Before further discussing the pathology, I shall read you the history of my second case.

R. D., æt. 57, was admitted to the hospital, March 28th, suffering from severe pains, and lameness of the back, which we thought was probably of a rheumatic character. In February the patient had a severe attack of la grippe, from which he did not make a complete recovery. For some years he has been employed in a distillery and has taken a glass or two of whiskey a day. He is of full habit and has a marked acne rosaceæ. For the last few years he has been employed as an engineer. There is nothing of importance in his family history. Patient has suffered for the last six years with pain and stiffness of the back. This became much worse after the attack of la grippe, so that he was compelled to enter the hospital. After his admission various anti-rheumatic remedies were given, such as soda salicylate, alkalies, salol, etc., without any success. Hypodermic injections of $\frac{1}{8}$ gr. of pilocarpine were then ordered to be given each evening. Profuse perspiration was induced and the pain ank soreness of the back very much relieved. After four injections patient was seized with an acute attack of diarrhœa accompanied by pharyngitis. The temperature ranged between 99° and 100°. Pulse increased in frequency. The hyopdermic injections of pilocarpine were discontinued as the pain was relieved and it was thought he might have taken cold after the heavy sweating. Under medicinal and dietetic treatment he recovered so that at the end of a week he seemed to be in his usual health.

On April the 28th, three or four days after his recovery from the diarrhœa, and ten days after the last hypodermic injection of pilocarpine, an entirely new set of symptoms appeared. He felt first a tingling in the fingers of both hands, which was followed by pain which kept him awake during the night. On the following day, April 29th, the pain had disappeared, leaving a smarting sensation in the hands and arms, extending as far as the shoulders. In the afternoon he felt a tingling in the toes, extending upwards to the knees. This morning in attempting to rise he found he could scarcely stand up, owing to the weakness of his legs. He was not able to go to the bath-room without assistance. To-night he is unable to stand up, or to put his hands to his head. It was neceessary to use the catheter to empty the bladder.

May 1st—All motor power is completely gone. Paralysis of the bladder; urine normal in character. I visited the patient in the afternoon and found him quite sensible and free from pain. There was complete paralysis of the upper and lower extremities, also of many of the trunk muscles. Respiration was altogether diaphragmatic. Sensation was apparently normal. During the evening the signs of bulbar paralysis became manifest; respiration was impeded, and deglutition became difficult; coma ensued, and he died shortly after midnight. There was almost complete absence of sensory disturbances during the illness.

I regret very much that, owing to the short duration of the illness, and to my being otherwise engaged, no electrical examinations were made. A post-mortem was made within twelve hours after death. Upon opening the spinal canal the meninges were found normal. There was no excess of cerebro-spinal fluid. After removing the cord and making cross sections, the grey matter was found to present a distinctly pink color, which I was able to demonstrate to the students at the time. The grey matter of the lumbar and cervical regions were principally affected. Nothing abnormal was found in the spinal column; nor was there anything special to note about the internal organs. Upon microscopical examination minute extravasations were found in the anterior cornua, both in the cervical and lumbar regions. Inflammatory exudation and destruction of nerve cells was made evident. In all the sections the inflammatory process appeared at its greatest intensity in the anterior cornua. We have, then, in this case, an acute myelitis affecting principally the grey matter, and especially the anterior cornua.

It was a singular coincidence that this patient occupied the next bed to that of the patient I just described. So far as the clinical history goes, there is much similarity between the two cases, and it is not difficult to believe that both may have been due to the same pathological process, but that in the last the inflammatory action was more severe and more extensive than in the first. Beyond the difference in intensity, the symptoms in both cases were very similar. In the second there was a paralysis of the bladder, a condition not present in the first.

Now, what is the nature of this pathological