Secondary neurological deterioration in traumatic spinal injury

DATA FROM MEDICOLEGAL CASES

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We assessed the frequency and causes of neurological deterioration in 59 patients with spinal cord injury on whom reports were prepared for clinical negligence litigation. In those who deteriorated neurologically we assessed the causes of the change in neurology and whether that neurological deterioration was potentially preventable. In all 27 patients (46%) changed neurologically, 20 patients (74% of those who deteriorated) had no primary neurological deficit. Of those who deteriorated, 13 (48%) became Frankel A. Neurological deterioration occurred in 23 of 38 patients (61%) with unstable fractures and/or dislocations; all 23 patients probably deteriorated either because of failures to immobilise the spine or because of inappropriate removal of spinal immobilisation. Of the 27 patients who altered neurologically, neurological deterioration was, probably, avoidable in 25 (excess movement in 23 patients with unstable injuries, failure to evacuate an epidural haematoma in one patient and over-distraction following manipulation of the cervical spine in one patient). If existing guidelines and standards for the management of actual or potential spinal cord injury had been followed, neurological deterioration would have been prevented in 25 of the 27 patients (93%) who experienced a deterioration in their neurological status.

Cite this article: Bone Joint J 2015;97-B:527–31.

Patients with traumatic spinal injury may have any level of neurological deficit at the time of injury, ranging from none through to complete motor and sensory paraplegia. A major concern in the initial management of spinal cord injury (SCI) patients is to immobilise the spine to prevent loss of, or further loss of, neurological function due to excessive movement at the level(s) of the unstable skeletal injury. There are reports that 3% to 25% of SCI patients deteriorate after the primary traumatic event.1-3 Up to 20% of spinal injuries are at multiple, non-adjacent levels, so the whole spine is potentially at risk.6-8 There has been widespread adoption of protocols for the routine spinal immobilisation of all actual or potential SCI patients9-14 with some evidence that such protocols lead to less severe cord injuries on first assessment in emergency departments.15 However there has also been criticism of routine spinal immobilisation for all trauma patients16-19 and some authors advocate selective spinal immobilisation based upon triage systems.20-22

We have reviewed the reports we have provided for the Court on patients with traumatic SCI, in order to determine the frequency and causes of neurological deterioration in these patients. We wished to establish the causes of the neurological deterioration and to see whether there were new lessons to be learned, or old lessons relearned, in the management of SCI patients.

Patients and Methods

All three authors prepare medicolegal reports for the purposes of potential litigation. We retrospectively reviewed our reports prepared between 2001 and 2011. The reports were based upon review of the medical records, witness statements and the radiological imaging. No patient was excluded. Age, gender, level of skeletal injury, nature of the injury, Frankel grade23 on first assessment, whether neurological deterioration had occurred, if there was any neurological deterioration, the Frankel grade after deterioration and the probable cause of neurological deterioration were recorded. In some patients analysing the evidence in relation to neurological function was straightforward. For example, there were a number of patients who were said to have ‘normal power’, ‘full limb movement’, ‘power = 5/5 L = R’ (MRC grading scale24) who deteriorated to areflexic motor and sensory paraplegia. In other patients there was incomplete or conflicting data. We dealt with that as follows: if there was a Court decision in relation to the
evidence we accepted that decision; if there had been agreement between the medical experts advising the Court on the case (for both sides) we accepted that decision; if neither of those applied we came to a consensus decision between the three authors. We looked to see whether there might be any other cause of neurological deterioration such as hypotension or hypoxia.

Results
There were 59 reports prepared for potential clinical negligence litigation; in two patients there were partial missing data. The age range was 5 to 88 years, the median age by decile was 51 to 60 years. There were 49 men and ten women (83% men). The levels of skeletal injury are set out in Figure 1. The injuries were cervical (n = 54, 92%), thoracic (n = 3, 5%) or lumbar (n = 2, 3%). The most common level of injury was the C6 cord level (C5-6 skeletal level) in 15 patients (25%). The type of spinal injury and the nature of the cord injury are shown in Table I.

The initial and subsequent Frankel grades are shown in Table II. The 'subsequent' Frankel grade was not a late Frankel grade, post-rehabilitation, it was an early post-injury Frankel grade assessed within seven days of the primary injury (as far as was possible from the clinical notes). All the patients who deteriorated, did so because of increased severity of the neurological lesion and no patient with a primary cord injury deteriorated because the level of the injury ascended. In total, 27 patients (46%) deteriorated neurologically, on clinical grounds; of those, 20 (74%) of those who deteriorated had no primary neurological deficit, four (15%) were Frankel D following the primary injury, that is they would probably have become long-term walkers, three (11%) were Frankel C, who would possibly become walkers with rehabilitation. The secondary loss of neurological function was therefore typically profound with 24 patients (89% of those who deteriorated) becoming wheelchair-dependent who would probably have been able to walk without the deterioration, with a further three (11%) who would possibly have recovered to the point of independent walking. Of those who deteriorated, 13 (48%) had complete motor and sensory paraplegia, Frankel A.

The causes of neurological deterioration are shown in Table III. Neurological deterioration occurred intra-operatively in one patient; he was Frankel D pre-operatively but became Frankel grade A post-operatively; the cause was not identified. Electrophysiological monitoring was not used. One patient with a Frankel C central cord syndrome deteriorated, for no known cause, to Frankel A. Neurological deterioration occurred in 23 (61%) of 38 patients with unstable fractures and/or dislocations. The consensus opinion of the authors, based on our interpretation of the medical records and the chronological developments, is that all 23 patients deteriorated because of excessive movement at the level of the unstable fracture and/or dislocation. Of the 38 patients with unstable fractures and/or dislocations, four were initially Frankel A. These patients could not develop a more severe lesion (although the level of cord injury could ascend); these patients would also not be expected to recover function. Excluding these four, there were 34 patients with unstable
injuries who could change neurologically, thus 23 of 34 patients (68%) of those patients who could deteriorate neurologically, did so. Of the 27 patients who deteriorated neurologically, neurological change was, probably, avoidable in 25 (93%); excessive movement probably occurred in 23 patients (85%) with unstable injuries, in one patient the cervical spine was manipulated and over-distracted, and one patient with an epidural haematoma was not treated. In those patients who deteriorated we could find no evidence that there was another cause for neurological deterioration such as for example hypotension or hypoxia.

Discussion

The patients reported on in this paper are a highly selected group who are not representative of SCI patients as a whole. This is because of two selection biases; the first is that these were the SCI patients who engaged in litigation and therefore they have amongst the worst, non-lethal, outcomes of all SCI patients. The second is that there may well be a selection bias, or biases, in the way in which the three consultants were chosen by their legal representatives. One example of the first bias is that there were initially only four Frankel A paraplegic patients (7%), while normally there would be about 50% of SCI patients with Frankel A paraplegia as a consequence of the primary injury.23 The majority of Frankel A patients do not recover; they remain Frankel A (the rare exception is the reversible, initially complete SCI, the ‘spinal shock’ patient).23 Whatever their medical management, a Frankel A patient is unlikely to be able to demonstrate that they have been harmed neurologically, in terms of the severity of the cord injury. This compares with Frankel E, D or C patients who, if they do deteriorate, have potentially lost a great deal of neurological function. The financial implications of loss of neurological function are potentially large and such patients will be over-represented in a study such as this. Also in this study cervical injuries were over-represented as normally about 40% to 55% of SCI patients have cervical spine injuries.23,26,27 The cervical spine is the most mobile part of the spine and any failure to immobilise the cervical spine might cause injury to the spinal cord. In addition, neurological deficits in the cervical spine are likely to result in much more profound disability than more distal injuries. Patients with spinal cord injury can deteriorate neurologically in two ways (i) an increase in the level of cord injury or (ii) an increase in the extent and severity of cord injury. In a patient with complete motor and sensory paraplegia (Frankel A) the level of the cord lesion can ascend in the acute phase typically one cord level, for example a C6 cord level becomes C5 with the level of subsequent cord injury descending over time to the original cord level.23 In patients with SCI the severity of cord injury can increase, for example Frankel E patients can deteriorate to incomplete SCI (Frankel D, C or B) or complete (Frankel A). Patients with incomplete SCI (Frankel D, C or B) can deteriorate to more severe cord injury (Frankel C, B or A). In our study all of the patients who deteriorated had an increase in the extent of their cord injury and no patient with a primary cord injury had ascent of the cord injury to a higher level.

The probability of an SCI patient deteriorating neurologically is low, 0% to 5%.26-29 Frankel et al26 described 682 patients admitted within 14 days of spinal injury to a single centre. Six of these patients deteriorated neurologically which is 0.88% of all 682 patients; it is 2.1% of those (Frankel E to B) patients who could deteriorate neurologically. In Frankel’s series there were 218 cervical injuries, and neurological deterioration occurred in four patients (1.8%), three patients who were admitted Frankel B deteriorated to Frankel A, one patient admitted Frankel C deteriorated to Frankel B, but no patient whose neurology was Frankel C or better deteriorated. Sapkas and Papadakis29 reported on 67 patients with a cervical fracture or dislocation who required surgical stabilisation and they found that no patient deteriorated neurologically. As expected, the motor and sensory paraplegic patients (Frankel A) of whom there were 20, remained paraplegic in the long-term; also as expected, incomplete SCI lesions tended to improve.29 Marshall et al27 reported on 283 spinal cord injured patients who were consecutively admitted to five trauma centres and who were assessed prospectively. A total of 14 patients (5%) deteriorated neurologically. Of those 14 patients, 12 (86%) deteriorated almost certainly because of movement of the spinal cord at the level of an unstable injury; three deteriorated when skeletal traction was applied, two deteriorated when a rotating frame was moved, four deteriorated shortly after surgery, two deteriorated when a halo was being applied and one deteriorated because of progressive subluxation despite external immobilisation. In one patient there was neurological deterioration that was apparently a consequence of severe metabolic imbalance. This large prospective study suggests that (i) ‘spontaneous’ neurological deterioration in patients with an incomplete spinal cord injury is very uncommon (1 of 283 patients) and (ii) neurological deterioration is most commonly associated with a specific event, such as excessive movement at the site of a fracture and/or dislocation. Toscano30 reported on 123 patients prospectively admitted to a single spinal injuries unit; 32 patients sustained major neurological deterioration between the injury and admission to the spinal injuries unit (26%). In all patients the cause of neurological deterioration was a failure to immobilise the spine. Poonnose, Ravichandran and McClelland31 retrospectively reviewed 569 spinal injury patients. The diagnosis of spinal cord injury was missed in 52 patients (9.1%). Of those 52 patients, 26 (50%) suffered neurological deterioration. All of these papers support the concept that the most common cause of clinically observed neurological deterioration is excessive movement of an unstable spinal skeleton injury. In our study 27 of 59 patients (46%) deteriorated neurologically. Of those patients who had an unstable fracture and/or dislocation and who had the potential to deteriorate neurologically,
secondary neurological deterioration occurred in 23 of 38 patients (61%). We found that the rate of neurological deterioration was much higher than in the clinical series, with a rate, overall, of 46%. Spontaneous neurological deterioration, for which no cause was identified, occurred in only one of 59 patients (2%) which is comparable with other studies. Overall, secondary neurological deterioration was probably avoidable in 25 of the 27 patients (93%). However, our rate of neurological deterioration cannot be compared with the available literature because of the selection bias in favour of those who deteriorate. Nevertheless, the causes of neurological deterioration are highly relevant and, as in so many of the previous studies, the overwhelming cause of neurological deterioration was a failure to maintain spinal immobilisation. In our study all 23 of the patients with unstable fractures and/or dislocations who deteriorated, immobilisation of the spine was not performed, or collars and/or blocks and/or tapes were removed and the patients were mobilised prior to radiological clearance of the spine. 

There are other causes of neurological deterioration in patients with acute spinal cord injury. Respiratory insufficiency, pulmonary dysfunction and systemic hypotension are common in SCI patients. In severe acute SCI there can be loss of spinal cord autoregulation and in these circumstances hypotension and/or hypoxia can potentially aggravate the primary neurological injury, or limit the potential for recovery. In experimental SCI secondary events such as oedema, microcirculatory failure, ion shifts, calcium or excitotoxic amino acid-mediated damage have been shown to cause neurological deterioration. Patients with spinal cord injury can have physiological instability and 5% to 10% of SCI patients will deteriorate neurologically if sat up; typically the neurological deficit reverses if the patient is laid flat. In our study 26 patients had evidence of spinal cord injury on first assessment of whom seven deteriorated neurologically. There remains the possibility that neurological deterioration in these patients could have been a consequence of physiological instability. However, even if we assume a rate of physiological instability of 10% in spinal cord injured patients this would only account for neurological deterioration in one of these seven patients. In our study once neurological deterioration was recognised all patients were laid flat, however none recovered any function. It is probable that physiological instability did not account for neurological deterioration in more than one of our 27 patients who deteriorated neurologically.

There has been criticism of a blanket policy of immobilising the spine. If an ankylosing spondylitis patient with an unstable fracture has a pre-existing kyphotic deformity, placing such patients into a cervical collar (or other type of cervical immobilisation) can lead to considerable movement of the cervical spine from the longstanding kyphotic deformity into a neutral position which can cause neurological deterioration. This has led to the modified concept that the cervical spine should be immobilised ‘in appropriate line’ that is in the appropriate line for that patient, which is not necessarily in all patients the normal neutral position. It is possible that the application of a cervical collar can raise intracranial pressure (ICP) and there is also an increased risk of pressure ulceration to the skin from the cervical collar, as well as respiratory problems from immobilisation. Whole body immobilisation can reduce tidal volume by an average of 15%, cervical immobilisation can increase the risk of aspiration, cervical immobilisation limits the view of the airway during laryngoscopy and there is an increased rate of failure of intubation. Some have suggested that the routine spinal immobilisation of all trauma patients has no firm evidence base and it is necessary to be more selective in the approach to spinal immobilisation in trauma patients. However, similar to other studies, our findings have shown that the most common single cause of neurological deterioration in patients with traumatic SCI is excessive movement at the level of an unstable fracture and/or dislocation. The most common cause of this excessive movement is either a failure to immobilise at all or removal of the immobilisation devices prior to clearing the spine radiologically. It is our view that the spine should be immobilised in all patients who have clinical evidence of spinal injury, where the mechanism of injury exposes the patient to a risk of such injury or where the spine cannot be cleared on clinical grounds. Although there are potential disadvantages to spinal immobilisation they certainly do not compare to a patient suffering a lifetime of avoidable neurological disability due to deterioration in spinal cord function.

In many ways it is regrettable that there were no new lessons to be learned but there is one in particular to relearn. The cause of neurological deterioration in our study was, overwhelmingly, the failure to immobilise the unstable spine, over-reduction/over-distraction in one patient with an unstable injury and a failure in one patient to evacuate an extradural haematoma. Prevention of neurological deterioration, in potential/actual SCI patients, is the application of principles that are long established, well-recognised and need to be rigorously applied. The costs that are associated with spinal injury are dependent upon the level and severity of the spinal cord injury, the patient’s age and the availability of high quality rehabilitation. Whether the patient returns to work is dependent upon these factors and is also influenced by the patient’s previous educational achievements. It was not possible in this study to quantify the economic impact of spinal injury in each individual.
patient. In Australia the current lifetime cost of paraplegia is £2 700 000, for quadriplegia it is £5 100 000. Using an average of these costs for the 25 patients whose neurological deterioration was probably avoidable, the cost of the harm was £3 900 000 per patient, £97 500 000 overall.

Author contributions:
N. V. Todd: Study design, Data collection and analysis, Writing the paper.
D. Skinner: Data collection and analysis, Writing the paper.
J. Wilson-MacDonald: Study design, Data collection and analysis, Writing the paper.
No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

This article was primary edited by S. Hughes and first proof edited by G. Scott.

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