

Editorial I

Spinal cord injury and direct laryngoscopy – the legend lives on

In 1989, Rosen and Wolfe questioned the prevailing dogma that direct laryngoscopy was an unsafe procedure in patients with unstable spines. They suggested that it was in fact a legend, arising mainly from the natural anxieties of physicians.¹ No case reports of spinal cord injury (SCI) at laryngoscopy could be found, nor any evidence to show that the method of intubation made any difference to outcome. In the decade since then, there have been five reports, dealing with nine patients, which allege that SCI resulted from tracheal intubation involving direct laryngoscopy.^{2–6} In addition, there has been one report incriminating airway management, when direct laryngoscopy was one of the procedures performed.⁷ Is Rosen and Wolfe's 'legend' now fact? Before giving an account of these case reports (where, notably, most of the authors were not anaesthetists), it may be helpful to consider what is currently known about the movements of the cervical vertebrae during intubation, and some experimental and clinical evidence about the induction of a myelopathy.

Most anaesthetists and surgeons probably believe that the risk of SCI during direct laryngoscopy is largely a matter of mechanical compression. Direct laryngoscopy can appear dramatic, even brutal, and it is not surprising that there is concern over the safety of the technique in patients with cervical spine disease, whether the spine is stable or not. The procedure has been accused of causing 'maximal movement and extension of the entire cervical spine' in one of the above reports.² However, two studies have concluded that in normal patients and volunteers there is minimal movement below C3 during direct laryngoscopy;^{8,9} this finding is supported by the reduced prevalence of difficult laryngoscopy found in patients with cervical disease below C3.¹⁰ Since cervical movement during laryngoscopy is concentrated at the occipito-atlanto-axial complex, it would seem likely that spinal disease at this level is the most dangerous in terms of the risk of SCI. It can be argued that the amount of movement in the cervical spine from C3 to C7, resulting from laryngoscopy, might be different in unstable spines. It will always be difficult to study the unstable spine, and our knowledge of its movements during intubation (whether the patient is awake or asleep) remains limited.

So far, investigations have been confined to cadavers. Donaldson and colleagues studied the movements at the C5–C6 joint before and after surgical destabilization.¹¹ They found a difference of <2 mm translation and 4° of angulation between the stable and unstable specimens at direct laryngoscopy. Earlier radiographic studies of unstable cervical spines showed that other airway manoeuvres, such as jaw thrust, caused similar degrees of movement.¹² If the deformations of the spinal canal are indeed so small, can an SCI be produced during the few seconds or minutes that is required for direct laryngoscopy?

The development of a myelopathy is believed to result from a combination of mechanical deformation and vascular compromise and, unless the deformation is extreme, the duration of the unfavourable conditions is likely to be a crucial factor.¹³ Experimental evidence from animal models of SCI indicates that minor or moderate degrees of compression require some time to produce lasting injury. Dolan, Tator and Endrenyi demonstrated a predictable relationship between the force of cord compression, its duration and the resultant neurological injury.¹⁴ When compression sufficient to produce 50% constriction was applied around dogs' spinal cords, Delamarter, Sherman and Carr found that ≥6 h of constriction led to permanent loss of function, whereas after ≤1 h of constriction there was nearly complete recovery (although initial neurological impairment was noted in all the dogs).¹⁵ Compression of the cord during laryngoscopy has not been quantified in relation to the movements that occur, although some evidence concerning probable qualitative changes is available. Flexion of the spine causes elongation of the cord with narrowing of the diameter of the longitudinal vessels.¹⁶ Extension causes an increase in diameter of the cervical cord and folding of the ligamentum flavum, which may exert pressure on the cord and the posterior longitudinal vessels.¹³ Flow through the radicular vessels is also believed to be obstructed during cervical movement.¹³ Dinsmore, Bacon and Hollway have shown that alteration of spinal curvature can increase CSF pressure (and reduce cord perfusion pressure) by 20 mm Hg.¹⁷ All of these effects might be accentuated in the injured or diseased spine. Magnaes, in a remarkable series of investigations on patients with spinal stenosis, showed

that alteration of head position in simulated laryngoscopy could obstruct CSF flow around the cord.¹⁸ The patients would almost certainly have needed to increase their perfusion pressure to avoid cord ischaemia, but none of them came to harm. It is tempting to suggest that hypertension, which is a notorious feature of direct laryngoscopy, represents a safety factor in patients with cervical spine disease. The contribution of vascular compromise to SCI during anaesthesia has probably been underestimated, while the consequences of spinal movement have been overemphasized. Forty per cent of patients with cervical fractures or dislocations have vertebral artery injuries,¹⁹ and reports of neurological injury induced by cervical manipulation have mostly been of a vascular type of lesion, such as posterior inferior cerebellar artery syndrome.²⁰

There is no doubt that hypotension²¹ and prolonged malposition can cause cervical cord damage, even in normal patients. Levy reported a case of a man who developed a cervical myelopathy after being tied up in flexion for 12 h.²² Merli and Staas reported three cases of myelopathy after abnormal body positioning, including one man who spent 8 h sitting with his neck flexed while drunk.²³ Dominguez and colleagues reported the case of a young woman who underwent surgery for the excision of tracheal rings.²⁴ At the end of the procedure, her chin was stitched to her chest to prevent tension on the tracheal repair. The patient had normal neurology on recovery, but after 8 h she developed an irreversible tetraplegia.

The patients described above suffered neurological damage after major degrees of malpositioning, but there is clear evidence that SCI can occur even when malpositioning is minor. We believe that the case reported by Deem and colleagues²⁵ is important. They described a man with cervical spondylosis, who required surgery on his lumbar spine. The patient was intubated and then positioned while awake, and normotension was maintained, but at the end of a 6 h procedure the patient was found to have sustained a cervical myelopathy of the central cord type. We have little doubt that, had Deem and colleagues not taken the precaution of intubating the patient awake, the lesion would have been blamed on direct laryngoscopy. Linstedt and colleagues described a 12-yr-old patient, with significant cervical stenosis at the C1 level as a result of mucopolysaccharidosis type IV, who underwent facial surgery for 11 h.²⁶ The patient was carefully positioned with the head and neck in a neutral position. Three hours into surgery, the cortical sensory evoked potentials (SEPs), which were being used to monitor spinal cord function, suddenly disappeared. The patient's head was slightly anteflexed and the signals returned to normal. Bejjani and colleagues described a patient known to have cervical stenosis and spondylosis, who was undergoing cerebral angiography.²⁷ The patient's head had been strapped in place, and her complaints that the position was uncomfortable and that her arms were becoming paralysed were treated with diazepam. Cervical myelopathy appears to have started about 45 min after the

procedure began. There have been reports of myelopathy occurring in the lumbar and thoracic regions, which have been ascribed to prolonged spinal malposition.²⁸ A feature common to almost all of these cases is the relatively lengthy period of time that elapsed before the onset of critical spinal cord ischaemia. The immobility induced by general anaesthesia is a highly abnormal condition; it is probably no accident that human beings rarely stay still for long, even in sleep.

Proving that direct laryngoscopy was the cause of an SCI is problematic. Direct laryngoscopy is followed by a period of unconsciousness, during which neurological assessment will be more or less impossible. Alternative possible causes of SCI, such as hypotension and malpositioning, may occur during the period of unconsciousness, but it is also hard to prove that direct laryngoscopy did not cause an SCI. The best we can hope for is likely to be a diagnosis based on a balance of probabilities. We have tried to identify features that would give weight to a diagnosis of direct laryngoscopy-induced SCI. The principal symptoms and signs of acute cervical myelopathy are weakness of the limbs, sensory abnormalities and areflexia; although these will be difficult to assess until the patient is fully conscious, we suggest that these symptoms and signs should be present on recovery. A short period of unconsciousness, which would not allow time for other causes to occur, might also be regarded as evidence that direct laryngoscopy was the cause of SCI. Signs reflecting damage to the autonomic nervous system, such as hypotension, bradycardia and dysrhythmias, which are seen in animal models,^{29 30} might be observed after a laryngoscopy that has caused SCI. However, autonomic disturbances are not seen in all myelopathic victims of cervical trauma,³¹ so the absence of these signs cannot exclude the diagnosis. The nature of the laryngoscopy must also be considered relevant. It seems probable that an easy laryngoscopy with minimal forces applied, is less dangerous than a difficult one. Disease or instability at the cranio-cervical junction, or gross instability below C3, could also be considered a risk factor. Features tending to add weight to the diagnosis of laryngoscopy-induced SCI could, therefore, include: (i) a myelopathy present on recovery; (ii) a short period of unconsciousness; (iii) autonomic disturbances following laryngoscopy; (iv) difficult laryngoscopy; and (v) cranio-cervical junction disease, or gross instability below C3 (see Table 1). The case reports alleging that SCI resulted from direct laryngoscopy can now be described, and examined for convincing evidence.

Farmer and colleagues⁵ reported a series of patients whose neurological condition had deteriorated after an initial injury. They stated that four cases had deteriorated after intubation, but so little information was supplied that it is not possible to assess their report. Farmer and colleagues stated that, in some cases, the patients had deteriorated without any obvious cause. Presumably this phenomenon could coincide with a period of anaesthesia. Redl,³ Yan and

Table 1 Features given in the text which appear in the referenced case reports (1 = myelopathy on recovery; 2 = short period of unconsciousness; 3 = autonomic disturbances after laryngoscopy; 4 = difficult laryngoscopy; 5 = cranio-cervical junction disease or gross instability below C3). *Time of onset not given; †cardiac arrest before intubation; NA, not applicable; NR, not reported

Reference	Feature				
	1	2	3	4	5
Farmer and colleagues	NR	NR	NR	NR	NR
Redl ³	no	no	no	no	yes
Yan and Diggan ⁴	*	no	NR	NR	no
Muckart, Bhagwanjee and van der Merwe ²	yes	no	no	no	no
Yaszemski and Shepler ⁶	NA	NA	†	NR	yes
Hastings and Kelley ⁷	yes	yes	yes	yes	no

Diggan⁴, and Muckart, Bhagwanjee and van der Merwe² stated in the titles of their articles that cord damage was a result of intubation. Redl reported the case of a young man suffering from spondyloepiphyseal dysplasia congenita, who had a severe cranio-cervical abnormality, which was not diagnosed pre-operatively.³ The patient was intubated easily for an orthopaedic procedure on the lower limbs. The patient had no abnormality on recovery but by the first postoperative day had developed a spastic tetraparesis. The patient described by Yan and Diggan⁴ required intubation for AIDS pneumonia, had a normal cervical spine and had neurological abnormalities before intubation. No details of the intubation or intensive care sequence were given, and the patient was found later to have a myelopathy of the central cord syndrome type. An MRI scan was said to show changes consistent with cord contusion, at an unstated level. Muckart, Bhagwanjee and van der Merwe described two patients with undiagnosed cervical fractures, who had surgery for other injuries.² The cervical injuries were not discovered pre-operatively, so we presume that no particular attention was given to the alignment of these patient's heads and necks during the operations. Yaszemski and Shepler did not state in the title of their report that direct laryngoscopy had caused an SCI, but in the discussion they did suggest that it had been the cause.⁶ Their patient was suffering from rheumatoid arthritis and had reducible atlanto-axial subluxation. A general anaesthetic was administered for hand surgery. Awake fiberoptic intubation was performed and surgery, anaesthesia and recovery were uneventful. Ten hours after the operation, the patient suffered cardiac arrest and had an emergency intubation for cardiopulmonary resuscitation (CPR). Resuscitation was only temporarily successful. A post-mortem examination showed damage to the upper cervical cord and lower medulla. No details of the emergency intubation were provided. Hastings and Kelley⁷ were careful not to claim that the cervical myelopathy, which was found to be present after a failed intubation and cricothyrotomy, was a result of direct laryngoscopy. Their patient had developed airway obstruction because of a cervical haematoma, but also had congenital spinal stenosis, osteophytes from C3 to C7, and undiagnosed

fractures of the facet joint and lamina of C6–C7. A complex myelopathy was found to be present after resuscitation. The suspicious features in these case reports alleging direct laryngoscopy-induced SCI are summarized in Table 1.

In the case described by Redl,³ myelopathy was not present until several hours after recovery, so the author's opinion that it was caused by laryngoscopy is difficult to understand. The opinion of Yan and Diggan⁴ that the SCI was a result of laryngoscopy seems to have been based on an MRI scan report of 'contusion' of the cord. Our neuroradiological advice is that the MRI signs of contusion are not specific, and the changes could equally have resulted from ischaemia (Stevens JM, personal communication, June 1999). It is not unlikely that the patient had circulatory instability during intensive care admission. The only evidence to support the allegation that cord damage was caused by laryngoscopy in the cases described by Muckart, Bhagwanjee and van der Merwe² is that signs of SCI were present when the patients awoke, which is evidence of association rather than causation. It is possible that the alignment of these patients' heads and necks was less than optimal throughout anaesthesia. The case presented by Yaszemski and Shepler⁶ is interesting. It is certainly possible that an SCI occurred during direct laryngoscopy, but it is also possible that the damage occurred during CPR, when movement of the head and neck is often quite violent. Sudden death is an uncommon, but well described phenomenon in patients with atlanto-axial instability, so it is also possible that the cord damage was the cause rather than the result of the patient's cardiac arrest and CPR.³² No sign of a cardiac infarct was found at autopsy. The pathological data presented did not indicate when the cord damage had occurred, beyond saying that the changes must have been *ante mortem*. In the case described by Hastings and Kelley,⁷ there are factors other than the immediate presence of myelopathy to support the diagnosis of laryngoscopy-induced cord damage (hypotension at laryngoscopy and difficulty with laryngoscopy). However, the hypotension may already have been present, and the laryngoscopy failed, necessitating a cricothyrotomy, which might have called for extreme neck extension. Nevertheless, Hastings and Kelley's report is the most convincing, although the authors themselves stated that it was not possible to explain the neurological findings in their patient on the basis of laryngoscopy-induced damage alone. It would be foolish to deny the possibility that direct laryngoscopy was the cause of some of the neurological damage, but the severe hypoxaemia, acidosis and hypotension that resulted from the airway obstruction are confounding factors, and the greatest degree of spinal deformation was likely to have occurred during the cricothyrotomy. With the possible exception of Hastings and Kelley's case, the balance of probabilities suggests that direct laryngoscopy was unlikely to have been the cause of the myelopathies reported.

It cannot be denied that SCI caused by intubation may have occurred and yet remained unpublished or simply

unrecognized, although it is noteworthy that neither of the UK medical defence organizations has settled a claim for this type of accident. However, we do not seek to downgrade the care which must be taken with direct laryngoscopy in patients with cervical disease. Current recommendations concerning manual stabilization and the use of minimal force at laryngoscopy are *a priori* sensible steps to take. Difficult direct laryngoscopy has a definite associated mortality;³³ flexible fibreoptic technology should be preferred if laryngoscopy is or might be difficult.¹⁰ Pre-operative neurological findings, and any precautions taken should be recorded. A record of adequate care at laryngoscopy could be pertinent to later medico-legal proceedings. More important still is to widen the area of concern to include the whole of anaesthesia and the postoperative period. The overriding aim must be to maintain adequate spinal cord blood flow (SCBF).

The regulation of SCBF is believed to be similar to that of cerebral blood flow. SCBF may become inadequate if arterial pressure is low, because autoregulation of blood flow in damaged cords may be absent.²⁹ Spinal cord perfusion should be optimized by preventing hypotension, and some workers feel that an element of induced hypertension is beneficial.³⁴ Conversely, there is also evidence that hypertension may cause increased oedema and haemorrhage in damaged cords,²⁹ so induced hypertension should be carefully controlled. Perfusion pressure may also be inadequate because the pressure inside the spinal canal or dural sac is high. CSF drainage has been used to promote adequate SCBF in vascular surgery,³⁵ but its potential benefits in spinal surgery have not been established. Taking care to maintain favourable spinal posture during anaesthesia is clearly important, but it must be admitted that this can be largely guesswork. Deem and colleagues' report²⁵ demonstrates that even the patient may be wrong about whether a position will be tolerable for an extended period. Monitoring SEP is presently the most practicable way of demonstrating adequate cord perfusion during general anaesthesia. May, Jones and Crockard have used SEP monitoring to identify some risk factors associated with neurological deterioration during surgery on the cervical spine:³⁶ (i) poor pre-operative neurological function (one third of severely myelopathic patients deteriorated); (ii) use of instrumentation (the risk doubled in pre-operatively unimpaired patients); (iii) upper cervical and clival surgery (the risk tripled); (iv) multisegmental surgery (the risk increased with each additional level). There is, however, no good evidence that monitoring SEP makes any difference to outcome in patients with cervical disease, but the continuing presence of evoked potentials is at least enormously reassuring. It can be argued that the awake patient provides the best monitor of cord function, and the use of local anaesthesia and sedation for cervical surgery may be more practical than is generally supposed,³⁷ although sedation can make the patient's neurological status difficult to interpret.³⁸ It is probably true that more use could be made of local anaesthesia and sedation,

but successful cervical surgery in awake patients will require considerable commitment from both the surgeon and the patient.

Rosen and Wolfe's legend almost certainly remains a legend, so careful direct laryngoscopy should continue to be an acceptable procedure in patients with unstable cervical spines.³⁹ However, there can be no doubt that patients with cervical spine disease are at risk of SCI during anaesthesia. Furthermore, it seems that this risk is still present despite ideal anaesthetic care.^{5 25} The evidence suggests that spinal cord perfusion can become inadequate during prolonged periods of immobility, probably as a result of minor degrees of malpositioning.²⁵⁻²⁷ SEP monitoring of cord function is extremely desirable during high-risk cases, and may help to ensure that the combination of position, blood pressure, cervical pathology, surgery and length of surgery is tolerable.

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Editorial II

The effects of regional analgesia on the progress of labour and delivery

Despite an increasing amount of evidence to the contrary, there is still a widespread belief amongst many healthcare professionals that regional analgesia prolongs labour and increases the number of obstetric interventions. With the introduction of epidural analgesia some 30 years ago came concerns regarding an increased need for instrumental delivery. More recently, the focus of attention has switched to a possible association with Caesarean section, primarily that required for dystocia. Although an association between

the use of analgesia and labour outcome exists, a number of recent publications have demonstrated that this relationship is not causative. When examining the effects of regional analgesia on the progress of labour and delivery, study design is crucial, and none of those methods that have been employed is without problems. Retrospective analysis, although frequently cited, is almost worthless, as women do not choose analgesia at random. Consequently, those experiencing prolonged difficult labour, with increased