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LETTER TO THE EDITOR

Is the outcome in acute spinal cord ischaemia different from that in traumatic spinal cord injury? A cross-sectional analysis of the neurological and functional outcome in a cohort of 93 paraplegics

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Pouw *et al.* stress that any interventions to assess the recovery following a spinal cord injury by a variety of therapies must be based on a homogeneous group of patients who are considered to have the same neurological and functional recovery.[1]

They have selected two series of patients for comparison. Patients with traumatic spinal cord injuries and patients presumed to be suffering from acute spinal cord ischaemia. The criteria of selection being an acute neurological deficit, attributable to non-traumatic spinal cord lesion and computed tomography or magnetic resonance imaging findings that are typical of ischaemic lesion, excluded alternative diagnosis by means of cerebrospinal fluid examination. The origins of acute spinal cord ischaemia syndrome were idiopathic ($n=7$), aortic dissection ($n=6$), aortic aneurysm repair ($n=3$), embolism ($n=3$) and arteriovenous fistulae ($n=1$). I would question whether the seven idiopathic cases should be included.

Historically and before the advent of aortic surgery, ischaemia of the spinal cord comprised a miscellaneous group of causes and it was only when aortic surgery began to be performed that the relationship of infarction of the cord with the blood supply was established.[2]

It is necessary to make a positive diagnosis. Computed tomography and magnetic resonance imaging scans can only place lesions anatomically, and cerebrospinal fluid examination and other tests can only exclude other known causes of disease; further causes of spinal pathology are being regularly discovered.

When I came to look into this problem in 1974, in a study substantiated by two post-mortems, it was important to establish that the spinal cord infarction followed directly upon a fall in the perfusion pressure of the cord, leading to an immediate paralysis. This was in spontaneous causes of spinal cord ischaemia caused by coronary disease or suicide.[3]

Subsequently in 1994, I looked further at four cases in which the fall in the blood pressure had been produced iatrogenically by anaesthetists to produce a bloodless field. In these cases, the spinal cord lesion was immediately and directly related to the time of the episode of hypotension and what is more, the lesion was in the distribution of the major artery of Adamkewicz. These patients when they survived had a very substantial return of function.[4]

Failure to observe these criteria can lead to mistakes in diagnosis, and in the first series, one of the patients who was referred was considered to be suffering from a bilateral sciatic nerve palsy, after he attempted suicide inside the toilet and infarcted his cord because of a fall in blood pressure.[3] A recent case was diagnosed by magnetic resonance imaging as having a spinal cord infarction, and in this case, there had been excessive traction of the spinal cord from a surgical manoeuvre, and there had been no fall in the blood pressure and on this basis infarction of the cord had been misdiagnosed.[5]

Strict criteria should be adopted. Interestingly, in three of the studies, which the authors refer to, also based on the magnetic resonance imaging findings, 7/20, 9/28 and 17/28 are idiopathic cases, which are very similar to Pouw *et al*'s findings of 7/20.

JR Silver
8 High Street, Wendover, Buckinghamshire, UK
E-mail: Jrussellsilver@btconnect.com

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