

## Society Proceedings

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### THE PHILADELPHIA NEUROLOGICAL SOCIETY

REGULAR MEETING, MARCH 26, 1920

The President, DR. S. D. INGHAM, in the Chair

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#### SPINAL CORD TUMOR AT LUMBAR ENLARGEMENT

DR. J. HENDRIE LLOYD showed a patient forty-three years of age, who had had a chancre nineteen years previously. He had always been well. The present difficulty began with weakness of the right foot accompanied by burning sensation in the toes. Foot drop then developed and fibrillary tremors in thigh muscles, dysuria and sacral pain showed. Both legs then became exquisitely hyperesthetic, this manifestation replaced later by anesthesia and paresis of bladder and rectum with a complete paraplegic picture at the end of six months. Wassermann tests of the cerebrospinal fluid were negative.

Autopsy revealed a large medullary growth of the lumbar enlargement, with extension of an infiltrative process in the retrolumbar lymph nodes. A marble sized growth was also found in the right cerebellar lobe. Histologically the process was tuberculoma according to Dr. B. L. Crawford who reported no tuberculosis and had not stained for spirochetes. Dr. Lloyd was inclined to believe the process a mixed one, gummatous and tuberculous, a coexistence which though rare nevertheless was known.

*Discussion.*—S. LEOPOLD thought the growth a tuberculoma, W. G. SPILLER also argued for the extreme rarity of isolated medullary gummata saying that in a collection of eight hundred specimens not a single tumor of that type has been seen, whereas the tuberculomata were quite common and resembled the specimens presented.

#### THE CLINICAL INVOLVEMENT OF THE PERIPHERAL NERVES IN DIABETES MELLITUS

DR. WALTER M. KRAUS, of New York, presented the results of a statistical survey of four hundred and fifty case reports of diabetes mellitus. These reports were selected from nearly seven hundred accumulated during ten years at the Metabolic Department of the Vanderbilt Clinic,

New York. The discussion was limited to those cases having involvement pointing to disease of the spinal cord or peripheral nerves.

The symptoms and signs considered were:

1. Areflexia.
2. Neuralgia.
3. Ulceration and gangrene.
4. Pseudo tabes.
5. Spinal cord disease.
6. Herpes zoster.
7. Peripheral neuritis.

Absence or diminution of knee reflexes, on one or both sides was found in thirty per cent. of the cases. Exaggeration ten per cent.

The Achilles jerks were absent in sixty-four per cent. of the cases tested (eight per cent.).

Age, sex, race, nutrition (determined by the loss of weight and the weight divided by height index at the time of examination) the duration of the disease, the condition of the arteries, the level of the blood pressure and the presence or absence appear to play no part in determining hypo- or areflexia.

The severity of the disease as determined by the course and carbohydrate tolerance, seemed to play a part since knee reflexes were absent in twenty-two per cent of the mild cases, thirty-three per cent. of the moderately severe cases and fifty per cent. of the severe cases. Neuralgic pains were present in the extremities in about twenty-five per cent. of the cases.

A group of cases showing objective changes in light touch sensation, reflexes and with some neuralgia, were not uncommon. These are due to spinal cord involvement—the intramedullary portion of the posterior roots. This was shown pathologically by Williamson, Kalmus, Schweigger and others. These cases are not neuritis and should not be spoken of as a "peripheral neuritis." Peripheral neuritis in diabetes is very rare. None of the pathologically reported cases were true sensorimotor peripheral neuritis. In the seven hundred cases at the Vanderbilt Clinic none were such except the single case of a patient who was also an alcoholic. This is striking. The coincidence of alcohol in those few cases showing peripheral neuritis is emphasized by all writers on the subject. It seems therefore that true peripheral sensorimotor neuritis is either a very great rarity as a sequel of diabetes or is never a sequel, but must be attributed to an intercurrent condition other than diabetes.

Ulceration and gangrene occurred in very few cases, herpes zoster in none.

The cases showing absence or diminution of deep reflexes, sensory changes in the skin, neuralgia, ataxia (diabetic pseudo tabes) can all be explained anatomically by a partial lesion of the intramedullary portion

of the posterior root. The changes in the posterior columns follow. Gangrene may have the same origin. Herpes zoster and peripheral neuritis appear to be rare conditions in diabetes and not dependent upon it as a sole cause. Diabetes may predispose to these conditions, but does not cause them. [Author's abstract.]

*Discussion.*—DR. W. B. CADWALLADER believed the clinical evidence pointed to a general radicular process quite dissimilar to the myelitic changes noted in pernicious anemia to which latter process the reader of the paper had referred it. Dr. Kraus called attention to the intramedullary involvement of the root fibers pointed out by Williamson of England.

#### PARIETAL TUMOR

DR. J. H. W. RHEIN presented a colored man, forty-nine years of age, with negative previous history. Seven years ago he had been struck on the parietal region by a club. Two years later a small tumor grew there and rapidly expanded. Two years ago patient had a generalized convulsive seizure followed by confusion, and paresis of right side, including the motor speech mechanisms. Later twitching of face, numbness of right side and paresis of foot and speech musculature were apparent. These would come on in attacks averaging two a month. Parietal pain was evident at these times. In 1918 he had a second generalized convulsion, a third in 1919 and another in October of that year. Weakness of the right side and twitchings were continuous and the speech was disturbed and orientation involved.

In November, 1919, his eye sight began to fail and he had pain in parietal region and difficulty in recalling words. Neurological status at that time showed slightly enlarged right pupil, prompt eye reflexes, normal eye grounds, no other cranial nerve signs. Weakness and astereognosis of right hand, with hypermetria, accented knee and ankle jerks of right side, slight hemiplegic gait; x ray examination revealed large shadow of right parietal side 6 by 4 inches, chiefly osteitic in character with softened areas. Nearly positive Wassermann reaction.

Later attacks were observed in 1920 with increasing involvement of cranial nerves, facial paresis, hemianopsia, right homonymous, speech involvement and increasing hemiplegic signs, apraxic and agraphic syndromes. No results following antisyphilitic treatment diagnosis of bony sarcomatous growth was made. Surgical intervention was advocated.

*Discussion.*—J. H. LLOYD spoke of the extension of pressure as explanatory of the hemianopsia.

C. W. BURR spoke of the good results from radium treatment in a somewhat analogous case.

S. D. INGHAM explained the absence of choked disc as due to the spontaneous decompression due to the eroding tumor mass.

## ORBICULARIS TIC

T. A. WILLIAMS presented the history of a woman of fifty years who, while nursing a sister two years previously, had developed an obstinate spasmodic contraction of the orbicularis muscle. The reaction was believed to be psychogenic and improved greatly under reeducative and combined pharmaco and dietotherapy.

## PARAPLEGIA

T. A. WILLIAMS presented the notes on an engineer thirty-seven years of age who had three years previously developed a paraplegia while organizing a union and suffering a back injury at that time. It cleared up in eight months after manipulative therapy. He was greatly relieved by a common sense reeducative treatment of persuasion within a comparatively short time.

## UNUSUAL SENSORY DISSOCIATION

T. A. WILLIAMS presented the history of a student of twenty years who in December, 1918, suddenly became exhausted. In the following May he noted a dimness of vision in the right eye, closely followed by fainting attacks with rigidity and spasms, numbness and hypoaesthesia in the feet with unsteady gait. A few months later further progression and paraplegic crippling. By February, 1920, a fairly complete multiple sclerosis syndrome was evident with what the reporter considered a rare syringomyelic dissociative syndrome also present.

ASSOCIATED UPWARD AND DOWNWARD OCULOMOTOR  
PARESIS

W. B. CADWALLADER presented a boy of sixteen who in June, 1919, first complained of visual difficulties. In November, 1919, he had been hit by an automobile but apparently had not been severely injured. A month later bilateral temporal headaches developed with increasing difficulty in writing, with marked tremor. In February, 1920, neurological status showed enlarged right pupil, immobile to light, sluggish to other stimuli. Globes could not be raised above median plane but were slightly movable downward. Other ocular movements were free and ample. Superior rectus of each eye was paralyzed—the inferior rectus paretic. Small vessels and slightly swollen right disc. Ataxia of right upper extremity, with increased tendon reflexes, no paresis. Ataxia and clumsy movements of the right leg and right sided staggering were present. Tendon reflexes also exaggerated on this side. Diagnosis of mesencephalic tumor was made.

## BILATERAL LENTICULAR DISEASE

GEORGE WILSON presented a boy ten years of age who showed delayed walking (five years) and who had just begun to talk. The gait was stiff and the speech was dysarthric, dysphagic and explosive and facial grimacing was present with other partly athetotic choreic like movements. There was slight hypertonicity of the extremities but no sensory changes or muscular weakness. The author proposed it as a lenticular syndrome.

C. B. BURR was disposed to regard the case as one of congenital cerebellar disease. W. G. SPILLER concurred in the assumption of lenticular disease.