

LETTER TO THE EDITOR

Assessment of the sympathetic level of lesion in patients with spinal cord injury

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We have read with great interest this challenging study by Previnaire *et al.*¹ evaluating the completeness of sympathetic injury in 34 tetraplegic and 47 paraplegic patients using vasomotor responses (skin axon-reflex vasodilatation (SkARV)). Applying mechanical stimulation to the skin on both sides of the trunk, the sympathetic level, the sympathetic completeness of injury and the 'excitability' of the isolated spinal cord were assessed. The authors conclude that there was an 'excellent correspondence between ASIA A and a complete sympathetic lesion, which was always found at or below the lesion.'

After reading this article carefully, we would like to raise some questions and add a few comments concerning the design and the validity of the study's outcome.

The documentation of the impact of spinal cord injury (SCI) on the remaining autonomic function is the focus of research. The balance between sympathetic and parasympathetic components in patients with SCI and the degree of functional alterations within the autonomic system is still uncertain.² In the discussion part Previnaire *et al.*¹ agreed that decreased SkARV below the lesion cannot clearly be assigned to an increased vasoconstriction or reduced vasodilatation.

Complete sympathetic injury was defined by occurrence of 'an abnormal response below the lesion,' which could either be a vasoconstrictor response in upper motor neuron lesions or a total absence of SkARV in lower motor neuron lesions. As the authors also defined an absence of the sympathetic level of lesion when SkARV extended across the lesion down to T12, a small part of the study population was assumed to have 'no sympathetic level of lesion.' The possibility of an incomplete sympathetic level of lesion, the role of the parasympathetic counterpart and the detail that 34% all

ASIA A patients had a 'sympathetic zone of partial preservation' have not been discussed.

Unfortunately, the authors neither give reasons why traumatic (79 patients) and non-traumatic SCI (2 patients) were included, nor reported on the exact origin of SCI. Further, the time interval between the performance of the initial SkARV test (after the acute phase), the ASIA and the day of injury (1, 3, 6, 12 months after injury?) would have been interesting. Without taking into account spontaneous neurological recovery in complete tetra- or paraplegic patients, the conclusion that complete sympathetic and somatic lesions are associated, is questionable.³

Despite raised questions, we praise the efforts that have been made by Previnaire *et al.*¹ We found the SkARV test useful in clinical practice and easy to perform. However, it would be more appropriate to retest SkARV and ASIA after certain time intervals and to clarify definitions of autonomic disorders in patients with SCI.

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