The mental condition of the child is improving slowly, but I have grave doubts if the restoration in this respect will be perfect. The condition of the muscular system is very bad. Both upper extremities are very much involved, and, no doubt, in spite of all efforts there is going to be considerable deformity. The lower extremities are not so seriously implicated as the upper. The somatic muscles are also affected, and the neck is still stiff. I have noticed, occasionally, some indications of athetoid movements. The condition mentioned by S. Weir Mitchell of lead pipe paralysis is exceedingly well shown in this case. Flex or extend either of the upper extremities, and it remains in the new position. This is true, to a lesser degree, of the legs. The reflexes are increased, but the spastos is so marked as to interfere with the exhibition of the myotatic contractions.

As to the prognosis, I might very appropriately quote the words of Hirt, that "*quoad valitudinem* it is absolutely bad. Complete recovery is impossible." Certain important sections of the brain are irretrievably ruined. The direct consequence of this is that the perverted muscular phenomena must persist.

The case is so clearly one of cerebral palsy that I refrain from making any differential diagnosis between it and the spinal type of spastic paralysis in a child where the lateral tracts are diseased, without any accompanying lesion in the brain.

To what is the spastic condition of the muscles due? This is an easy question to ask, but by no means an easy one to answer. J. Hughlings Jackson and H. Charlton Bastian have held and taught for some time that if the dorsal part of the cord is completely destroyed in its transverse axis, there will be no reflexes in the parts of the body supplied by nerves coming off from the cord below the injury or disease. They hold that the cerebrum inhibits the deep reflexes, and that the cerebellum increases them. When the motor path anywhere in the cerebro-spinal portion is destroyed, the controlling power of the cerebrum is lost, while the cerebellar influx, still going on, produces the spastic condition. In support of this view, cases are cited where there were complete transverse lesions of the cord, in the upper region, with absolute loss of the knee jerk.

Against this view we must range such men as Gowers, Buzzard, Bristowe, Hirt, etc. W. R. Gowers shows that in cases of injury to the dorsal part of the cord, with loss of muscular reflex, there is also loss of skin reflex, and also muscular atrophy, with the electrical reaction of degeneration. His view of these cases is that, in injury to the cord in the dorsal region, the degeneration that ensues is of an irritative character; and, when it descends to the lumbar region, it excites into activity changes that destroy the roots of the lumbar nerves, and in this way differs from those cases of descending degeneration where the reflexes are increased, instead of being lost. There are heavy arguments in favour of both views. In my papers on the knee jerk, which appeared some time ago in the *New York Medical Record*, several cases were reported that would seem to uphold the opinions of Jackson and Bastian. We must wait patiently for pathological investigation on these cases, as the clinical evidence alone is not sufficient. If the view of Gowers should be finally proven correct that, in all cases of injury of the cord, in the dorsal region, where the knee jerk is lost, there is a descending irritative change in the cord, as well as a descending degeneration, we must regard the cord as a chain of reflex centres, and that these centres pass into a condition of abnormal activity when the influence of the cerebrum is cut off. The final settlement of this vexed problem will be of great value in diagnosis and prognosis, and will do much to throw light upon the normal physiological functions of the human spinal cord.