In spite of the facts that there is much wasting of muscles, and that the reflexes are all absent, it is quite easy to eliminate all the causes of pseudo-paraplegia, of which these facts would at first make us think. Let us briefly mention the causes in order. The dystrophies are evidently put out of question here, by the patient's age and sex, by the absence of any similar cases in the family, by the distribution of the muscular wasting, and above all by the pronounced sensory disturbances. Affections of the peripheral nerves never cause such widespread contractures or such profound sensory loss; there is further no tenderness over any nerve, nor has there been any pain. Acute affections of the anterior horn are not to be thought of, for the onset here was gradual. Chronic affections of the anterior horn are equally easy to exclude, for not only is progressive muscular atrophy rare in a girl of this age, but its onset is localised, and it is not accompanied by marked sensory loss.

It is unlikely that the condition is one of extrinsic paraplegia, i.e., due to pressure on the spinal cord, for the three cardinal signs of this affection, namely, root pain, unilateral onset, and precedence of motor over sensory symptoms, are all absent. An intrinsic paraplegia, due to a local lesion in the cord, is more difficult to exclude, and a diagnosis of it might readily, but erroneously, be made in this case. The following considerations, however, speak strongly against it. To produce such profound sensory changes, the lesion would have to be very severe, indeed practically complete, and it is difficult to conceive of such a lesion existing without ever causing incontinence of urine or fæces. Again, as the sensory loss extends up to the level of the sixth dorsal nerve, it is incongruous that there is no trace of weakness of the abdominal or back muscles, for extensive local lesions implicate the motor fibres to a greater extent than the sensory ones. It is hard also to picture the nature of any local lesion that could cause the symptoms present. Thrombosis and myelitis have either an acute or rapid onset, and not a very gradual one, as was here the case, while against the idea of a tumour speaks not only the absence of any tumour elsewhere in the body, in spite of the long duration, but also the marked improvement that has recently taken place.

Most of the diffuse degenerations of the spinal cord can be excluded here by the absence of other signs that accompany these conditions. For instance, with Friedreich's ataxia, there would be an early onset—before puberty—a hereditary history, the presence of nystagmus, optic atrophy, or choreiform tremor. Amyotrophic lateral sclerosis would have caused an increased