This description approaches that of the juvenile form of amaurotic idiocy of Tay-Sachs, but in the case reported the nerve cells have not the typical form of those in the Tay-Sachs disease nor is there the same degree of optic degeneration. The clinical similarities to general paresis are the progressive character of the dementia and the epileptiform attacks. The anatomical ones are the cortical degeneration, the disappearances of many cells, the gliomatosis of the marginal zone, hydrocephaly accompanied by granulations of the ependyma and the degeneration of the fibers. But in general paresis there is a much more complete cortical destruction. In the vascular alterations also the difference is evident between that of cerebral syphilis and general paresis. The patient under discussion does not therefore belong under the idiocy of Tay-Sachs nor under general paresis. The spastic paralysis is probably the result chiefly of the degeneration of the pyramidal tracts. The author suggests that the pathogenesis of idiocy may depend also upon the amount of white matter as well as the gray, that is upon the association fibers. It is the lack of these which actually interferes with thought processes. The bad state of the circulation has been accountable for the later neuroglial proliferation, which followed the first aplasia of the fibers. The diminution of the tension, occasioned at first by the internal hydrocephaly, the destruction of the white substance, does not explain sufficiently the extension of the furrows nor the striking disproportion of the cortical surface as compared with that of the white substance. It would seem that the furrows serve to nourish the brain and gain in depth and ramifications as this becomes more difficult.

Thus the author summarizes: Congenital spastic paralysis of a hereditary syphilitic origin is a morbid entity. It is generally associated with idiocy and it is progressive. The development of the central nervous system is retarded and there is degeneration of those fibers which are myelinated more slowly, particularly of the pyramids and the long associative tracts. The vascular walls are thickened and there is visible evidence of a former luetic meningo-encephalitis without gummatous formation. [J.]

Mendicini, A. PSEUDO-TABES OF TRAUMATIC ORIGIN. [Il policlinico, Feb. 1, 1918.]

A traumatic lesion of the cauda equina may simulate in its clinical aspects a sclerosis of the posterior columns. This is a known clinical fact and is further demonstrated in a case recently published by this observer.

The patient, a soldier, presented a syndrome characterized by amyasthenia of the legs, more marked on the right side, associated with hypotonia of the muscles, abolition of the Achillar reflexes and the left knee-jerk, Romberg's sign, ataxic gait, lightning pains, and intermittent paresthesia, with disturbance of sensation in the regions of the fifth lumbar and all the sacral nerves on both sides, but partly also of the third and

fourth right and fourth left lumbar, together with derangement of the functions of micturition and defecation. These phenomena followed a wound by shell splinters of the lower lumbar vertebræ, which at its onset caused a complete paralysis. The resemblance to tabes was so marked that the doubt arose whether the patient might not have been the subject of a latent form of locomotor ataxy, but against this supposition it was ascertained that previous to the injury he had no symptoms referable to the nervous system; moreover, the Argyll-Robertson sign was absent. The seat of the injury and radiographic examination pointed, on the other hand, to a lesion of the cauda equina, the fibers of which were either contused by the fragments of shell, which were numerous, although it is more probable that these produced an endothecal hemorrhage. To this and the shock of the wound itself the initial paraplegia was certainly due, which passed off in about three weeks owing to gradual absorption of the blood extravasated into the meningeal sac. In fact, the irritative symptoms (retention of urine, pains, and painful priapism) became gradually lessened without giving place to noticeable paralytic phenomena. The progressive improvement of the patient justified the exclusion of a secondary inflammatory process of the meninges. The pathology of the case therefore resolves itself into one of peripheral radicular pseudo-tabes, which differs from the classic neuro-tabes in the topography of the sensory changes and in the presence of disturbances in the sphincters.

Dr. Mendicini's patient had Romberg's phenomenon and ataxic gait; when in bed, however, or seated, he could move the legs correctly into any position and retained the sense of position except in most of the toes. This fact is of some importance as showing that only the paths proceeding from the terminal segments of the lower limbs were impervious to the transmission of deep sensation; it explains the Romberg sign and disturbance of gait, and demonstrates that if Romberg's phenomenon is met with chiefly in relation to degeneration of the posterior roots it may also be caused by simple plantar anesthesia. On the other hand, the retention in this patient of deep sensibility in the proximal part of the lower limbs gives the reason of the absence of static and dynamic ataxy in the recumbent or sitting posture. In tabes, on the contrary, an ataxic gait associated with Romberg's sign is never met with in conjunction with absolute static and dynamic integrity of the legs in the supine or sitting position, not even in cases of tabes inferior in which the lesion is limited at the onset to the sacral or last lumbar roots.

4. MENINGES.

Carter, W. W. SINUS THROMBOSIS FOLLOWING SKULL FRACTURE. [New York Med. Jour., June 23, 1917.]

Following being struck by a truck the patient suffered a fracture of the skull in the right temporal region. There was profuse hemorrhage