

In 1877 it was observed that this member had emaciated in its whole bulk. With the eyes shut, he walked hesitatingly. Sensibility was diminished, and the fulgurant pains were severe. On the side operated on, nothing analogous to what happened on the sound side was observed.

In summary then, all the facts which we have passed in review demonstrate:—

1st. That the peripheral irritation of a nerve may determine in the nervous cord, and in the central axis, inflammatory modifications. 2nd. That these inflammatory lesions are produced either in the involucre of the medulla, or in those of the nerve; on the constituent elements of the conducting cord, and of the medullary axis. 3rd. That the medullary lesions have their seat principally in the grey substance, but they may extend also to the white substance. 4th. That they may be limited to the meninges, according to the observances of Klemm. 5th. That in the majority of the cases, if not in all, the propagation of peripheral irritation to the medulla is effected by means of the centripetal cord, under the form of a neuritis, now disseminate, or again continuous, having its seat in the interstitial connective tissue, and probably also in the nerve tubes. 6th. That these central lesions are often manifested with predominant muscular atrophy, and in some cases with signs of locomotor ataxia.

*B. Lesions central, produced by irritation of visceral nerves.*—We are now interested in seeing whether lesions of the internal organs may bring about consequences on the nervous centres, in the same manner as the peripheric lesions of members. A great number of facts published under the name of reflex or sympathetic paralysis, have no other known pathological mechanism.

It was observed in an individual who for several years had suffered under an affection of the urinary passages, that without appreciable cause a dorso-lumbar myelitis more or less rapidly was developed. Gull, combating the vaso-motor theory of Brown-Sequard, showed that urinary paraplegia supervenes principally in those individuals who, for several years, have suffered from vesical or urethral diseases. Leyden records an observation, in which, in sequence to a cystitis from cold, with retention of urine, symptoms of paralysis appeared at the end of four weeks; at the autopsy there was found a red softening of the lumbar medulla. An Italian

author, Namias, observed a case of central atrophy of the medulla, consecutive to a chronic enteritis, in a woman of 38 years.

Wier-Mitchell says that, in some cases observed by him, "*intestinal diseases had produced effusions and medullary softenings,*" and that the scrofulous and the scorbutic are often subject to softening and chronic myelitis. But he does not give any details, nor cite any autopsy. Leyden has published his observance of a man, who, in sequence to dysentery, had symptoms of a lumbo-sacral neuritis, to which there succeeded those of an ascending spinal meningitis, mounting up to the superior dorsal region. In a memoir of Zabriskie, we read the following fact: A boy entered the hospital with chronic diarrhœa, which had so weakened him that his lower limbs had become paralyzed both in sensation and motion; his evacuations passed involuntarily, and he died from marasmus. The intellectual faculties had remained sound. At the autopsy, extensive lesions were found in the small intestines. The medulla and its involucre did not present, to the naked eye, any alteration. All the viscera were sound. But though the author realized the integrity of the medulla by the naked eye, the complete paralysis of sense and motion, and the paralysis of the sphincters, prove the existence of lesions in the grey substance of the medullary axis, which would not have escaped microscopic examination.

All these facts, above exposed, establish, though not in a very definite manner, the possibility of medullary lesion as a consequence of inflammation of the viscera. But as yet the studies have been rather defective: there have been observed only these few cases in the urinary and intestinal organs, of medullary affections consecutive to irritation of the visceral nerves. As regards lesions produced by irritation of the other viscera, no example is known.

It remains also to know by what mechanism the visceral affection is transmitted to the central nervous system. Gull admits that the inflammation may be propagated, in certain cases, by the veins, to the rachidian plexuses, and thence to the involucre of the medulla. But the more rational hypothesis is that offered by Leyden, and supported by Charcot, that is, the centripetal propagation of the irritation through the nervous trunks; and this accords with the observations of Leyden, who, in