middle fossa just a little above the base of the brain on the right side. In view of the fact that the atrophy followed soon after the original injury, it is fair to assume that the present position of the bullet has nothing to do with the causation of the atrophy.

The cause of central wasting of cerebral origin is still a matter of discussion. Various theories have been advanced to explain it. It has been attributed to lesions of the pyramidal cells in the cortex (Senator). By some authorities the atrophy has been connected with trophic cerebral centers of the cortex or optic thalamus (Borgherini, Eisenlohr and Quincke). Saville placed the trophic center in the limbic lobe.

v. Monakow and Chatin opposed the theory that the brain possessed a trophic center for muscles in the motor region and in the central ganglia. Oppenheim also believed that this theory had no sufficient foundation.

Caracciolo removed portions of the brain of dogs and explained the trophic changes in the muscles which followed as due to the propagation of the cerebral lesion to the cells of the spinal gray matter by way of the descending fibers which degenerate, an influence which extends to the nerve and muscle fibers. These changes he demonstrated histologically.

The cerebral influence is invoked in explanation of the atrophy by Parhon and Popesco and Roth and Muratow. The action of the cerebral neurons in the vasomotor system causes the atrophy according to this theory.

Marinesco upholds the muscular origin for the atrophy. At the same time he admits that lesions of the sensorimotor zone or degeneration of the pyramidal tracts give rise to circulatory disturbances which the muscles cannot endure and they become atrophic. Goldschieder and v. Monakow look upon the atrophy as sensorimotor, and vasomotor in origin. Chatin believes that the equilibrium of the sensorimotor reflex is disturbed.

Gaussel cited the case in which there was atrophy of one side of the tongue as well as of the paralyzed limbs. It was due, he believed, to a lesion of the internal capsule and the anterior and posterior parts of the postero-inferior thalamus.

The amyotrophy while constant is variable and is due to the suppression of the cerebral nervous influx acting on the large cells of the anterior horns, and on the vasomotor centers. (Schaffer and Marinesco and Parhon and Popesco.)

The cause is assigned to degeneration of the anterior horns by Charcot, Hallopeau and Leyden.

Pitres cited a case in which the internal capsule was the seat of a lesion and in which anterior horn changes were observed.

Mills believed that the atrophy was to be attributed to degeneration of the pyramidal tracts and involution of the anterior horns and to degeneration subsequent to peripheral neuritis occurring in the affected members. He also believed that in some instances it may be due to destruction of the trophic centers in the cerebrum.

Cases have been cited in which the anterior horns and the peripheral nerves have been normal, however, by Babinski, Quincke and Borgherini.

Dejerine has demonstrated that there may be peripheral neuritis with normal anterior horn cells.

Babinski believed that the disturbances in the anterior horns may be manifested by lesions of the nerve cell and muscle and nerve or the peripheral nerves and muscles, or finally in the muscles exclusively, the trouble being sometimes organic and sometimes dynamic.

Gowers believed that the muscular atrophy was due probably to the irritative character of the descending degeneration in the pyramidal tracts. He states that while the degeneration did not descend to the motor nerve cells when irritative in character, it seemed to affect the nutrition of the cells and nerve fibers proceeding from them, and through these the nutrition of the muscles.

Steinert reported sixty-four cases of atrophy of cerebral origin, with seventeen autopsies. He described an early change, consisting of a thinning of the muscle fiber, increase in the nuclei, and fatty degeneration with wax-like degeneration in some. Later these phenomena regressed, the structure of the fibers improved, while the fibers were narrowed with increased nuclei. Nothing was found in the peripheral nerves. In the older cases there was an atrophy of the anterior horns of the affected side.

Gaussel believes that at the present most authors admit a myelopathic or neuritic origin for hemiplegic amyotrophies.

Finally, some authorities assume an arthritic origin for the atrophy.

The case reported supports the hypothesis that the central neurons exercise a trophic influence on the musculature.

Dr. George Wilson asked might not this case fall under the head of arrested development instead of atrophy. He would compare this to the lack of development seen in infantile hemiplegia rather than true atrophy after development had occurred.

Dr. Rhein replied that the patient was only fourteen years old when this occurred and therefore still in the developmental period. The symptoms could scarcely be entirely due to lack of development as the atrophy was apparent, the patient states, shortly after the accident.

Dr. Alfred Gordon presented a case of syringomyelia.

PATHOLOGICAL FINDINGS IN TWO CASES OF PARALYSIS AGITANS

By Grayson Prevost McCouch, M.D., and E. M. Auer, M.D.

In brief the pathological changes noted in the region of the basal ganglia were:

1. Areas of rarefaction containing neuroglia cells and débris, giving the tissue a moth-eaten appearance (previously described by William G. Spiller in a case of contracture of the limbs of the right side).
2. Clean punched-out holes, possibly excessively enlarged perivascular spaces from which the vessels have dropped out (probably similar to those described by S. A. K. Wilson in bilateral progressive lenticular degeneration).
3. Round and oval basic staining deposits chiefly in the perivascular spaces and adjacent tissues (described by F. H. Levy).
4. Diminution in the number of the external medullary laminae and of the radial fibers of the lenticular nucleus with some evidence of degeneration of the latter. Similar changes have been described by Jelgersma, F. H. Levy, and others.
5. Failure of the cells of the corpus striatum to stain well, probably due to the age of the material.