

ganglion-cells of the posterior cornua in all the sections appear to have undergone changes. Through the entire length of the cord many of them have lost their prolongations. In the upper dorsal region they are much larger; they have a bulged and swollen appearance, and several can be seen with their nuclei in a state of division—some in the act of dividing, others with two separate nuclei. In many of them no trace of nuclei or nucleoli can be discovered; further down in the dorsal portion of the cord the cells present a somewhat atrophied appearance, and the posterior cornua of the grey matter are thin and translucent, a small segment in the region of the ganglion-cells being much atrophied, and not taking the hæmatoxylin staining.

In the lumbar region of the cord the ganglion-cells of the posterior cornua are very much diminished, both in size and in number, in some of the sections only two or three small and imperfect cells can be seen; the cells of the anterior cornua seem to be perfectly normal. This portion of cord is very much more vascular than the lower dorsal region, some of the vessels presenting a peculiar knotted, or rather double-looped appearance.

In all the sections of the dorsal region of the cord the central canal is completely obliterated, owing to its being plugged with epithelial cells and granular matter. In the lumbar region it is pervious, and almost rectangular in shape.

The nerve fibres of the white matter in the anterior dorsal region are in a state of granular degeneration, their axis cylinders are but faintly seen—can only be occasionally distinguished from the cells and nuclei which, with granular matter, swell the neuroglia.

The white columns in the lumbar region present no abnormal appearance.

In referring to the pathology of this case, the first questions are:—Is this a case of spontaneous spinal apoplexy, analogous to frequently recurring cases of cerebral apoplexy, in which no lesion of the nervous centre itself exists before the escape of blood into its substance? Or, is it the result of previously existing inflammatory action in these centres? That blood vessels can spontaneously rupture into the substance of the healthy cord, as in some forms of cerebral apoplexy, is proved by a few

carefully reported cases, more especially one by Goltz in a recent number of Virchow's Archives.

Writing in 1876, he says, that but thirty cases of spinal apoplexy are recorded, of which at least twenty showed symptoms of previously existing myelitis. The case just reported must, I think, be considered one of this class, although the indications were ill-defined. The symptoms present pointing to the probable existence of myelitis before the occurrence of paralysis were:—A lichenous eruption on the body, flushes of heat, chills, slight pains in the limbs, and a feeling of not being quite so well able to work as usual, although he managed to follow his trade as a carpenter up to the day previous to the paralysis. To these may be added two at least of the symptoms which were present when he entered Hospital—abolition of reflex action and almost complete loss of electric excitability of the muscles; the latter symptom especially, I look upon as important, since without it the former would lose its significance at this early stage of the disease. In animals reflex action is abolished for a certain length of time after division of the spinal cord, varying with the species; thus in the frog only two or three minutes, whilst in the rabbit as many or more hours frequently will elapse before the reflex irritability returns—this condition being due to shock.

In the cases of injuries to the spinal cord in man, which most nearly approach the conditions experimentally produced in animals, the length of time that has elapsed after occurrence of injury before appearance of reflex action is very varied; in some of the reported cases it has been observed within a couple of hours, in others, three or four days, sometimes more, have elapsed before the cord recovers from the shock and reflex action appears. Cases of injury are also frequently recorded, some of them in the cervical region, in which at no time during the progress of the case were there any manifestations of reflex action; in these its continued absence might, I think, be fairly ascribed to changes extending to the ganglion-cells of the cord below the lesion, as they have been for the most part cases in which inflammatory softening had been produced at site of injury by a dislocated or fractured vertebra. At no time during the progress