

## SENILE PARAPLEGIA.

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THE clinical history of the case is as follows: G. B., aged 79 years, widower, white, native of Scotland, jeweller by occupation, was admitted to the outwards of the Philadelphia Hospital, May 3, 1895, and to the men's nervous wards of the same hospital, February 13, 1896, and the following notes obtained:

No history of any hereditary disease. He was treated in the surgical wards of the Philadelphia Hospital during September, 1895 for urethral stricture, and subsequently again transferred to the outwards. He has been a moderate drinker: syphilis is denied. About three months prior to his admission to the nervous wards, he noticed that he was weak in the legs. He never had had any distinct apoplecticiform attack. Two days before his admission, on attempting to arise in the morning he found that he was paralyzed from the waist down. He had retention of urine and incontinence of feces.

Examination on admission from the outwards revealed complete paralysis of all the muscles below the waist. There were no areas of anæsthesia. The third day after the paralysis came on, bed sores began to form over the sacrum and heels. Although he was put on a water bed, these bed sores rapidly became worse. Examination of the urine showed a trace of albumin and a large number of granular casts. Symptoms of uremia began to develop and spots of consolidation in both lungs could be detected. The fourth day after his admission he became delirious. The temperature never went above normal. He became rapidly weaker, and died seven days after admission, apparently of uremia (February 20, 1896, 5 P.M.)

Post-mortem examination by Dr. Jamison, February 22, 1896, 11 A.M. Body of well-nourished old man, a small bed sore over the left heel and a large one over the sacrum. Bladder distended and inflamed. Kidneys show parenchymatous and interstitial nephritis. Heart nor-

mal. Lungs emphysematous; a few spots of senile pneumonia. No gross lesions of the brain or spinal cord, except a chronic leptomeningitis.

For the specimen (spinal cord) and the above notes, I am very much indebted to Dr. Charles K. Mills.

Microscopical examination of the cord revealed changes which, though intimately associated in a causal relationship, it will be convenient to describe under two headings, first, those affecting the nerve fibres; second, those affecting other tissues.

Upper cervical region: In the postero-median columns, close to and parallel with the posterior two thirds of the median fissure, there is a narrow area in which the nerve fibres are diminished in number and in thickness. There is a quite marked degeneration of conical shaped area with base inward, bounded by the anterior one third of the posterior fissure, the posterior commissure, and the posterior cornua, and extending pointedly into the postero-external columns almost to the periphery. In the lateral columns not strictly localized to the pyramidal tracts although there much more marked, the nerve fibres are degenerated in part.

Cervical enlargement: The degeneration in the postero-median columns along the posterior fissure is more marked than in the previous section, while the conical-shaped area alluded to, is larger. The degeneration in the lateral columns is marked, but is not limited to the pyramidal tracts. While these tracts are much more affected than are other regions, the degeneration extends forwards into the anterior ground fibres.

Lower cervical: The degeneration in the posterior columns, while more marked along the posterior median fissure is distributed to some extent throughout the columns. The degeneration in the lateral columns as heretofore affects particularly and more markedly the pyramidal tracts, though it is not confined to them.

Upper, middle and lower thoracic regions: The degeneration in the posterior columns is much more general and much more marked than in previously described sections. The degeneration in the lateral columns is of about the same extent as in the last mentioned section.

Lumbar cord: The degeneration in the posterior columns is very marked. In the lateral columns as heretofore.

Sacral: The fibres along the lateral margin appear fewer in number and narrower than elsewhere

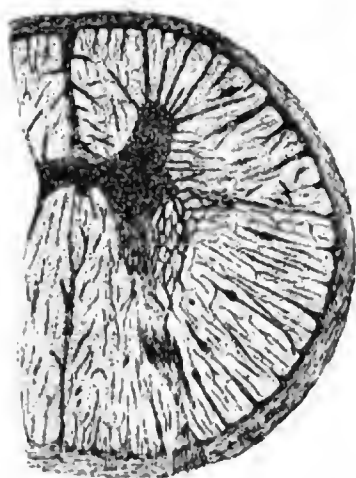


FIG. 1. Drawing showing the excessive intra-spinal overgrowth of connective tissue and its sources of origin, the sclerosis of blood vessels and pia mater. Ammonio-carmin preparation.

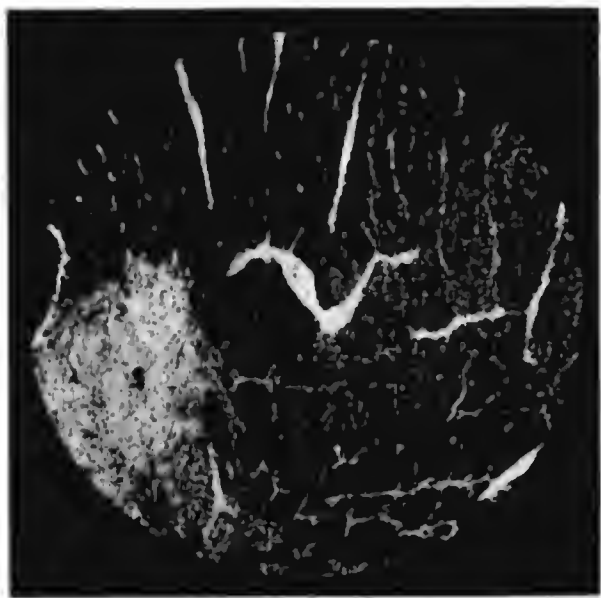


FIG. 2. Photograph showing the blood-vessel sclerosis, the associated perivascular connective tissue overgrowth, and the numerically increased and much thickened trabeculae of connective tissue and the numerous islets of sclerosis in connection therewith. Weigert-Pal preparation.

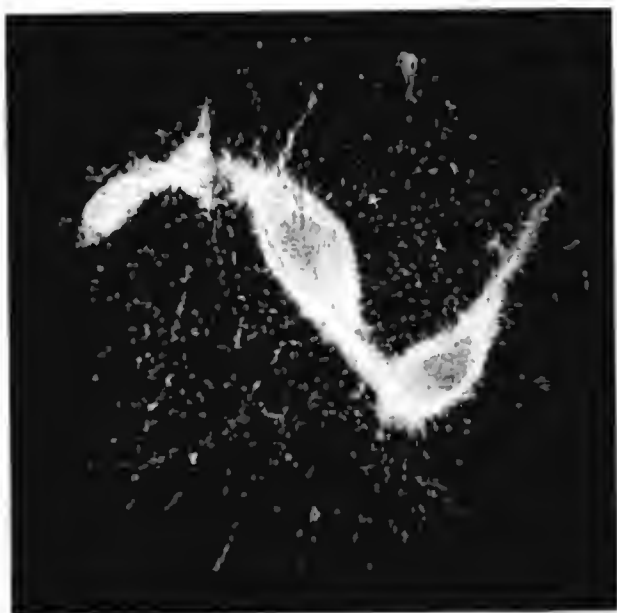


FIG. 3. Higher magnification of Fig. 2.

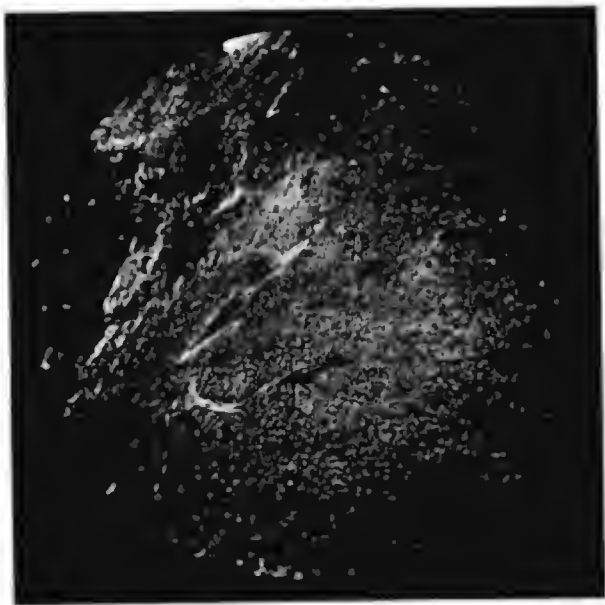


FIG. 4. Photograph showing changes similar to those evident in Figs. 1, 2, and 3. Hematoxylin—Picric Acid—Fuchsin preparation.

When not otherwise stated, the nerve fibres of the various tracts appear normal.

Throughout the cord at all levels, but especially in the regions noted as revealing nerve fibre degeneration that is, in the posterior and lateral columns—there is a great increase of the connective tissue. This connective tissue overgrowth is intimately associated with changes in the blood-vessels and pia-mater. With the exception of the very minutest ones, the blood-vessels are everywhere distended, and all have greatly thickened walls. This applies to vessels of all calibres, from minute spinal ones to larger pial vessels. The smallest spinal vessels have greatly thickened walls and the lumen of many of them has apparently become obliterated. Careful examination of somewhat larger ones, reveal slight proliferation of the intima and great increase in the thickness of the media, which is poor in nuclei, concentrically laminated, and apparently somewhat hyaline in structure. Surrounding this latter is a layer of variable thickness of less homogeneously staining connective tissue. These changes are very marked in some of the median sized and larger vessels, which themselves are occasionally the seat of some round cell infiltration.

The pia-mater is very greatly thickened, contains a very few nuclei, and has become united with the arachnoid. The peripheral layer of the spinal neuroglia is also greatly thickened. From this greatly thickened pia and neuroglia, and from the blood-vessels with thickened walls as noted above, there extend throughout the cord numerous connective tissue trabeculæ. These are very greatly in excess in number and much thicker than normally, and while present throughout the cord, are much more in evidence in the areas already referred to as revealing nerve fibre degeneration.

These trabeculæ are frequently very thick, inclosing within their confines bundles of nerve fibres, whereas, again they are finer and surround only individual fibres, leading thus to degeneration of single fibres. The blood-vessel sclerosis is, of course, not confined to the vessels of the white matter of the cord, but implicates of necessity those supplying the gray matter, which latter is not, however, appreciably altered. The central canal is filled with proliferated epithelium.

The most evident and important pathological alteration discovered in these sections is, therefore, the excessive overgrowth of connective tissue; the nerve fibre

degeneration being very properly considered secondary thereto. The intimate association of this sclerosis with the blood-vessels is very patent. The blood-vessels themselves are the seat arteritis and endarteritis, and around them as foci proceed trabeculæ of connective tissue encircling in places numbers, in others, fewer nerve fibres. The neuropgia along the periphery of the cord is also greatly thickened, and from it proceed also into the cord, trabeculæ of fibrous tissue, much increased in thickness and in number.

This sclerosis originating around the vessels and periphery of the cord, while marked in all regions of the cord, is, however, much more intense in the posterior and lateral columns, especially in the area of the pyramidal tracts, and is more marked in the thoracic region than elsewhere. In areas slightly affected, more particularly where the sclerosing tissue surrounds groups of nerve fibres, rather than individual ones, the fibres for the most part appear normal. In other regions, however, in which the sclerosing connective tissue envelops frequently individual fibres, these have suffered greatly therefrom and have become atrophied, axis cylinder and medullary sheath in many instances, having disappeared, in others being much narrower than normally. There remain, therefore, in consequence, many minute islets of fibrous tissue. In places where several larger vessels are in close opposition there are quite large spots of sclerosis.

There was discovered no embolic, thrombotic, hemorrhagic, or other process to account particularly for the acute manifestation of the severe symptoms. The case is simply an illustration of one of the many instances with which the neuropathology abounds in which definite lesion are for a long time devoid of any very manifest symptoms. These when they do come on, frequently make their appearance abruptly.

That the patient, however, was not entirely without symptoms prior to the acute attack of paraplegia, is evidenced by the weakness of his legs. And, in the light of the microscopical examination, and reasoning *a priori*, one may very safely assume that there were present other symptoms, unfortunately overlooked as is but too frequently the case in the gradual and progressive weakness which attends old age. One cannot, therefore, but regret imperfect history.

The designation "Senile Paraplegia," is admittedly

ill-chosen, but has been selected because of its conciseness, and because it is sufficiently expressive of the clinical condition. We do not, however, desire to be understood as suggesting that the changes described are essentially senile in nature. They are distinctly pathological, and while frequently an accompaniment of old age, they are to be considered apart from changes of a purely senile character. It will hardly be appropriate to here enter upon the discussion of this subject in detail. For the photographs I am much indebted to Dr. Schively.