Indications for cervical spine surgery

Most surgery is designed to relieve compression of the spinal cord or spinal nerve roots. Reduction in the bore of the vertebral spinal or root canals (spinal stenosis) can occur in many conditions, and some patients have congenitally narrow canals. Osteophytic projections at the intervertebral joints (cervical spondylosis) are the commonest problem. Tumours, infection, trauma, disc protrusions, and rheumatoid arthritis are less common causes of canal stenosis. In some of these conditions (notably trauma, infection, tumours, and rheumatoid), stenosis can also be caused by vertebral instability causing or threatening a reduction in volume of the spinal or root canals (Figs. 1 and 2).

Cervical instability

It is difficult to define instability, which is a spectrum ranging from complete failure of intervertebral connection, through abnormalities which result in pain or deformity over a period of years, to completely asymptomatic radiological findings (Table 1).

Asymptomatic cervical instability is seen in rheumatoid arthritis where up to 50% of patients with anterior atlanto–axial subluxation (AAS) may be unaware of the abnormality. Symptoms of AAS include neck, occipital, and facial pain, sometimes lancinating or like an electric shock (L’Hermitte’s phenomenon). Neurological impairment as a result of AAS is characteristically subtle, although sudden death has been described. The question is often asked as to whether symptomless rheumatoid patients should have flexion–extension radiographs before anaesthesia. Although there is no evidence of benefit in terms of survival, it is probably sensible to get the radiographs because of ‘outcome bias’ (see below), and because current opinion recommends early fixation of AAS.

Below C2, a two ‘column’ model (anterior and posterior) is generally used in considering spinal stability. A spine with all the elements of one column intact with at least one component of the other is regarded as functionally stable (though this is not evidence based). The column model is inappropriate for the complex area of the occipito–atlanto–axial (OAA) articulations. The commonest pattern of instability at the OAA complex is anterior AAS, which results from laxity of the transverse and apical dental ligaments. AAS is seen in Down syndrome, Morquio syndrome, rheumatoid arthritis, and infections of the head and neck (Grisel’s syndrome).

The basis of management in instability is immobilisation, or at least reduction of movement, since few immobilisation systems prevent all movement. Immobilisation is a standard practice when cervical instability is suspected, but it must be acknowledged that there is no grade I evidence to support the policy, and that there are potential and actual hazards.1

‘Clearing’ the cervical spine

Considerable practical difficulty arises when patients are admitted with suspected cervical injury. The patient must be immobilised until the spine is ‘cleared’. There are agreed criteria for conscious patients, but the requirement for normal cervical movement may not be satisfied in many cases because of pain (Table 2). In these patients, the collar must be left on (but the anterior portion should be released for airway management). Unconscious patients (50% of patients with a traumatic cervical cord injury also have a head injury) cannot be assessed clinically. They require a radiological opinion on cervical images, but there may be local variation in the methodology.2

Neurological deterioration during anaesthesia

Patients sometimes deteriorate neurologically during the perioperative period. In some cases, the problem will be a surgical misadventure
but, in many, the cause is not obvious. Complete transverse lesions are unusual; most patients suffer an anterior or central cord syndrome. It is difficult to be sure of the causation of these injuries and hence how to prevent future episodes. The rigour of a double-blinded trial is unlikely to be applied to this area of anaesthetic practice. Although unproven, the current opinion is that most neurological injuries during anaesthesia are the result of prolonged deformation, impaired perfusion of the cord, or both.3 There are many reports of cord injury in normal subjects after prolonged or even short periods of spinal deformation,3 such as the recent report of a 14-yr-old wrestler who sustained a central cord syndrome after performing a series of ‘sit-ups’ with his neck flexed.4 It seems likely that spinal disease increases this risk of cord damage.

It is probably true that anaesthetists not accustomed to cervical spine surgery would be more concerned about a diagnosis of cervical instability than a diagnosis of disc protrusion. Instability is really just another cause of canal stenosis, and other causes of canal stenosis should generate equal concern. It must be emphasized that spinal stability does not confer immunity from risk. The risk is greatest in patients undergoing long, complicated operations indicated by preoperative myelopathy.

Concerns about neurological deterioration during anaesthesia are perhaps most marked after traumatic injury. Approximately 5% of patients admitted with traumatic cervical injury will deteriorate neurologically. Most deteriorate early (24 h), some later (1–7 days), and occasionally patients undergo late deterioration (weeks—subacute post-traumatic ascending myelopathy).5 The causation of late deterioration is not well understood, but the concern that such an episode might coincide with an anaesthetic is easy to appreciate.

**Prevention of neurological deterioration during anaesthesia**

**Maintaining spinal cord perfusion**

Prolonged minor malposition may be the cause of some cord injuries so, as far as requirements for surgery render possible, the position should look comfortable. This, of course, is merely guesswork, but any position that looks uncomfortable must be deplored. Most patients will have some degree of cord or root compression, and it must be sensible to maintain a good perfusion pressure. Maintaining normotension under anaesthesia is usually difficult and might involve inotrope infusions. Hypertension may, in theory at least, promote cord swelling in damaged areas. Surgical bleeding may also demand a lower than normal pressure.

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**Table 1** Definitions of instability

<table>
<thead>
<tr>
<th>Some definitions of instability</th>
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<tbody>
<tr>
<td>1. Symptomatic</td>
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<tr>
<td>‘Loss of the ability under normal physiologic loads to maintain relationships between vertebrae in such a way that there is neither initial nor subsequent damage to the spinal cord or nerve roots, and there is neither development of incapacitating deformity or severe pain’</td>
</tr>
<tr>
<td>2. Radiological measurements</td>
</tr>
<tr>
<td>(a) Translation</td>
</tr>
<tr>
<td>C1–C2: anterior atlanto-dental interval &gt;5 mm, posterior ADI &lt;13 mm</td>
</tr>
<tr>
<td>C2–T1: &gt;3.5 mm between points on adjacent vertebrae</td>
</tr>
<tr>
<td>(b) Angulation</td>
</tr>
<tr>
<td>&gt;11° between vertebrae</td>
</tr>
<tr>
<td>These values have been widely used, but there is a poor correlation between radiographic abnormality and neurological symptoms and signs</td>
</tr>
<tr>
<td>3. Integrity of anterior and posterior spinal columns</td>
</tr>
<tr>
<td>The spine can be thought of as two columns (anterior and posterior), anterior column disruption tending to make the spine unstable in extension and posterior column damage favouring instability in flexion</td>
</tr>
</tbody>
</table>

**Table 2** Criteria for stability after cervical trauma

<table>
<thead>
<tr>
<th>Conscious patient</th>
<th>Unconscious patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alert, no distracting injuries</td>
<td>Plain radiographs are inadequate</td>
</tr>
<tr>
<td>No midline pain</td>
<td>The combination of plain films and CT scans is adequate to diagnose bony and ligamentous instability</td>
</tr>
<tr>
<td>Normal movement</td>
<td>MR scans are not required for the exclusion of instability</td>
</tr>
</tbody>
</table>
In practice, a systolic pressure of >100 mm Hg is the authors’ aim in normotensive patients. The blood supply to the spinal cord is believed to be similar to the cerebral circulation in terms of regulation of flow, and it has been suggested that an area of relatively reduced reliability of perfusion exists at the cervico–thoracic junction. However, a recent report suggests that the cord is most likely to suffer ischaemic events at the C2–C3 level.8

Drainage of CSF is used to promote spinal cord perfusion in aortic surgery, but the practical difficulties of inserting a drain in patients with spinal disease generally militates against this technique in cervical surgery.

**Cord monitoring**

Evoked potential monitoring of both sensory and motor cord pathways is increasingly practised. Motor potentials are adversely affected by volatile agents and neuromuscular blocking agents. Hypotension decreases evoked potentials, so they give some guide to the adequacy of perfusion pressure. Some practitioners eschew neuromuscular blocking agents during surgery on cord or roots, so that unwanted stimulation can produce a reflex movement.

**Airway management**

Airway management continues to be a talking point, because of the lack of evidence that one method is better than another in terms of neurological outcome. Most of us are aware instinctively of ‘outcome bias’, which causes reviewers of a case to be more critical of management when the outcome has been poor,7 and this factor probably contributes to a preference for ‘high-tech’ over ‘low-tech’ tactics, but a patient’s interests are likely to be better served by a familiar technique done promptly and well, rather than an unfamiliar one done slowly and badly.

The available evidence suggests that, in unstable cervical spines, basic airway manoeuvres such as chin lift and jaw protrusion cause as much displacement as direct laryngoscopy.8 Since the necessity of basic airway management cannot be ruled out, whenever a patient is anaesthetised, it can be argued that airway devices should be placed in the awake patient. However, there is no evidence that using an awake procedure results in better neurological outcome, and the most recent analysis of the ASA’s Closed Claims contained several examples of serious morbidity and mortality caused by awake fiberoptic intubation.9

Manual in-line stabilization (MILS) of the head and neck during direct laryngoscopy seems a priori a sensible idea, although one without evidence of efficacy in terms of improved outcome. However, there may be greater difficulty in obtaining a good laryngeal seal when MILS is applied,8 so that as with cricoideal pressure, judgement must be used as to when the necessity of establishing an airway should take precedence.

Practitioners should use a technique in which they are competent. In practice, many patients, particularly ‘unstable’ patients, are functionally rigid, persevering with difficult direct laryngoscopy. There is no place for difficult direct laryngoscopy—an alternative technique should be used. These techniques include the laryngeal mask airway (LMA) with fiberoptic guidance, the intubating laryngeal mask airway (ILMA), the lightwand, alternative laryngoscopes such as the Glidescope or Bullard, fiberoptic intubation (asleep or awake), or a surgical tracheostomy.

Airway management is almost certainly a minor factor in cases of neurological deterioration, but case reports alleging that airway management caused spinal cord injury continue to appear. Alternative, more plausible, explanations are available in these cases, but even the worst quality reports are examples of the fact that patients with cervical abnormalities are at risk of neurological injury during anaesthesia.10 11

**Difficult laryngoscopy and cervical spine disease**

Cervical spine disease is a major cause of difficult direct laryngoscopy. Lesions affecting the OAA complex are the most likely to result in difficult laryngoscopy.12 Iatrogenic causes (fixators, collars) and arthritic processes are the common aetiologies. Reduced mouth opening often compounds the problem of poor OAA extension. Arthritic involvement of the temporomandibular joint (TMJ) may be present, but OAA extension is necessary for normal mouth opening even in subjects with normal TMJs.13

Pre-anaesthetic prediction of difficult laryngoscopy remains problematic. Clinical estimation of reduced OAA extension is surprisingly difficult, because it is hard to separate OAA movement from the movements of the cervical spine as a whole. Patients with reduced OAA extension have been shown to compensate with increased movement at sub-axial levels.14 The Mallampati examination continues to have a place and is a more reliable indicator in this population than in any other group, doubtless because of the relationship between mouth opening and OAA extension. Radiological evidence of reduced OAA mobility is useful. If there is poor separation of the posterior elements of the occiput, C1, and C2 on a neutral or flexion lateral view, then difficult laryngoscopy is very likely.12

**Postoperative airway obstruction**

Some degree of airway obstruction is not uncommon after anterior cervical surgery. It is sometimes because of a haematoma, but in many cases it is because of tissue swelling. It usually presents within 6 h, but can occur later. Airway obstruction is particularly likely after combined anterior–posterior cervical surgery.15

There are some important diagnostic points:

1. Stridor is unusual—the obstruction is because of swollen tissue in the supra and peri-glottic regions.
2. The patients say they ‘can’t breathe’ and want to sit up.
3. Oximetry may register almost normal values until very late.
4. The presence of a drain in the neck does not prevent swelling.
Heliox, dexamethasone i.v. and nebulised epinephrine are all worth trying, but the priority is to open the wound, which may relieve tissue tension enough to restore an airway.\textsuperscript{16} Re-intubation may be difficult. The patients must be managed in a semi-sitting posture. Awake fibreoptic intubation is sometimes a good option, but direct laryngoscopy after sevoflurane and oxygen induction may be easier. The gum-elastic bougie is often vital and an LMA (± fiberscope, ± gum elastic bougie or Aintree catheter) or an ILMA-guided technique may save the day. The use of succinylcholine in myelopathic patients is hazardous because of abnormal potassium shifts.

Airway obstruction because of bilateral recurrent laryngeal nerve palsy is rare, but unilateral nerve damage is seen from time to time and causes a ‘lowing’ cough and a weak voice.

**Eye damage during anaesthesia and surgery**

There are three causes of oculopathy: corneal abrasion, ischaemic optic neuropathy (ION), and central retinal artery thrombosis (CRAT). ION and CRAT result in permanent visual loss in most cases.\textsuperscript{17} The ASA has established a registry of cases of postoperative visual loss at www.asaclosedclaims.org.

Most corneal abrasions are hypoxic injuries after corneal drying because of failure to tape the eye shut. Such abrasions are painful and can take many months to heal. Ocular contamination by skin-prep fluids is a hazard in prone patients.

Although ION can occur in supine patients (1:64 000), it is much more common (1:11 000) after prone surgery. Visual loss may be unilateral and is painless. Major blood loss, hypotension, and prolonged surgery are associations, but it can occur after short, bloodless surgery without hypotension. Reduced nerve perfusion as a result of an increased orbital and intra-ocular pressure in the prone position is believed to be the cause.\textsuperscript{18} Male sex, diabetes, hypertension, and smoking are believed to be risk factors. It is accepted that external pressure on the eye is not a cause. Ophthalmoscopic appearances may be normal if the lesion is posterior, but the disc is swollen and there may be flame-shaped haemorrhages when the anterior portion of the optic nerve is affected.

Usually, CRAT occurs when external pressure damages the eye; however, it can also be because of emboli from carotid disease. Absent eye movements, proptosis and orbital swelling have been seen in nearly all reported cases. Most cases have followed prone positioning using the ‘horseshoe’ type of headrest. It is currently unclear as to whether CRAT can occur in prone patients without embolism or external pressure. The ophthalmoscopic appearances are of retinal oedema (‘cloudy swelling’) and a cherry red spot at the fovea. The flow in the retinal vessels may be interrupted and emboli visible.

It is essential to prevent external ocular pressure. In prone patients, the use of a pin fixation system is the most satisfactory method. The horseshoe headrest should not be used for prone patients. Hypotension and excessive haemodilution should be avoided if possible. It is probably wise to warn at least those patients with risk factors about the possibility of postoperative visual loss.

**References**


*Please see multiple choice questions 9–12*