The consideration of the parts paralysed enables us to fix pretty accurately upon the seat of lesion. Thus, neither the upper extremities nor any of the muscles engaged in respiration are involved; the lesion, therefore, must be below the last dorsal vertebra. The first lumbar gives off the ilio-hypogastric and ilio-inguinal nerves; the second, the genito-crural and external cutaneous; the third, the anterior crural, dividing into the middle cutaneous, the internal cutaneous, and the long saphenous; the fourth, the obturator, supplying the adductor muscles. Now, not only was there in this case no paralysis of the cutaneous nerves, but, even whilst unable to use the leg in any other way, the patient retained the power of bringing it towards the middle line, showing that the obturator was unaffected. Part, however, of the fourth lumbar, with the fifth lumbar and the first four sacral nerves, unite to form the great sciatic, the small sciatic and the pudic nerves, supplying not only most of the muscles of the leg and foot, but also the accelerator urinæ; whilst a branch of the fourth sacral supplies the sphincter ani. This muscle is also supplied from the inferior hæmorrhoidal branch of the pudic nerve. The sphincter vesicæ is supplied mainly from the sacral plexus, derived chiefly from the four upper sacral nerves. Both the sphincter vesicæ, and the external and internal sphincter ani, the latter especially, derive some of their nervous supply from the hypogastric plexus of the sympathetic; and this plexus, again, is mingled with nerves from the fourth and fifth lumbar ganglia and the four upper sacral ganglia, with nerve connections with the fourth and fifth lumbar and the four upper sacral nerves. The seat of lesion is therefore tolerably plain. It is unilateral, confined to the left side, and situated not above the origin of the fourth lumbar nerve.

Such, then, being the mode of access, and such the position of the lesion, what is its nature? It cannot be spinal meningitis, for this lesion is not accompanied by paralysis; nor, does the patient lie in any peculiar position, showing an instinctive dislike to being moved from fear of pain in the back and limbs that such movements would cause. It cannot be myelitis, though myelitis sometimes follows hæmorrhage, because the access was so sudden, the paralysis so one-sided; and there is no priapism. It cannot be congestion of cord, for, again, the mode of access is too sudden, and congestion only leads to very partial paralysis, and that of a paraplegic form.

I would say in all fairness that Professor Leyden throws doubt on the existence of spinal congestion as a lesion causing symptoms, from the difficulty in verifying it by post-mortem observation. Though doubtless congestion is difficult of proof, it is equally impossible to disprove; and the transient nature of the paralysis supposed to follow it, and its recovery under remedies known to influence the calibre of the vessels, such as ergot, belladonna, strychnia, etc., are reasons for accepting the real existence of this lesion.

The absence of tonic spasm, and the presence of paralysis, prevent any thoughts of tetanus. There is no tenderness down the spine; and this symptom is never absent in the so-called spinal irritation. Here again the paralysis is a diagnostic mark. The paralysis would be at once too sudden and too presistent to depend on pure shock. Under such circumstances the symptoms would scarcely be unilateral; and did they occur with such intensity from shock, whether they may mean spinal congestion, spinal anæmia, or some peculiar cell-change of a temporary nature, they could not well presist for several weeks, unless the shock had determined myelitis, meningo-myelitis or hemorrhage.

Locomotor ataxy differs from the lesion before us in that its progress is extremely gradual; it is accompanied with no true paralytic symptoms affecting either the limbs or the sphincters until a very long time has elapsed, if ever; and it is manifested by a want of co-ordination that is absent in our patient. The ocular phenomena, also, so frequently met with in locomotor ataxy, are wanting in the case before us.

The suddenness of the access of paralytic symptoms, with the marked improvement of the patient under treatment, entirely forbids the idea of tumour of the spinal cord. And lastly, the absence of reaction in the paralysed muscles to galvanic stimulus is sufficient proof that the lesion is spinal, and not cerebral.