9

Anesthesia for Cervical Spinal Cord Injury

Apolonia E. Abramowicz and Maria Bustillo

Incidence and Causes of Spinal Cord Injury

The February 2013 National Spinal Cord Injury Statistical Center at the University of Birmingham, Alabama publication reports that approximately 12,000 new cases of spinal cord injury (SCI) occur annually in the United States, not including those who die at the scene of the accident [67]. The figure is an approximation as no studies on the incidence of SCI have been carried out since the 1990s. The National Spinal Cord Injury Database compiles data on an estimated 13 % of new SCI cases in the USA reported to federally funded Model System Centers. Since 2010, 52.2 % of patients reported to the database were categorized as having either incomplete (40.6 %) or complete (11.6 %) tetraplegia at discharge. The term quadriplegia has been largely abandoned; tetraplegia should be used instead. Of note, only less than 1 % of injuries resulted in complete neurologic recovery, but the percentage of incomplete tetraplegia has increased, while complete tetraplegia has decreased. Based on these figures, it may be inferred that approximately 6,000 persons annually suffer cervical spinal cord injury with some degree of neurological impairment. Vehicular accidents and falls account for the majority of the SCIs (65 %), with falls causing more injuries than in the past. Sports injuries and violence are currently responsible for a declining share of SCI and account for 23.5 % of the injuries. Males constitute 80.7 % of all SCI cases, and although the average age at injury is presently 42.6 years, almost half of injuries occur between the ages of 16 and 30. Alcohol is a major factor in 25 % of SCI [17]. The decrease in life expectancy after SCI is due primarily to pneumonia and septicemia. Advances in urologic management have decreased the incidence of renal failure as the leading cause of mortality. The annual cost of healthcare and living expenses in tetraplegia decreases from a high of $1 million in the first year after injury to $110,000 per year thereafter [67].

Of all patients with complete cervical spinal cord injury, approximately 10 % will regain some sensory function and another 10 % will regain some motor function, but 80 % will not improve.

In victims of severe blunt trauma, injury to the cervical spine occurs in 1.8 % of cases. Patients with head trauma are more likely to have a cervical spinal cord injury. The most common level of injury is the C2 vertebra, followed by C6 and C7 [31]. Spinal Cord Injury Without Radiologic Abnormalities (SCIWORA) occurs most commonly in pediatric patients whose elastic spines have more cartilaginous elements, in adults with acute disc prolapse and in patients with cervical spondylosis.

A.E. Abramowicz, M.D. (✉) • M. Bustillo, M.D.
Department of Anesthesiology, Montefiore Medical Center, Albert Einstein College of Medicine,
111 East 210th Street, New York, NY 10467, USA
e-mail: apabramo@montefiore.org

C.S. Scher (ed.), *Anesthesia for Trauma*, DOI 10.1007/978-1-4939-0909-4_9,

Penetrating or blunt trauma to the neck with injury to the vertebral artery may result in cervical spinal cord infarction. The vertebral artery supplies the cervical spinal cord through its single anterior and two posterior branches which descend from the skull base. Additionally, segmental branches of the vertebral arteries provide collateral flow. Penetrating ballistic trauma to the neck (from explosions and gunshot wounds) carries a very high mortality rate. In a study of 90 British Iraq and Afghanistan war casualties, it was associated with cervical spine and/or cord injury in 20 soldiers (22 %). Of those, only 6 (6.6 %) survived to reach hospital care, but 3 of them died of their wounds and only 1 (1.8 %) of the 49 survivors who reached surgical care had an unstable cervical spine injury and lived until emergency surgical spinal intervention; 2 others (3.7 %), despite spinal cord contusion, had spinal fractures not deemed unstable [77].

Of interest to anesthesiologists are perioperative cervical spinal cord injuries. Hindman et al. studied cervical spine injuries reported to the ASA Closed Claims Database and associated with surgery under general anesthesia [38]. Cervical spinal cord injuries ($n = 37$) occurred mostly in men (73 %), in the absence of preexisting spinal trauma (81 %) and instability (76 %) and were permanent and disabling. In the study, 81 % of patients had preoperative nontraumatic anatomic abnormalities such as cervical spinal stenosis and 65 % underwent surgery on the cervical spine. The authors hypothesize that cervical spondylosis, much more prevalent than spinal instability, appears to confer a susceptibility to cord injury related to positioning (Fig. 9.1) and perhaps systemic hypotension, as 24 % of patients had surgery in the sitting position [38, 49]. Cord injury was attributed to airway instrumentation in only 11 % of patients. Ahn and Fehlings of the influential Toronto Spine Program have reviewed 84 papers on perioperative spinal cord injury published between 1966 and 2008, in an attempt to identify patients at risk and in order to propose evidence-based approaches for prevention and improvement of outcomes [3]. The overall incidence of perioperative SCI for surgery on the spine at all levels is estimated by the authors to vary between 0 to a frightening 3 %. The more common medical causes of spinal instability include Rheumatoid Arthritis, Down syndrome, and hypermobile transition zones between fused regions of the cervical spine, as in Klippel–Feil syndrome. The authors advocate “a careful fiberoptic intubation without hyperextension as an important consideration to prevent SCI” and “careful control of the spine” during positioning. Little quality evidence to support this recommendation is given. Nevertheless, the authors list hyperextension of the neck during intubation and positioning in patients with severely tight cervical canals, as well as in those with nontraumatic spinal instability as the “potential” cause of perioperative SCI. A large population of patients could be at risk, as the authors list those with congenital stenosis, diffuse idiopathic skeletal hyperostosis, ossified posterior longitudinal ligament (OPLL), but also those with severe spondylosis of the lumbar spine which correlates with spondylosis of the cervical spine. Kudo described two older women with transient tetraplegia after general anesthesia with endotracheal intubation for retinal detachment and lumbar fusion surgery, respectively, and who recovered after a few hours; postoperative MRI revealed cervical spondylosis with severe spinal canal narrowing [47]. Systolic blood pressure varied between 85 and 150 mmHg. The widely quoted and highly recommended editorial by McLeod in the *British Journal of Anaesthesia* published in 2000 addresses the issue of spinal cord injury and direct laryngoscopy [61]. It emphasizes the importance of positioning and its impact on spinal cord perfusion during prolonged periods of immobility in the perioperative period, in addition to the common sense approach to the selection of the technique of airway instrumentation for the purpose of establishing mechanical ventilation in all patients with cervical spine disease; mechanical instability of the cervical spine is but one risk factor for spinal cord injury during anesthetic care.

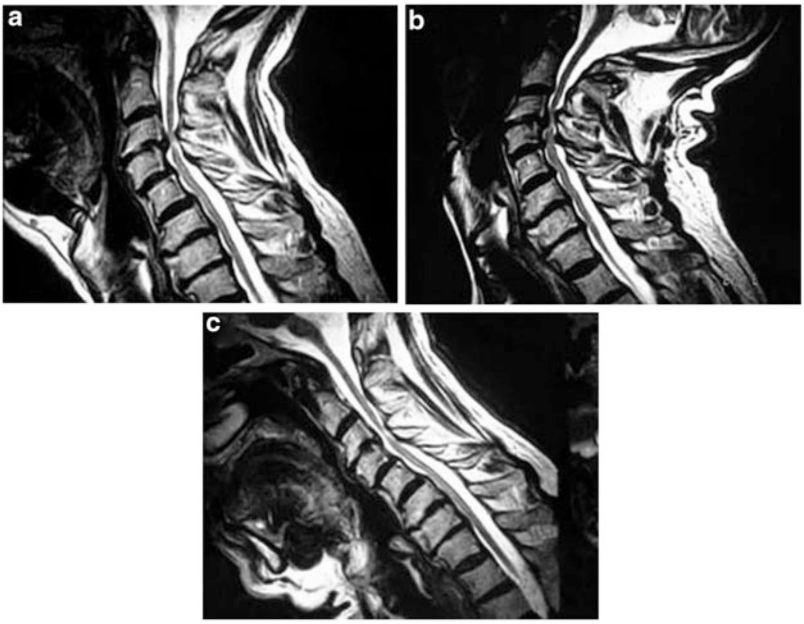


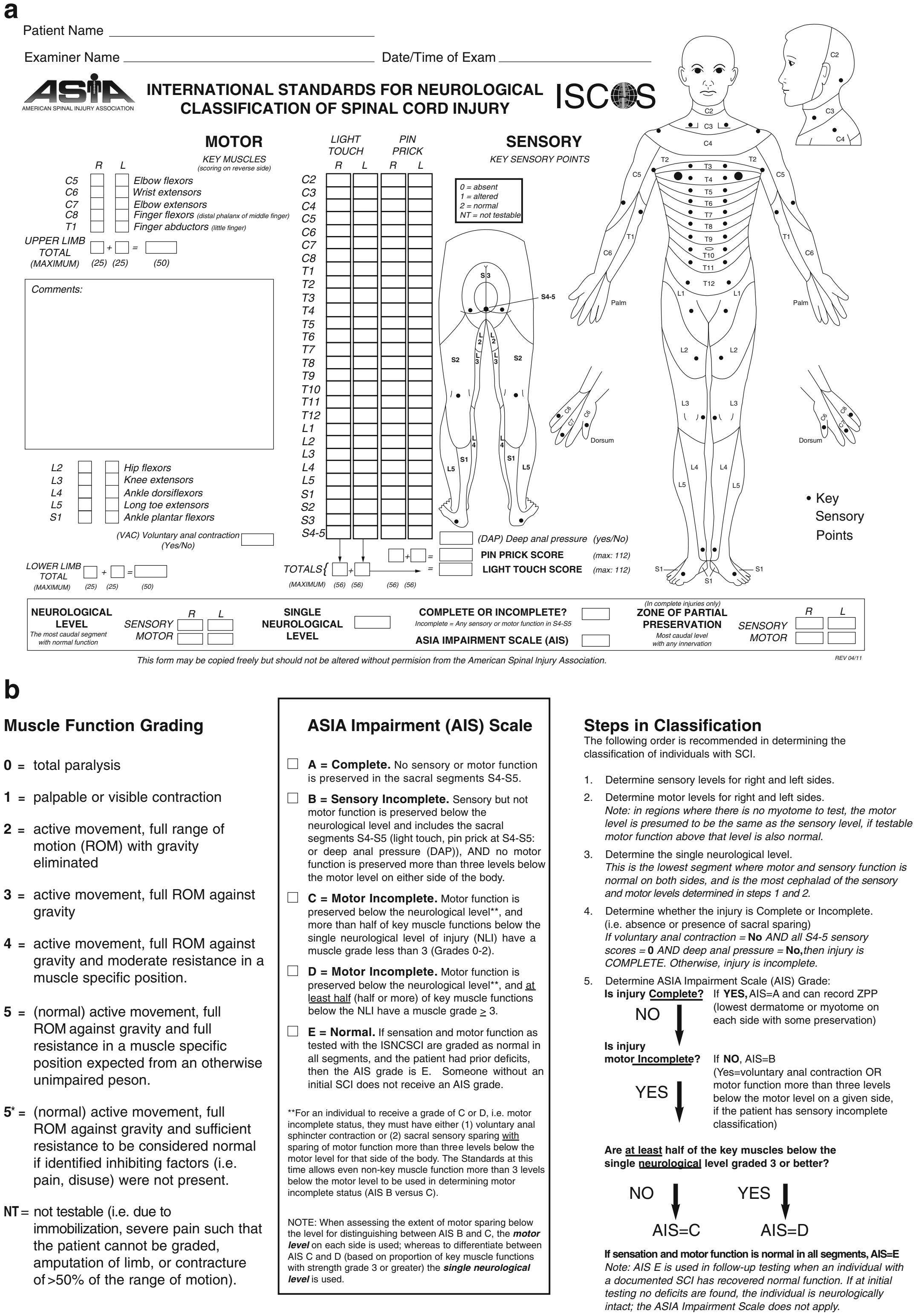
Fig. 9.1 Sagittal MRI scans of the cervical spine; (**a**) showing ventral spinal cord compression from disc herniation at C3–C4 and vertebral body osteophytes. Note the compression of the spinal cord in extension (**b**) that is diminished flexion (**c**). (From Shedid D, Benzel EC: Cervical Spondylosis Anatomy: Pathophysiology and Biomechanics, Neurosurgery 60;S1-7-S1-13, 2007)

Classification of Spinal Cord Injury

The acute management of a patient with cervical spinal cord injury begins with neurological assessment and culminates in a functional outcome that often depends on the deficits at presentation. In 2000, the American Spinal Injury Association (ASIA) has promulgated its International Standards for Neurological Classification of Spinal Cord Injury which has been universally adopted as the best scoring system for the neurological assessment of adult patients with acute SCI. The acute neurological assessment may be hindered by concomitant head injury, drug effects, and the presence of an artificial airway. Nevertheless, the ASIA Impairment Scale (AIS) is considered the most accurate and reproducible neurological assessment tool in acute SCI [4] (Fig. 9.2). The AIS replaces the similar, but older and less stringently defined Frankel scale, while retaining the same injury categories. The AIS is a five level scale, where level E (Normal) implies recovered normal neurological function in someone with an initial spinal cord injury clinical presentation, and level A (Complete) implies lack of motor and sensory function in the sacral segments S4–S5. Level B is a Sensory Incomplete injury, where sensory but no motor function is preserved below the neurological level, and levels C and D reflect Incomplete Motor injury, with D level patients having better strength than C. The "neurological level" of injury is defined as the first spinal segmental level which shows loss of function. In addition, ASIA classifies incomplete spinal cord injuries into five types: central cord syndrome, where function of the upper limbs is more impaired than the lower limbs, the Brown-Sequard syndrome, where only one side of the cord is lesioned and the anterior cord syndrome, where the motor function is absent but the sensory function is spared. Finally, distal spinal injuries may result in conus medullaris and cauda equina syndromes.

The term "spinal shock" means transient absence of all reflex neurologic activity below the level of injury; sensorimotor activity is absent as well. Patients have flaccidity of bowel and bladder, priapism is common; the reflex arcs below the level of injury, for example the bulbocavernosus reflex, recover within days to weeks.

The term "neurogenic shock" describes the symptoms of vasodilatation with hypotension, bradycardia, and hypothermia which result from the interruption of the sympathetic nervous system



a

Patient Name ____________________

Examiner Name ____________________ Date/Time of Exam ____________________

INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

MOTOR

KEY MUSCLES (scoring on reverse side)

	R	L	
C5	☐	☐	Elbow flexors
C6	☐	☐	Wrist extensors
C7	☐	☐	Elbow extensors
C8	☐	☐	Finger flexors (distal phalanx of middle finger)
T1	☐	☐	Finger abductors (little finger)

UPPER LIMB TOTAL ☐ + ☐ = ☐
(MAXIMUM) (25) (25) (50)

Comments:

	R	L	
L2	☐	☐	Hip flexors
L3	☐	☐	Knee extensors
L4	☐	☐	Ankle dorsiflexors
L5	☐	☐	Long toe extensors
S1	☐	☐	Ankle plantar flexors

(VAC) Voluntary anal contraction (Yes/No) ☐

LOWER LIMB TOTAL ☐ + ☐ = ☐
(MAXIMUM) (25) (25) (50)

SENSORY

KEY SENSORY POINTS

	LIGHT TOUCH R	LIGHT TOUCH L	PIN PRICK R	PIN PRICK L
C2				
C3				
C4				
C5				
C6				
C7				
C8				
T1				
T2				
T3				
T4				
T5				
T6				
T7				
T8				
T9				
T10				
T11				
T12				
L1				
L2				
L3				
L4				
L5				
S1				
S2				
S3				
S4-5				

0 = absent
1 = altered
2 = normal
NT = not testable

TOTALS{ ☐ + ☐ ☐ + ☐
(MAXIMUM) (56) (56) (56) (56)

☐ (DAP) Deep anal pressure (yes/No)

☐ **PIN PRICK SCORE** (max: 112)

☐ **LIGHT TOUCH SCORE** (max: 112)

• Key Sensory Points

NEUROLOGICAL LEVEL
The most caudal segment with normal function

	R	L
SENSORY	☐	☐
MOTOR	☐	☐

SINGLE NEUROLOGICAL LEVEL ☐

COMPLETE OR INCOMPLETE? ☐
Incomplete = Any sensory or motor function in S4-S5

ASIA IMPAIRMENT SCALE (AIS) ☐

(In complete injuries only)
ZONE OF PARTIAL PRESERVATION
Most caudal level with any innervation

	R	L
SENSORY	☐	☐
MOTOR	☐	☐

This form may be copied freely but should not be altered without permision from the American Spinal Injury Association.

REV 04/11

b

Muscle Function Grading

0 = total paralysis

1 = palpable or visible contraction

2 = active movement, full range of motion (ROM) with gravity eliminated

3 = active movement, full ROM against gravity

4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position.

5 = (normal) active movement, full ROM against gravity and full resistance in a muscle specific position expected from an otherwise unimpaired peson.

5* = (normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (i.e. pain, disuse) were not present.

NT = not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of >50% of the range of motion).

ASIA Impairment (AIS) Scale

☐ **A = Complete.** No sensory or motor function is preserved in the sacral segments S4-S5.

☐ **B = Sensory Incomplete.** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5 (light touch, pin prick at S4-S5: or deep anal pressure (DAP)), AND no motor function is preserved more than three levels below the motor level on either side of the body.

☐ **C = Motor Incomplete.** Motor function is preserved below the neurological level**, and more than half of key muscle functions below the single neurological level of injury (NLI) have a muscle grade less than 3 (Grades 0-2).

☐ **D = Motor Incomplete.** Motor function is preserved below the neurological level**, and at least half (half or more) of key muscle functions below the NLI have a muscle grade ≥ 3.

☐ **E = Normal.** If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

**For an individual to receive a grade of C or D, i.e. motor incomplete status, they must have either (1) voluntary anal sphincter contraction or (2) sacral sensory sparing with sparing of motor function more than three levels below the motor level for that side of the body. The Standards at this time allows even non-key muscle function more than 3 levels below the motor level to be used in determining motor incomplete status (AIS B versus C).

NOTE: When assessing the extent of motor sparing below the level for distinguishing between AIS B and C, the ***motor level*** on each side is used; whereas to differentiate between AIS C and D (based on proportion of key muscle functions with strength grade 3 or greater) the ***single neurological level*** is used.

Steps in Classification

The following order is recommended in determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
2. Determine motor levels for right and left sides.
   Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.
3. Determine the single neurological level.
   This is the lowest segment where motor and sensory function is normal on both sides, and is the most cephalad of the sensory and motor levels determined in steps 1 and 2.
4. Determine whether the injury is Complete or Incomplete.
   (i.e. absence or presence of sacral sparing)
   If voluntary anal contraction = ***No*** *AND all S4-5 sensory scores =* ***0*** *AND deep anal pressure =* ***No,*** *then injury is COMPLETE. Otherwise, injury is incomplete.*
5. Determine ASIA Impairment Scale (AIS) Grade:

Is injury Complete? If **YES,** AIS=A and can record ZPP (lowest dermatome or myotome on each side with some preservation)

NO

Is injury motor Incomplete? If **NO**, AIS=B (Yes=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)

YES

Are at least half of the key muscles below the single neurological level graded 3 or better?

NO → AIS=C

YES → AIS=D

If sensation and motor function is normal in all segments, AIS=E

Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

Fig. 9.2 (**a**) ASIA spinal cord injury classification and assessment; (**b**) ASIA impairment scale control of hemodynamics in acute cervical and high thoracic spinal cord injury. At the time of injury, there is an initial sympathetic surge of brief duration, with hypertension and potential for subendocardial ischemia, arrhythmias, and "neurogenic" pulmonary edema.

Autonomic hyperreflexia manifests as severe hypertension with reflex bradycardia. It occurs after spinal reflexes have returned, usually after 4–6 weeks of injury. It is common in patients with spinal cord injury lesions above T6. It occurs classically when bladder or bowel distension but also other somatic or visceral triggers stimulate the sympathetics within the spinal cord below the level of injury. As the descending central inhibitory pathways are disrupted, the splanchnic and peripheral bed vasoconstriction below the level of injury remains unopposed and causes severe hypertension, which may be life-threatening. Loss of consciousness, seizures, and hemorrhagic stroke may occur. Compensatory baroreceptor-mediated vasodilatation is present above the level of injury; the vasomotor center activates the vagal parasympathetics and severe bradycardia ensues. Patients are well aware of the episodes; in addition to the pounding headache, they experience anxiety, malaise, nausea, flushing, and sweating above the level of injury and nasal congestion.

The Spinal Cord Independence Measure (SCIM), since 2007 in its third iteration, hence III, is the preferred functional assessment tool used by clinicians involved in the care and follow-up of patients with spinal cord injuries [16]. It evaluates three subscales of self-care, respiration and sphincter management, and mobility for a total score of 0–100. It quantifies the patient's ability to perform everyday tasks and the impact of the disability on the patient's medical condition; in that it is vastly superior to neurological improvement measures which rely on assessing the dermatomal or myotomal level of the lesion.

The injury to the cervical spine may be categorized as atlanto-occipital, atlanto-axial, high (C3, C4), and low cervical.

Management of the Cervical Spinal Cord-Injured Patient

The primary traumatic injury of the spinal cord results from spinal cord compression and contusion. The main elements of treatment comprise immediate resuscitation and stabilization of vital organ function, prevention of cord injury propagation from spinal displacement and ischemia, surgical management and neuroprotective strategies which target the putative inflammatory, neurotoxic and oxidative local processes commonly called "secondary spinal cord injury," the pathophysiology of which has not been elucidated. It is believed that neuroprotection must be applied within a short period of time after injury; the length of this therapeutic window is yet to be defined. It is important to remember that additional injuries are frequently associated with SCI. Although in many ways unique, SCI patients' initial management should follow the ATLS primary and secondary surveys so that no other important injuries are overlooked. In March of 2013, the Congress of Neurological Surgeons has published an update to the 2002 Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries [34]. Reflecting the lack of good quality studies of cardiopulmonary management of SCI patients in the intervening decade, there is no change in the pertinent recommendations relative to the 2002 Guidelines. Explicitly excluding anesthesia citations in its updated review, the guideline on Acute Cardiopulmonary Management offers three level III (based on case series) recommendations: in SCI, patients should be managed in an intensive care unit and "systolic blood pressure below 90 mmHg should be corrected when possible and as soon as possible," the mean arterial blood pressure should be kept between 85 and 90 mmHg for the first 7 days after acute SCI and finally all monitoring devices necessary to detect cardiac, hemodynamic, and respiratory dysfunction should be used.

Cervical Spinal Immobilization After Injury

Victims of blunt trauma with either multiple injuries or isolated head injury, those with altered mental status for any reason, and those who have pain over the spine or severe pain from an associated injury (severe enough to be "distracting" and hence obscuring neck pain) are presumed to have spinal instability and are therefore considered at risk for motion-induced secondary spinal cord injury. Patients with motor or sensory deficits have spinal cord injury which may be aggravated by spine motion. The standard element of management, which starts at the scene of the injury, is spinal immobilization. It is continued until spinal instability resulting from disk and ligament disruption, facet joint subluxation, or bone fracture has been ruled out, ideally in less than 24 h. In general, spinal immobilization consists of a rigid cervical collar, supports (sand bag) on both sides of the head and a backboard with multiple straps which attach the patient to the board and restrain the movement of the entire body. Specific clearance protocols vary, with good quality, fine-cut computed tomography reviewed by an experienced radiologist being recommended by the State of NY [67]. It has been known for over 20 years that patients with spinal cord injury should be transferred to a specialty SCI center as soon as possible, as it results in a decrease of acute care length and in the incidence of preventable respiratory complications and pressure ulcers [63]. In addition, the incidence of paralysis in patients with acute spinal cord injury admitted to a specialized trauma center is significantly lower. In a study of discharge files of 4121 patients with traumatic spinal cord injury, Macias found that when the American College of Surgeons' guidelines are followed and patients are admitted to a level I or II trauma center, the adjusted odds ratio for paralysis at discharge was significantly lower (0.67 at $p < 0.001$), while mortality remained unchanged. Higher surgical volume was associated with reduced paralysis; the authors conjecture that was possibly due to greater use of spinal surgery [54].

The efficacy of cervical spinal immobilization with various devices has been measured primarily in normal human volunteers using different methods [81] such as clinical assessment, plumblines, radiography, cinematography, computed tomography, and MRI, generating a normative body of knowledge. SCI patients have not been well studied yet. The difficulty is in establishing what constitutes a neutral neck position and in quantifying movement between individual vertebral segments. The "neutral" position has been defined as the "normal anatomic position of the head and torso that one assumes when standing and looking ahead" [85]. Alternatively, De Lorenzo, in a MRI study of 19 adults found that a slight degree of flexion, equivalent to a 2 cm occiput elevation, produces a favorable increase in the size of the spinal canal at C5 and C6, regions of frequent unstable cervical spine injury [23]. These findings indicate that some degree of occipital padding is required in adults strapped to a board to achieve neutral positioning of the head and neck relative to the torso. Body habitus and muscular development must be taken into account when determining the thickness of padding.

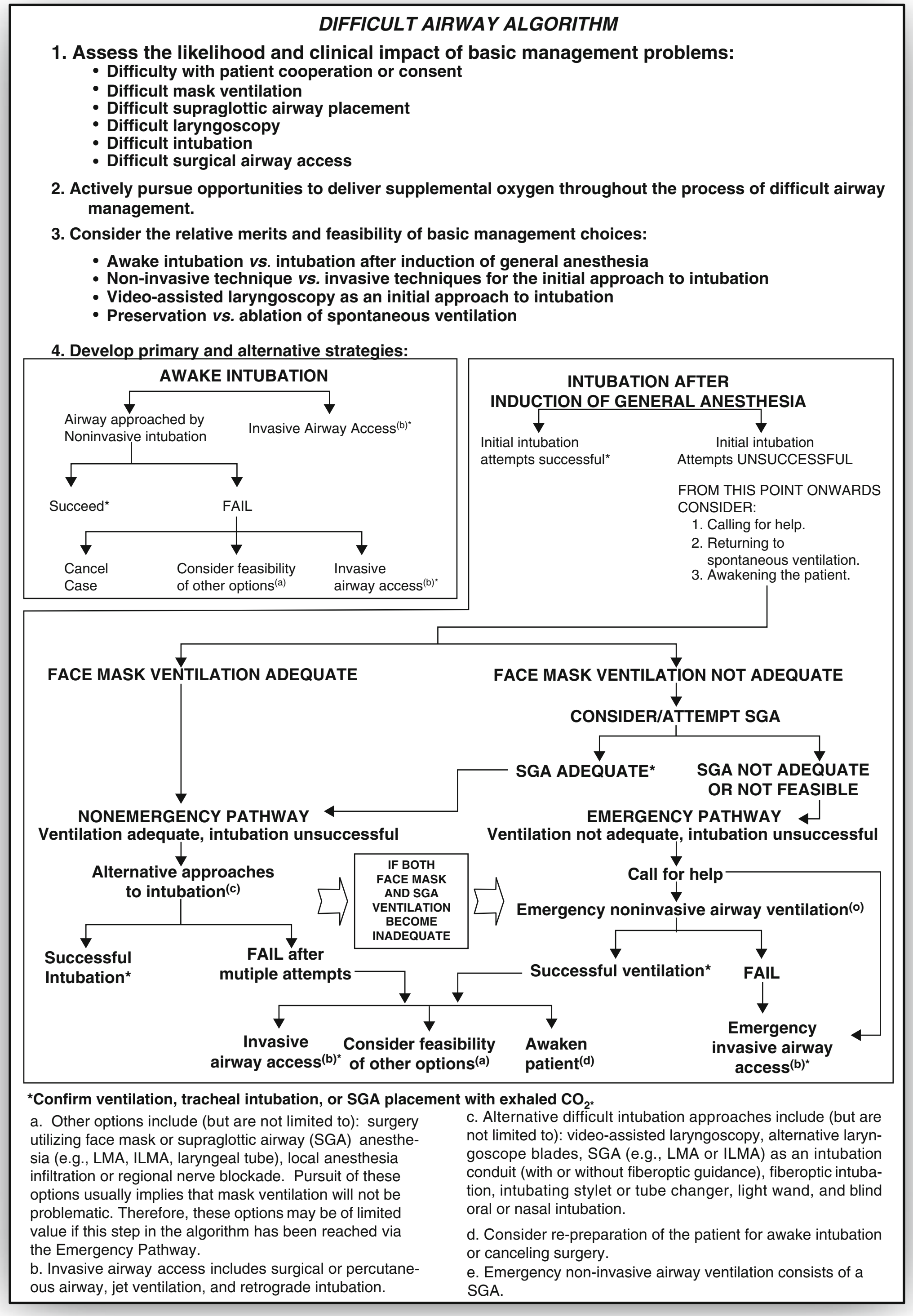
The safety record of the cervical collar and rigid board immobilization is far from perfect. Tightly applied cervical collars have been associated with intracranial pressure elevation by 4.5 mmHg [21, 45]. The length of time a patient with SCI spends on a rigid board increases the risk of early decubitus ulcers, probably after only a 2 h period. Transient marginal mandibular nerve palsy from pressure from a hard cervical collar has been reported, as has skin breakdown over the occiput. Spinal immobilization increases the risk of aspiration and may restrict respiratory function. It has been reported that the presence of a rigid Philadelphia® Collar and backboard restricts ventilation by 15 % [90]. Contraindications to the use of spinal immobilization include penetrating trauma to the spine as it increases the death rate twofold relative to non-immobilized patients, because it delays transport and definitive management of the associated injuries, more life-threatening than SCI. Likewise, it is contraindicated in ankylosing spondylitis, as spinal immobilization may cause neurological deterioration.

Manual in-line stabilization (MILS) is an acceptable cervical spine stabilization technique when the rigid collar interferes with endotracheal intubation by preventing mouth opening. In fact, it is recommended by current ATLS guidelines. After the anterior portion of the collar has been removed, the maneuver is performed by an assistant standing at the patient's side, facing either the feet or the head. The assistant's hands rest on the sides of the patient's head and act as lateral and anti-extension supports, by applying just enough pressure to oppose the atlanto-occipital extension and, to a lesser extent at the C1–C2 joint, occurring during direct laryngoscopy. In addition, when traumatic disruption of the alignment of the subaxial (below C2) vertebrae is present, airway instrumentation may cause vertebral motion, i.e. distraction and or subluxation, distal to the atlanto-occipital junction. MILS would not be expected to prevent this cause of spinal motion. This has been confirmed in a variety of cadaver studies [55, 56]. MILS significantly degrades laryngoscopic view and as a result direct laryngoscopy transmits higher pressure on the tissues adjacent to the unstable vertebral segments, perhaps even doubling it [56, 84, 88]. Illustrating the caveats of theoretical recommendations is a case report of CT-documented odontoid fracture reduction as a result of cranio-cervical motion during emergency direct laryngoscopy with MILS [71]. Despite mounting evidence that MILS, which has been adopted in the 1980s as an off-shoot of "manual in-line traction" and only on the basis of the efficacy of cervical immobilization during general care, may extend the time to intubation with resultant hypoxia, intubation failure, and airway complications, it is still considered a recommended immobilization maneuver during urgent airway instrumentation in un-cleared or unstable cervical spine, when the cervical collar must be opened [55].

Airway Management in Cervical Spinal Cord Injury

Emergency airway management may be required in patients with high cervical cord injury due to acute respiratory failure, or to protect the airway in the setting of coexisting head trauma or polytrauma. Because of the lack of diaphragmatic function, injury above C3 is lethal unless rescue ventilation is rapidly established; these patients will be intubated in the field by emergency personnel. The issues related to spinal cord protection through cervical spine immobilization during airway instrumentation have been discussed above. Further controversies include the choice of intubation technique, airway adjuncts, the utility and role of cricoid pressure, and the place of bag-mask ventilation during rapid sequence induction in SCI. In addition, the emergency setting presents challenges related to the necessity of promptly securing the airway without previous airway examination, the risk of aspiration, lack of patient cooperation, hemodynamic instability, preexisting hypoxemia, craniofacial injuries and airway compromise from prevertebral tissue swelling or hematoma [18, 32]. The recently updated ASA Practice Guidelines for Management of the Difficult Airway (Fig. 9.3) include a difficult airway algorithm which now incorporates the use of supra-glottic devices as rescue ventilation tools or intubation conduits, and videolaryngoscopes; this algorithm does not specifically address the problems of the cervical spine-injured patient, but should be followed whenever unexpected difficulties are encountered despite airway management planning [6].

Although bag-mask ventilation is contraindicated in the classic rapid sequence induction scenario because of the risk of gastric distension and the resulting increased risk of aspiration, it is even more problematic when protecting the cervical spine is an additional concern. Older studies on human cadavers concluded that chin lift and jaw thrust and mask ventilation caused more cervical spine displacement than other airway procedures, notably than



DIFFICULT AIRWAY ALGORITHM

1. **Assess the likelihood and clinical impact of basic management problems:**
    - **Difficulty with patient cooperation or consent**
    - **Difficult mask ventilation**
    - **Difficult supraglottic airway placement**
    - **Difficult laryngoscopy**
    - **Difficult intubation**
    - **Difficult surgical airway access**
2. **Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management.**
3. **Consider the relative merits and feasibility of basic management choices:**
    - **Awake intubation *vs.* intubation after induction of general anesthesia**
    - **Non-invasive technique *vs.* invasive techniques for the initial approach to intubation**
    - **Video-assisted laryngoscopy as an initial approach to intubation**
    - **Preservation *vs.* ablation of spontaneous ventilation**
4. **Develop primary and alternative strategies:**

***Confirm ventilation, tracheal intubation, or SGA placement with exhaled CO_2.**

a. Other options include (but are not limited to): surgery utilizing face mask or supraglottic airway (SGA) anesthesia (e.g., LMA, ILMA, laryngeal tube), local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

b. Invasive airway access includes surgical or percutaneous airway, jet ventilation, and retrograde intubation.

c. Alternative difficult intubation approaches include (but are not limited to): video-assisted laryngoscopy, alternative laryngoscope blades, SGA (e.g., LMA or ILMA) as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, and blind oral or nasal intubation.

d. Consider re-preparation of the patient for awake intubation or canceling surgery.

e. Emergency non-invasive airway ventilation consists of a SGA.

Fig. 9.3 ASA Practice Guidelines for Management of the Difficult Airway. (From Anesthesiology. 2013 Feb;118(2):251-70. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Apfelbaum JL, Hagberg CA, Caplan RA, Blitt CD, Connis RT, et al.)

direct laryngoscopy (DL) [8, 37]. Two newer studies showed that the displacement is either comparable to DL in human cadavers with posteriorly destabilized C3, or of smaller magnitude in healthy patients with anatomically normal airways and with MILS simulation [14, 91]. Lacking more convincing data, it is probably safe to assume that in the absence of factors suggesting difficulty with mask ventilation, it is an acceptable maneuver in cervical spinal injury. Since proper preoxygenation in the setting of emergency intubations in not always possible, and since the benefit of the correction of hypoxemia may in some circumstances outweigh the risk of aspiration, it appears that gentle mask ventilation could be acceptable, when necessary, from the standpoint of cervical cord protection.

Cricoid pressure (Sellick's Maneuver) aims to occlude the lumen of the esophagus between the cricoid cartilage and the C5–C6 level of the cervical spine by applying external pressure to the cricoid cartilage in an attempt to reduce aspiration of passively regurgitated stomach contents. The effect of the maneuver on spine motion was examined in human cadavers with intact cervical spine and was found to be modest. When utilized in a cervical spinal injury patient, a bimanual technique may be safer, with one hand under the neck, counteracting the downward pressure on the cricoid cartilage. Recent trauma emergency tracheal intubation guidelines removed cricoid pressure during rapid sequence induction as a level 1 recommendation as there is doubt it decreases the incidence of aspiration in the emergency room setting, while having been shown to worsen the laryngoscopic view and impair mask ventilation efficacy in that setting [60]. Similarly, the 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care state that "routine use of cricoid pressure during airway management of patients in cardiac arrest is no longer recommended." The comprehensive review of the efficacy of cricoid pressure and its potential to interfere with airway management techniques has been included in the 2010 Scandinavian Practice Guidelines on General Anesthesia for Emergency Situations by the working group of the Scandinavian Society of Anesthesiology and Intensive Care Medicine. In light of the lack of scientific evidence of its effectiveness, numerous reports questioning its efficacy and its potential for airway distortion and obstruction, even when applied properly, resulting in more difficult laryngoscope insertion, glottis visualization and reduced tidal volumes with increased airway pressures during mask ventilation, cricoid pressure can be used based on individual judgment of the anesthesiologist but is not recommended [41]. In US hospitals with anesthesia residency programs, cricoid pressure remains commonly used by anesthesiologists during rapid sequence induction [28].

There is no evidence-based recommendation for the choice of tracheal intubation technique in cervical SCI. The available data do not include outcome trials [79]. Despite a large volume of publications addressing surrogate end-points of vertebral angulation, anterior and posterior displacement of the spine, atlanto-occipital and atlanto-axial extension in normal patients and injured spine cadaver models with and without MILS simulation, using a myriad of airway devices, we are left with the unsatisfying conclusion that individual judgment must guide us in the selection of intubation technique and airway adjuncts. Awake fiberoptic intubation has theoretical advantages in that little spinal movement occurs during visualization and intubation, although topical anesthesia of the trachea commonly results in coughing. In addition, a neurological evaluation can be performed after intubation, and after the patient is positioned awake, with muscle tone aiding in avoiding potentially harmful motion. Awake fiberoptic intubation may not be practical in the uncooperative or unstable patient or a patient with a soiled airway. Failed awake intubation carries its own morbidity. Since the publication of Crosby's exhaustive Airway Management in Adults after Cervical Spine Trauma paper, many new airway devices have been studied in anesthetized healthy patients and established human cadaver models with one standardized type of injury [19]. The research continues to focus on the

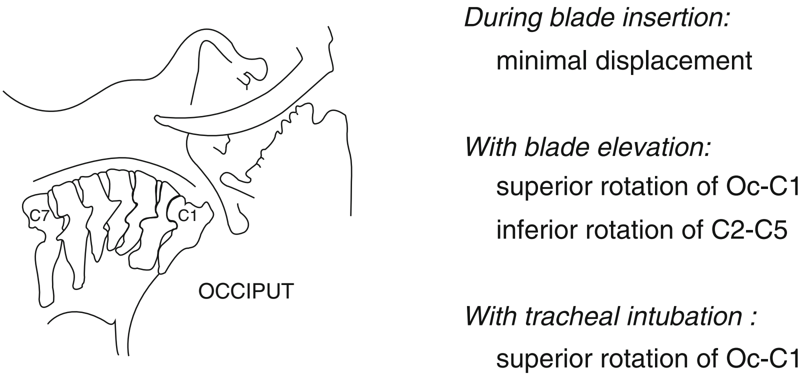


Fig. 9.4 Impact of Macintosh blade laryngoscopy on cervical spine movement. From Crosby ET. Anesthesia for cervical Spinal Cord Injury, Anesthesiology 2006;104(6):1293-318

mechanical effects of various devices and compares the measurable displacement they cause (Fig. 9.4). Brimacombe reported that in his cadaver model with C3 instability, the Combitube did worst and only nasal intubation over a fiberoptic bronchoscope produced no spinal movement [14]. The standard LMA and fiberoptic intubation through the intubating laryngeal mask had intermediate motion scores. We also know that the Torchlight® blind Intubating Lighted Stylet causes 57 % less motion than a Macintosh blade direct laryngoscopy (DL) at the Occiput-C1, C1–C2, C2–C5, and C5-thoracic segments studied in anesthetized patients, that the GlideScope® decreases only the C2–C5 segment motion by 50 % but at the expense of a 62 % prolongation of the duration of intubation, which, 8 years of experience with it later, might no longer be a valid finding [10, 91]. Robitaille reported that the GlideScope® improved glottic visualization during intubation with MILS in normal patients, but did not decrease the degree of extension at the occiput or motion at the rostral C spine relative to Macintosh DL [80]. Lately, Kill compared the GlideScope® to Macintosh DL in anesthetized normal patients without MILS [44]. He found that the GlideScope reduced movement of the cervical spine, particularly in the hands of more experienced operators, while improving the intubation success rate. Curiously, the rigid fiberscope Bonfils® Stylet caused less extension at the atlanto-occipital, C1–C2, and C3–C4 levels, but not C2–C3, than the Macintosh DL [82]. The rigid but malleable Shikani Optical Stylet®, however, did not reduce motion at C1–C2 segment, but did better by 52 % than Macintosh DL at the other levels studied [93]. We also know that compared to Macintosh DL, the Airtraq® decreases cervical spinal motion by an average of 66 %, but not at the C1–C2 junction [92].

Aziz reviewed the use of video-assisted intubation in the management of patients with artificially applied MILS. The papers compare the intubation success rate, Intubation Difficulty Scale, intubation time or laryngeal view obtained using the different video laryngoscopes and Aitraq® versus DL, or in one case, versus GlideScope® [9]. The findings favor video laryngoscope use and are summarized in Table 9.1. Video laryngoscopes decrease intubation difficulty in patients with cervical spine immobilization and may be easy to learn. The author concludes that in the absence of neurologic outcome data, intubation techniques which maximize success rates during MILS are likely to gain acceptance.

Elective airway management in spinal cord injury patients undergoing surgery creates favorable conditions for unhurried airway topicalization and awake fiberoptic intubation. The nasal route is favored by some, but the oral route allows placement of a larger tube and avoids the risk of septic complications from bacteremia during nasal instrumentation and from sinusitis, if the patient must remain intubated after surgery. Sedation is optional and must take into account the patient's injury level, respiratory reserve, and baseline blood pressure. If a halo is in place, difficulty in securing the

Table 9.1 Compilation of studies of videolaryngoscopy and intubation performance

Author	Device	Control	Sample	Outcome assessed	Major findings
Malik MA Br J Anaesth 2008	GlideScope (Veraton, Bothell, WA) Airway Scope AWS (Pentax, Hoya, Japan)	DL	120	Laryngeal view IDS intubation time success rate	Improved laryngeal view and IDS Slower intubation time. No difference in success
Maharaj CH Anesthesiology 2007	Airtraq (Prodol, Vizcaya, Spain)	DL	40	IDS intubation attempts laryngeal view	Reduced number of intubation attempts. Improved IDS. Improved laryngeal view
Smith CE Anesthesiology 1999	WuScope (Pentax, Orangeburg, NY)	DL	87	IDS laryngeal view, intubation attempts	Improved IDS and laryngeal view. No difference in success or number of attempts
Malik MA Br J Anaesth 2009	Airway Scope (AWS)	DL	90	IDS, laryngeal view	Improved IDS and laryngeal view.
Enomoto Y Br J Anaesth 2008	Airway Scope (AWS)	DL	203	Laryngeal view, intubation time, success rate	Improved laryngeal view, increased success rate, faster intubation time
Liu EH Br J Anaesth 2009	Airway Scope (AWS)	GlideScope	70	IDS, intubation time, success rate within a defined time interval	Faster Intubation time, lower IDS, improved laryngeal view, and higher intubation success with AWS
McElwain J Br J Anaesth 2011	Airtaq, C-MAC (Karl Storz, Tuttlingen, Germany)	DL	90	IDS success rate. laryngeal view. hemodynamic stability	Reduced IDS, improved laryngeal view with Airtraq

Modified with permission from Aziz M. Use of video-assisted intubation devices in the management of patients with trauma. Anesthesiol Clin. 2013 Mar;31(1):157-66
DL direct laryngoscopy, *IDS* Intubation Difficulty Scale Score

airway should be anticipated, as access to the airway is limited and glottic visualization may be hindered by the relatively flexed position of the head. Alternatively, some centers perform fiberoptic intubation after induction of anesthesia. The advantage of this approach is that coughing-induced spine motion is prevented, but a jaw thrust might be necessary. As in emergency situations, general anesthesia followed by an intubation technique other than flexible fiberoptic is acceptable as long as MILS is maintained. The choice depends on specific airway characteristics, practitioner's expertise, and patient's preference and ability to cooperate [53].

Respiratory Management

Patients with cervical spinal cord injury have intercostal muscle weakness or paralysis and rely on diaphragmatic function for ventilation. The level of respiratory dysfunction correlates with the level of injury; forced vital capacity (FVC) may be severely decreased. The main inspiratory muscles are the diaphragm, innervated by the C3–C5 nerves and the intercostal muscles, innervated by the T2–T11 nerves. Without simultaneous intercostal muscle contraction, the contraction of the diaphragm sucks in the anterior chest wall, causing paradoxical chest wall movement. The accessory inspiratory muscles are the sternocleidomastoid and the trapezius muscles, innervated by the accessory nerve (cranial nerve XI), and the scalene muscles, innervated by the C3–C8 nerves. Forced expiration requires abdominal muscle contraction; their innervation is provided by the T6–T12 nerves. Injuries above C3 cause instant apnea; survivors are ventilator-dependent unless a diaphragm stimulator is used. Injuries at C4 (the fifth cervical nerve originates between the C4 and C5 vertebrae) or above often result in immediate respiratory failure. Because of abdominal muscle paralysis and low VC, cough is ineffective. Restricted ventilation and impaired clearance of airway secretions predispose to atelectasis, pneumonia, and respiratory failure which occur on average within 4 days of injury and are the leading causes of death in SCI patients. Reduced lung volume and atelectasis reduce lung compliance, increasing the work of breathing. Rapid, small tidal volume breathing, barely exceeding the volume of dead space, is inefficient and may further contribute to diaphragmatic fatigue. Monitoring FVC is an objective way of assessing ventilation; values below 12–15 mL/kg indicate the need for assisted ventilation. Noninvasive ventilation may help reduce the work of breathing by improving lung compliance; BiPAP is usually required and can be considered before endotracheal intubation. Patients with a FVC below 25 % of predicted value have a high incidence of respiratory failure requiring mechanical ventilation. In a case series, all patients with complete SCI at C5 or above required a tracheostomy; of those with a C6 injury or below, 79 % required intubation and 50 % eventually required tracheostomy [83]. FVC increases approximately by 9 % per decreasing level of injury. Interestingly, patients with adequate alveolar ventilation and normal p_aCO_2 often have relative hypoxemia, likely because of ventilation perfusion mismatch occurring immediately after SCI. Maintaining the supine position has advantages for respiratory function in acute cervical SCI at all levels: the abdominal contents push the diaphragm higher into the chest, decrease its radius of curvature (make it less flat), which causes more efficient contractions. Supine FVC and FEV_1 are larger compared to seated values [24]. Abdominal binding is useful in improving diaphragmatic mechanics as well. Regardless of the level of injury, FVC improves after 5 weeks and doubles 3 months after injury. Over time, developing spasticity of the intercostal and abdominal muscles minimizes paradoxical, "seesaw" pattern of abdominal and chest wall excursions on inspiration and contributes to improvement of respiratory function [87]. The expiratory function and therefore the ability to cough also improves over time, as the pectoralis major muscle's clavicular portion contraction helps raise the intrathoracic pressure. Interruption of sympathetic innervation to the lungs appears to have physiological significance due to heightened cholinergic tone and may explain obstructive airway changes, common in tetraplegic patients. There is good responsiveness to the anticholinergic Ipratropium bromide inhalation. When instituting mechanical ventilation for purely restrictive respiratory failure without associated lung pathology, larger than the usual "lung protective" tidal volumes have been advocated to decrease atelectasis and accelerate weaning [24]. In consideration of these facts an arterial blood gas must be obtained as quickly as possible, supplemental oxygen should be given to all patients with an acute cervical SCI and mechanical ventilation, noninvasive or invasive, should be considered in all patients with C4 or C5 lesions, even if they appear compensated. Meticulous attention to optimization of respiratory function and clinical pathways for respiratory management decrease mortality from SCI and may prevent some aspects of secondary spinal cord injury. Anesthesiologists should be aware that in patients with acute cervical SCI, immediate extubation after anesthesia close to the time of injury should not be contemplated.

Hemodynamic Management

Patients with acute cervical spinal cord injury often present with neurogenic shock manifesting as hypotension due to the loss of sympathetic vasomotor tone with decreased preload related to blood pooling; associated bradycardia is due to the loss of function of cardiac accelerator fibers and contributes to decreased cardiac output. Despite lack of evidence beyond case series reports, it is believed that aggressive blood pressure management in the face of potentially disrupted spinal cord blood flow autoregulation is important in preventing secondary spinal cord injury and leads to improved functional outcomes. The 2013 updated Acute Cardiopulmonary Management of Patients with Cervical Spinal Cord Injuries guideline remains unchanged from its first version from 11 years ago. As an option, or level III recommendation, it calls for restoration of systolic blood pressure to above 90 mmHg as quickly as possible and maintenance of mean arterial pressure between 85 and 90 mmHg for 7 days after injury. Between 60 and 85 % of patients with complete high SCI have a systolic blood pressure below 90 mmHg; the average BP on admission was 66 mmHg in a series of patients with ASIA A injury. This incidence may be lower in patients with lower levels of injury and incomplete injuries. Similarly, in 71 % of severely injured patients, heart rate was below 45 beats per minute for at least one day but this was present in only 35 % of patients with milder, incomplete injuries [83]. Patients with cervical spinal cord injury are at risk for life-threatening episodic and recurrent hemodynamic instability for up to 10–14 days after injury; asystole has been described with tracheal suctioning. The highest risk involves the most injured patients; stable hemodynamic function at presentation is not a predictor of lack of complications. Persistent orthostatic hypotension is present in 29 % of chronically injured tetraplegic patients. Because up to 30 % of SCI patients may have multiple trauma and hemorrhage, it is important to identify hypovolemia as the coexisting cause of hypotension [11]. Since reflex tachycardia does not occur in the neurogenic shock period, bradycardia does not rule out hypovolemia. The initial management of neurogenic shock is volume expansion; if fluid resuscitation alone does not raise the systolic blood pressure above 90 mmHg, vasopressors should be added. Treatment of bradycardia should be undertaken simultaneously; sinus node slowing may be profound enough to produce hypotension and even cardiac arrest. Hypotensive patients who do not respond to Atropine require temporary pacing; up to 17 % of patients with complete injury required pacing in another case series [83]. Among 37 acute SCI patients with neurogenic shock admitted to one institution, adrenal insufficiency was identified in 22 % of cases; there was no correlation with the duration of vasopressor use to maintain the MAP at 85–90 mmHg [74]. The choice of vasopressor for neurogenic shock has not been well studied. In a review of vasopressor use in patients with acute spinal cord injury, arterial and central line placement is recommended; dopamine is favored over phenylephrine because of concern for bradycardia, and vasopressin is suggested as a reasonable choice [65]. Others use norepinephrine or epinephrine [24, 40]. Vasopressors may have deleterious effects in case of concurrent brain injury. Dopamine may impair hepatosplanchnic oxygenation; β-receptor agonists and exogenous catecholamines have recently come under scrutiny as a cause of stress-induced cardiomyopathy (SIC) (takotsubo cardiomyopathy) [1]. The cardiomyopathy is characterized by transient left ventricular apex akinesia with hypercontractile basal segments of the heart in the absence of coronary artery disease, associated with significant left ventricular dysfunction. SIC has been described after traumatic brain injury, status epilepticus, subarachnoid hemorrhage and in cases of neurogenic pulmonary edema [22]. SIC has been attributed to a catecholamine surge or sympathetic nervous system activation in the setting of acute neurological injury. SIC has been described in a male patient with chronic C5 tetraplegia 10 days after removal of a spinal opioid pump, demonstrating that systemically elevated catecholamines, presumably from opioid withdrawal, can cause SIC in the absence of an intact nervous system [78]. The patient recovered after a week of extracorporeal membrane oxygenation. Lethal neurogenic pulmonary edema with severe acute cardiomyopathy has been described in a chronic incomplete C5 injury patient after two episodes of autonomic hyperreflexia presumed to be only the second such report [15]. Catecholamines could be harmful in spinal cord injury patients with cardiac dysfunction and/or pulmonary edema. It appears that the association of neurogenic pulmonary edema with SIC is being diagnosed more often as cardiac echocardiography is used in cases of acute decompensation following a neurological injury. In the absence of evidence that blood pressure augmentation in the week following acute spinal cord injury is unsafe, this Cervical SCI recommendation should probably be followed.

Temperature Management

Vasodilatation impacts temperature regulation and predisposes to hypothermia in low ambient temperatures. Conversely, hyperthermia may easily occur in high temperatures because sweating below the level of injury is impaired. Patient temperature must be closely monitored and warming devices, including fluid warmers, should be used.

Thromboembolism

Patients are at high risk within hours, because of immobility. Prophylaxis should be instituted within 72 h of injury (level II recommendation, 2013 Guidelines). In patients who are not candidates for anticoagulation and/or sequential compression devices, vena cava filters are recommended (level III recommendation).

Gastrointestinal System

Acute SCI causes ileus and delayed gastric emptying, which may persist for 2–3 weeks. Pulmonary aspiration is a risk, especially since patients are positioned supine to optimize pulmonary function.

Steroids in the Treatment of Cervical Spinal Cord Injury and Therapeutic Hypothermia

As of March 2013, large dose methylprednisolone should not be used in acute spinal cord injury patients. Until now, as the only widely used neuroprotective therapy, steroid administration has been the mainstay of treatment of blunt spinal cord injury since the NASCIS (National Acute Spinal Cord Injury Study) II and III trials of the 1990s [12, 13]. Both studies have been repeatedly criticized for serious methodological and statistical analysis flaws, but steroid use has continued in the face of lack of other treatment options for a devastating injury in predominantly young, healthy patients [64]. For this reason, and despite evidence of harmful effects of steroids, and just a hint of benefit measured only in minor improvements across many muscle groups, but not in functional neurological improvement, the 2002 Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries recommended methylprednisolone for either 24 or 48 h as an option. Methylprednisolone is the only steroid studied by NASCIS; it was never approved by the FDA for this indication. In March 2013, the Congress of Neurological Surgeons (CNS) and the American Association of Neurological Surgeons (AANS) published their updated Guidelines [35, 39]. There is now level I evidence that high-dose steroids are harmful, but only level III evidence of inconsistent benefits of steroids in acute blunt SCI; the updated guideline unequivocally recommends against the use of methylprednisolone in these patients [5]. The adverse effects attributable to steroids include increased incidence of pneumonia, sepsis, GI complications, thromboembolism, hyperglycemia and finally, acute corticosteroid myopathy. This iatrogenic entity tends to improve spontaneously over 6–8 months and its resolution is thought to perhaps underlie some of the motor function improvement observed in the NASCIS studies.

There has been an increasing interest in the use of therapeutic mild or modest (33 °C) hypothermia after acute spinal cord injury, along with some anecdotal reports of its efficacy. In the 2010 clinical case series of systemic hypothermia in cervical SCI from the Miami Project to Cure Paralysis at the University of Miami, 14 patients with complete (ASIA grade A) cervical spinal cord injuries were cooled using venous femoral endovascular heat exchange cooling catheters to maintain the core temperature at 33 °C for 48 h, with rewarming at the rate of 0.1 °C per hour, over a 24 h period. Improvement in one AIS grade or better occurred in 42.8 % of the patients within three months; a very significant improvement compared to data reported for spontaneous recovery from other trials [51]. As none of the patients showed improvement over the first

2 weeks after injury, misclassification of the severity of injury either due to spinal shock or sedative administration seems not to be at issue in this report. A more recent publication from the same group describes a prospective cohort of 21 additional ASIA grade A patients managed with the same hypothermia protocol [26]. They report late improvement of at least one ASIA grade in 35.5 % of cases; most observed complications were respiratory and included pneumonia in 60 % of patients and pulmonary edema in 43 %. Thromboembolic complications with two pulmonary embolisms occurred in 14 % of patients, which does not represent an excess of these events despite the use of a femoral venous cooling catheter system. The same group has reviewed the current state of knowledge on hypothermia in SCI and reports that a randomized trial of systemic hypothermia with 17 centers participating and the intent to recruit 200 patients is being planned [2]. At this time, although advances in endovascular cooling technology allow the institution of hypothermia with minimal delay which is important for the effectiveness of neuroprotective strategies, moderate hypothermia is not a proven therapeutic modality in acute spinal cord injury.

Timing of Surgery

Issue is now actively debated, but it is far from resolved. The current surgical management trend of acute cervical SCI patients favors early decompression and stabilization of the cervical spine. Logistical difficulties with early intervention in this patient population, because of delayed transfers to specialized centers and patient eligibility due to coexisting injuries or morbid conditions, are common. The term “early surgery” is therefore usually arbitrarily defined as surgery occurring within 24 h of injury. The best evidence in favor of early surgery comes from a multicenter, prospective, but nonrandomized cohort study of 313 patients with acute cervical SCI who underwent definitive open surgical decompression and fixation with instrumentation of the cervical spine [29]. The study concluded that surgery prior to 24 h after SCI is safe and is associated with improved neurologic outcome, defined as at least two grade ASIA impairment scale (AIS) improvement at 6 months of injury. Despite a debate as to the significance of the results for surgical practice standards, there is a definite momentum towards early decompression/fusion in SCI patients; this may be performed via an anterior, posterior, and combined approach [72]. This change is of importance to anesthesiologists, because of the complexities of intraoperative management of patients with neurogenic shock and the ensuing necessity for urgent evaluation and optimization of these patients.

Fig. 9.5 Jumped bilateral facets X-ray. Courtesy of Dr. John Houten

A subset of patients with acute SCI present with cervical facet dislocation (Fig. 9.5). Although relatively rare among patients with cervical spine trauma, facet dislocation, especially if bilateral, results in spinal cord injury in up to 90 % of patients. The injury occurs during high-velocity neck hyper-flexion with slippage of the upper vertebra lower facets relative to the upper facets of the lower vertebra; as a result, the diameter of the spinal canal is decreased,

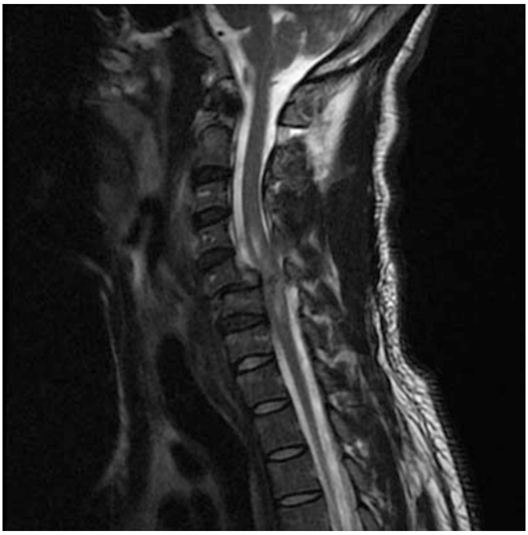


Fig. 9.6 MRI of cervical spinal cord after bilateral facet dislocation at C5, C6 level. Courtesy of Dr. John Houten

compressing the spinal cord and causing cord contusion (Fig. 9.6). These patients often undergo urgent skeletal traction and closed reduction of the dislocation, as rapid restoration of the alignment of the spine may, in certain cases, lead to immediate neurological recovery and is therefore advocated as a means of prompt decompression of the spinal cord [94]. Skull tongs are placed under local anesthesia, and weights are sequentially added via a pulley system to distract and realign the cervical spine; this is sometimes done at the bedside (Fig. 9.7). In patients breathing spontaneously, the pain the procedure causes is usually treated with intravenous opioids. In some instances, closed reduction is performed in the operating room under general anesthesia; if unsuccessful, it may be immediately converted to an open, surgical reduction (Figs. 9.8 and 9.9).

Surgical Interventions

Anesthetic implications vary depending on the surgical access. Anterior approaches are either open cervical for anterior discectomy and plate fusion, or percutaneous for odontoid fractures. Cervico-medullary junction decompression is carried out through a trans-oral approach. Minimally invasive spine surgery is making inroads—an endonasal endoscopic approach for odontoid and C1 arch resection has been described. Posterior approaches involve laminectomy decompression with fixation, usually with lateral mass screws and rods, for subaxial injuries and atlanto-axial-occipital fusion. A minority of patients will be placed in a halo vest after surgery.

Fig. 9.7 Skeletal traction to reduce facet dislocation. Courtesy of Dr. John Houten

Complications of Surgery of Relevance to Anesthesiologists

The most common complication of the *anterior cervical approach* to spinal fusion is transient dysphagia. The most dangerous complication is postoperative hematoma; the reported incidence of clinically apparent hematoma is 0.2–5.6 %. Wound drainage does not completely safeguard against this complication. Approximately half of postoperative hematomas require emergency

Fig. 9.8 Anterior reduction and fusion of facet dislocation. Courtesy of Dr. John Houten

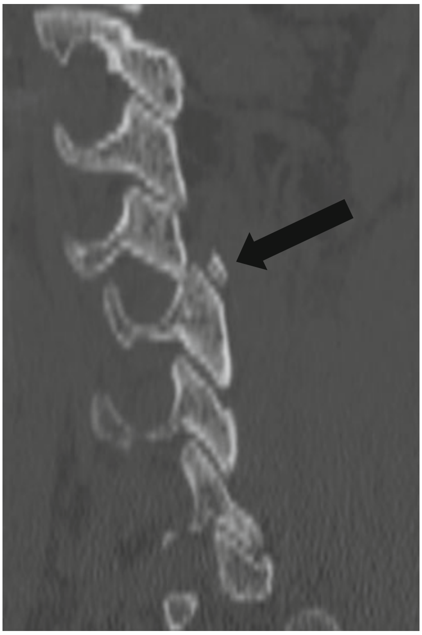


Fig. 9.9 *Arrow* points to cervical facet dislocation—CT scan. Courtesy of Dr. John Houten

surgical evacuation, due to complaints of dyspnea despite normal oxygenation, overt respiratory compromise, worsening dysphagia, and hematoma expansion associated with neck pain [30]. When a postoperative neck hematoma is present, difficulties securing the airway may occur despite a reassuring initial intubation course. This may be due to either direct airway compression with luminal impingement and deviation, or more ominously, because of airway edema. The edema is thought to occur as blood collects in the anterior neck and hinders venous and lymphatic outflow. The swelling of the arytenoids and the epiglottis may be present, even when the total volume of hematoma is low [73]. Thought should be given to either attempting to visualize the glottis or performing the endotracheal intubation awake. Opening the wound under local anesthesia to decompress the hematoma may facilitate intubation.

Symptomatic recurrent laryngeal nerve palsy is another common complication of the anterior approach with an incidence of 1.1–3.1 % in large patient series from the last 20 years [30]. Asymptomatic, and presumably transient, recurrent laryngeal nerve dysfunction is much more common. The vagus and the recurrent laryngeal nerves are not visualized during surgery. The cause of recurrent laryngeal neuropraxia and paralysis has been attributed to nerve stretch and compression between the surgical retractor and the endotracheal tube balloon, observed anatomically in cadaveric investigations [46]. In a clinical study by the same authors, deflating the endotracheal cuff and re-inflating it after retractor blade placement, which presumably allows the endotracheal tube to “recenter” itself in the larynx, decreased the rate of temporary vocal cord paralysis from 6.4 to 1.69 % [7]. The highest rate of vocal cord dysfunction has been observed after surgery at T1 level, while C7 had a rate of 3.9 %. Higher surgical levels were associated with an incidence of 1.3 % at C5 and 2.2 % at C6 in the same case series. Trauma as the indication for surgery was not associated with an increased incidence of recurrent laryngeal nerve palsy. The importance of this observation is that recurrent laryngeal nerve injury may occur in cervical spine fractures, especially if the injury involves lower vertebrae. Indeed, in a report of two patients with

C6–C7 and C7–T1 fracture dislocation, respectively, and who presented with dysphonia, recurrent laryngeal nerve injury presumed to be caused by the injury was confirmed preoperatively [33]. The left-sided approach is theoretically more protective of the recurrent laryngeal nerve, because the left nerve has a longer loop and is better protected within the tracheo-esophageal groove. Most right-handed surgeons prefer the right-sided approach however. Several studies refute the superiority of the left-sided approach [43]. A high level of suspicion for recurrent laryngeal nerve injury in cervical spine injuries should arise when hoarseness or dysphonia is evident in awake, talking patients. Anterior surgery in patients with preexisting recurrent laryngeal nerve injury should be performed from the approach ipsilateral to the injury to prevent bilateral nerve paralysis. Recurrent laryngeal nerve EMG monitoring to prevent intraoperative nerve damage, although commonly used, has not been systematically studied in anterior cervical spinal fusion. The 2013 Guidelines for Improving Voice Outcomes after Thyroid Surgery recommend intra-operative recurrent laryngeal nerve EMG monitoring to identify the nerve as an option only, emphasizing the importance of proper placement of the laryngeal electromyography endotracheal tube.

Rare complications of the anterior approach to spine surgery are esophageal and pharyngeal perforation (with possibly subcutaneous emphysema), dural perforation and CSF leakage, vertebral artery injury, Horner's syndrome, and neurological deterioration.

The posterior approach necessitates prone positioning with the attendant risk of neurological injury in unstable cervical spine injuries during rotation from the supine position. This may be minimized by the use of a cervical collar with manual in-line stabilization during manual patient transfer, awake self-positioning in neurologically intact patients after intubation, or via the "Jackson table" technique, where the patient is transferred to the table supine with the usual immobilization precautions, then secured to the table which is then rotated 180°. Continuous electrophysiological monitoring may be utilized before and immediately after positioning prone. A pin head-fixation device is commonly used in posterior cervical spine surgery. It has a potential to cause excessive neck motion during both patient transfer and while it is being secured to any operating table, including the Jackson table.

Paraspinal muscle dissection and laminectomy may cause blood loss, although this is seldom very significant at the cervical level. Vertebral artery injury is a very rare but potentially important cause of blood loss and intra-operative neurological injury.

Prolonged spine surgery may cause rhabdomyolysis, and when performed in the prone position, it is a risk factor for peri-operative visual loss, especially in the male, obese patients positioned on a Wilson frame [20, 75].

Improper positioning may cause outright compression of the inferior vena cava and severely decreased preload. Prone positioning decreases blood pressure and cardiac output in anesthetized patients, likely because of a decrease in venous return and decreased cardiac compliance [27]. The hemodynamic effects of prone positioning depend on the type of positioner used. The Jackson table and the longitudinal bolsters have the least effect on cardiac function by echocardiography performed immediately after positioning when adequate fluid replacement is provided after turning the patient prone [25]. Because it is important to minimize venous engorgement of the orbital content and the airway in the prone position, some degree of head-up tilt is necessary. This may contribute to a decrease in venous return and cardiac output, even when using the Jackson table. The hemodynamic changes occurring in the prone position are expected to have more clinical significance in the elderly, those with chronic cardiovascular comorbidities, and patients with neurogenic shock. The hemodynamic effects of the prone position have anesthetic implications: the plasma concentration of Propofol is increased when cardiac output is lower, presumably because of decreased drug clearance [50]. Therefore, if needed, the choice of vasopressor must take into account its effect not just on the blood pressure, but also the cardiac output and by extension, on the hypnotic effect of Propofol [66]. In general, hemodynamic management in the prone position has not been well studied; CVP does not predict fluid responsiveness and correlates poorly with left ventricular end-diastolic volume, especially in the prone position [58].

The rare occurrence of venous air, fat, and bone fragment embolism must be considered in cases of sudden hemodynamic instability [27]. The increasingly common direct spinal wound application of powdered Vancomycin to prevent postoperative infection, intended to maximize local antibiotic concentration without high plasma levels, may nevertheless result in circulatory collapse as in the intravenous route of administration [57].

The respiratory effects of the prone position are positive in terms of lung volumes, preferential blood flow to dependent lung areas and ultimately, improved ventilation/perfusion matching leading to the utilization of the prone or near-prone position in the mechanically ventilated ARDS patients. Surgery on the cervical spine in the prone position involves the use of large radiographic equipment like a C-arm, or more commonly now an O-ring CT scanner which is manipulated in close proximity to the endotracheal tube. The risk of accidental extubation has to be anticipated as this complication has been reported. A recent case report reviews the airway management options should this occur [89]. Additional causes of airway loss in the prone position include endotracheal tube obstruction by bloody secretions or inspissated secretions; reinforced endotracheal tube malfunction with obstruction has been reported as well [76]. Although turning the patient supine in an emergency may be delayed by the need to remove instrument constructs from the wound, having a stretcher available at all times is a basic prone patient-operative safety feature. Airway rescue maneuvers in the prone position may include tube reinsertion over a flexible fiberoptic bronchoscope, ventilation and intubation through an LMA, aspiration of secretions and clearing the tube lumen with an arterial embolectomy catheter [27].

Neurophysiological Monitoring During Cervical Spinal Decompression with Fusion and Instrumentation

Intra-operative neurophysiological monitoring (IOM) is widely used in spine surgery in the hope of the early detection of iatrogenic neurological insults related to the mechanical stress of positioning, surgical manipulation, and cord perfusion from hypotension. The risk of neurological injury during cervical spine surgery, although ever-present and potentially devastating, has not been well quantified.

Because the dorsal and ventral portions of the cord have separate blood supply, simultaneous monitoring of the somatosensory (SSEPs) and transcranial myogenic electrical motor-evoked potentials (MEPs), as well as spontaneous electromyography (EMG) from relevant muscle groups, is usually performed, and is called multimodal IOM. SSEPs monitor the sensory dorsal column-medial lemniscus pathway, recording subcortical or cortical responses to stimulation of a peripheral nerve. Because SSEP acquisition requires signal averaging, the temporal summation provides a reading with some delay, sometimes of a few minutes duration, depending on the number of averages needed. The time interval between the stimulus applied to the peripheral nerve and the evoked response is called latency, while the size of the response is its amplitude. The alert or warning criteria differ, and many clinical papers do not report them, but a decrease in amplitude of more than 50 % and increase in latency by 15 % or more are considered significant changes that must be communicated to the surgical and anesthesia teams. SSEPs can localize the site of injury or ischemia more exactly than the MEPs provided the sensory pathway is affected, as recordings can be monitored from both the cerebral cortex and subcortical sites. MEPs assess the entire motor axis. Electrical stimulation with high-frequency trains of high voltage is applied percutaneously over the motor cortex and compound muscle action potentials (CAMPs) are recorded from muscle groups of interest. MEPs cause patient movement during stimulation, although this can be minimized by stimulus voltage adjustment, and as a result can only be assessed periodically. The warning criterion for MEPs varies. Some centers use an all-or-none criterion; others consider an 80 % reduction in amplitude at any one recording site to be an alert value. Another way of quantifying the alert value for MEPs is establishing a minimum stimulus threshold value that results in CAMPs; an increase in the threshold of 100 V is considered a warning [48]. MEPs may trigger seizures, albeit very rarely. Volatile anesthetics and nitrous oxide decrease the amplitude of MEPs and interfere with the ability to monitor MEPs, and are therefore proscribed. Spontaneous EMG allows continuous monitoring of selective nerve root function throughout the surgery. MEP and EMG monitoring precludes the use of muscle relaxants during surgery on practical grounds.

The American Academy of Neurology and the American Clinical Neurophysiology Society analyzed twelve studies meeting pre-defined quality and enrolment size criteria in an effort to determine whether spinal cord IOM predicts adverse surgical outcomes [70]. On this basis, the published evidence-based guideline concludes that “IOM is established as effective to predict an increased risk of the adverse outcomes of paraparesis, paraplegia and quadriplegia in spinal surgery.” This conclusion assumes, in the authors’ opinion, that a “knowledgeable professional clinical neurophysiologist supervisor” is responsible for the IOM, as opposed to IOM “conducted by a technician alone or by an automated device.” Armed with the knowledge that well-conducted IOM has an excellent, if not perfect, predictive value for the most severe neurological complications, we still do not know whether the use of IOM prevents neurological injury in spine surgery. There are no guidelines addressing this issue. A firm consensus exists only for scoliosis surgery. There is data addressing the sensitivity and specificity of modern multimodal IOM, but it does not specifically address surgery in spinal cord-injured patients. In general, the reported sensitivity and specificity of IOM in cervical spine surgery in the last decade, that is since myogenic MEP monitoring has become an integral part of IOM, is between 95 and 100 % [48]. However, as discussed previously, warning criteria vary and measures of neurological outcome are also variable. A retrospective analysis of 200 consecutive cervical spine surgery cases managed with IOM included 40 traumatic spine injury patients; it reports a specificity of 100 % of SSEPs and MEPs, and a sensitivity of 100 % of the combination of SSEPs and MEPs for detecting impending neurological injury [52]. Of note, the SSEPs were primarily useful in alerting of a malposition of the arm and, in one instance, of the need to raise the blood pressure while the MEP alerts were caused by hypotension and in one case, graft malposition.

Filling the void of information on the preventive value of IOM on intraoperative neurological injury is a simulation model which evaluates the cost-benefit of IOM, assuming a 5 % baseline neurologic complication rate, a cost of IOM of $1,535 per case, a prevention rate of an IOM alert of 52 % in 10,000 surgeries, and a lifetime cost of lost wages and health care of an intraoperative incomplete neurologic deficit involving either the spinal cord or nerve roots of $900,000 [69]. The simulation predicts considerable cost-savings if IOM is used; 2.3 million dollars would be saved for each 100 spine surgeries. The savings would occur even if the risk of neurological injury were 1 %, although only $3,500 per procedure would be saved, and even if the prevention rate were far lower than the one assumed. There being no economic or outcome down-side to poor specificity, the focus should be on the sensitivity of the IOM techniques used. In summary, the cost-benefit is most favorable if IOM is used in patients with severe underlying pathology and those undergoing high-risk procedures, and if there is corrective action in response to an IOM alert. This certainly applies to patients with incomplete spinal cord injury.

Pain Associated with Spinal Cord Injury

The SCI Guidelines have a new level I recommendation: the clinical assessment of Pain in SCI patients should be performed, preferably using the International Spinal Cord Injury Basic Pain Dataset to evaluate the severity of pain as well as physical and emotional functioning [42].

Pain associated with spinal cord injury is common; its prevalence is estimated at 80 %. Chronic pain of SCI interferes with the ability to achieve maximal functional recovery and degrades the quality of life. It may also lead to severe depression. Pain may be neuropathic or nociceptive, or both. Nociceptive pain may be related to spasticity and painful muscle spasms, as well as overuse of the arms and shoulders; it responds to standard pain management protocols. Neuropathic pain below the level of injury is more common in tetraplegics, but overall there is no relationship between the presence of pain and the completeness or level of the lesion [86]. In addition to its high prevalence, neuropathic pain associated with spinal cord injury is resistant to conventional pharmacological treatment and is therefore debilitating; there is also a tendency for the pain to worsen over time. In most patients the pain is spontaneous, but can also manifest as allodynia and hyperalgesia. The central neuropathic pain of SCI has several subsets determined by pain location: above, at, and below the level injury. It is believed that different mechanisms of abnormal neural activity and inflammation at spinal and supraspinal levels are involved in these pain subsets. Central neuropathic pain differs from peripheral neuropathic pain. Efficacy would therefore be expected from distinct treatment combinations; however the lack of understanding of the pathophysiology of central neuropathic pain hinders the development of pharmacological regimens targeted to central neuropathic pain processes. Certain drugs, such as tricyclic antidepressants, commonly used in peripheral neuropathic pain are not as effective in SCI patients and they may be poorly tolerated in patients with incomplete injuries because of urinary retention; bladder distension is a known trigger of autonomic hyperreflexia. Opioids are commonly utilized, but with only a 30 % longer-term response rate. Intrathecal opioids have been used in combination with intrathecal clonidine and baclofen and with ziconotide, a selective N-type calcium channel blocker [36]. Newer research however indicates that in the acute phase of injury, opioids may exacerbate injury-induced excitotoxic damage to neurons and cause glial activation in the spinal cord and may actually promote the development of pain after spinal cord injury by activating central sensitization pathways. In addition, in the setting of spinal cord injury opioids may negatively impact recovery of locomotor function through the same mechanisms [95]. Oral gabapentin shows some promise. Daily intravenous ketamine infusions have been added to oral gabapentin with some success; the improvement is transient and disappears some two weeks after the infusions are stopped. Neither deep brain stimulation nor dorsal column spinal cord stimulation is effective in SCI pain; combinations have not been studied [62].

In summary, the recommended SCI pain assessment tools should bring about more standardized information about pain syndromes, their time course and help design treatment modalities in more homogenous groups of patients and analyze their effectiveness, perhaps enhancing the understanding of the pathophysiological processes underlying SCI pain.

Anesthetic Technique Specific to Spinal Cord Injury

Little is known about the short- and long-term effects of specific anesthetic agents in the setting of acute spinal cord injury. Knowledge of the respiratory and cardiovascular implications of SCI is essential in managing an anesthetic in a spinal cord-injured patient. The presence of coexisting injuries and the risk of aspiration must be taken into account. Chiefly however, the anesthesiologist should do no harm and focus on preventing secondary spinal cord injury by maintaining spinal alignment during airway instrumentation to the extent dictated by its urgency and by ensuring adequate spinal cord perfusion. The newest guidelines of the Congress of Neurological Surgeons provide some guidance in the form of target blood pressure values, albeit at a low level of evidence.

The prolific group from Korea led by K.Y. Yoo has published a dozen papers in European and American anesthesiology journals over the last decade, examining the effects of laryngoscopy and intubation on cardiovascular responses, as well as anesthetic requirements for endotracheal intubation and prevention of autonomic hyperreflexia, in acutely and chronically spinal cord-injured patients [96, 97]. Summarizing their findings, cardiovascular responses to intubation are blunted in tetraplegic patients, irrespective of the time since injury: blood pressure does not increase and may fall, heart rate does increase, but to a lesser extent than in paraplegics. Norepinephrine levels do not increase at intubation in acute tetraplegia, but do so mildly after 4 weeks of injury. Predictably, the arousal response to intubation, measured by BIS values, is no different in SCI patients than in non-injured controls.

Succinylcholine is a drug with a very fast onset of action. It is ideally suited for emergency endotracheal intubations. Its administration poses the risk of hyperkalemic cardiac arrest in susceptible patients. Spinal cord injury with tetraplegia involves the majority of the body's muscle mass. Denervation causes upregulation of acetylcholine receptors at the muscle membrane within hours of immobilization. The appearance of extrajunctional receptors takes about 12 h. The extrajunctional receptors will in turn upregulate, since denervation persists. Within 48–72 h of injury, this upregulation may be critical enough to cause lethal hyperkalemia after succinylcholine administration to tetraplegic patients. As long as the paralysis persists, these patients are potentially at risk for hyperkalemia after succinylcholine administration [59]. It is worth noting that Yoo's group has used succinylcholine in the cohort of 214 acute and chronic SCI patients described in the reference, seemingly without ill effect [97].

Research in acute spinal cord injury must lead to a better understanding of the "secondary injury" processes, allowing for focused neuroprotection strategies. Some candidate interventions might be metalloproteinase inhibition by Minocycline or sodium channel blockade by Riluzole. The North American Clinical Trials Network (NACTN) for Treatment of Spinal Cord Injury is currently enrolling patients for a prospective evaluation of the natural history of spinal cord injury managed in selected, specialized centers. This work is likely to provide us with new best practices and of importance to anesthesiologists, test the emerging evidence that ultra-rapid spine decompression and fusion improves neurological outcome. The NIH funding for spinal cord injury research remains flat at about $79 million US$ annually, of which approximately $5 million is allocated to the longitudinal patient follow-up by NACTN [68].

Other research focuses on axonal growth and re-myelination, synapse formation, and the use of central nervous system stem cells in spinal cord injury repair. Although the body of knowledge about the injury and its healing is growing, the promise of a cure for SCI is as yet unfulfilled.

Acknowledgments The authors wish to thank Dr. John K. Houten, Professor of Clinical Neurological Surgery, Albert Einstein College of Medicine, Chief, Division of Spinal Neurosurgery, Montefiore Medical Center, Bronx, NY for graciously making available to us and giving us permission to reprint the images of cervical facet dislocation, cord compression, skeletal traction and anterior cervical fusion. We thank Ms. Samantha Rawana for her expert and cheerful assistance with copyright compliance and manuscript submission and Mr. James Sweeney for making everything appear in the required dpi resolution.

References

1. Abraham J, Mudd JO, Kapur NK, Klein K, Champion HC, Wittstein IS. Stress cardiomyopathy after intravenous administration of catecholamines and beta-receptor agonists. J Am Coll Cardiol. 2009;53(15):1320–5. doi:10.1016/j.jacc.2009.02.020.

2. Ahmad F, Wang MY, Levi AD. Hypothermia for acute spinal cord injury-a review. World Neurosurg. 2013;pii:S1878–8750(13):00015–6. doi: 10.1016/j.wneu.2013.01.008. [Epub ahead of print]
3. Ahn H, Fehlings MG. Prevention, identification, and treatment of perioperative spinal cord injury. Neurosurg Focus. 2008;25(5):E15. doi:10.3171/FOC.2008.25.11.E15.
4. American Spinal Injury Association [Internet]. Atlanta: International Standards for Neurological Classification of Spinal Cord Injury Exam Sheet. 2013. http://www.asia-spinalinjury.org/elearning/ISNCSCI.php. [Revised 2013 Feb].
5. Anderson, Pauline. New CNS/AANS Guidelines Discourage Steroids in Spinal Injury. Medscape [Internet]. 2013. http://www.medscape.com/viewarticle/781669.
6. Apfelbaum JL, Hagberg CA, Caplan RA, Blitt CD, Connis RT, Nickinovich DG, et al. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Anesthesiology. 2013;118(2):251–70. doi:10.1097/ALN.0b013e31827773b2.
7. Apfelbaum RI, Kriskovich MD, Haller JR. On the incidence, cause, and prevention of recurrent laryngeal nerve palsies during anterior cervical spine surgery. Spine (Phila Pa 1976). 2000;25(22):2906–12.
8. Aprahamian C, Thompson BM, Finger WA, Darin JC. Experimental cervical spine injury model: evaluation of airway management and splinting techniques. Ann Emerg Med. 1984;13(8):584–7.
9. Aziz M. Use of video-assisted intubation devices in the management of patients with trauma. Anesthesiol Clin. 2013;31(1):157–66. doi:10.1016/j.anclin.2012.10.001. Epub 2012 Nov 3.
10. Aziz MF, Healy D, Kheterpal S, Fu RF, Dillman D, Brambrink AM. Routine clinical practice effectiveness of the Glidescope in difficult airway management: an analysis of 2,004 Glidescope intubations, complications, and failures from two institutions. Anesthesiology. 2011;114(1):34–41. doi:10.1097/ALN.0b013e3182023eb7.
11. Bernhard M, Gries A, Kremer P, Böttiger BW. Spinal cord injury (SCI)—prehospital management. Resuscitation. 2005;66(2):127–39.
12. Bracken MB, Shepard MJ, Collins WF, Holford TR, Young W, Baskin DS, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. N Engl J Med. 1990;322(20):1405–11.
13. Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, Fazl M, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial National Acute Spinal Cord Injury Study. JAMA. 1997;277(20):1597–604.
14. Brimacombe J, Keller C, Künzel KH, Gaber O, Boehler M, Pühringer F. Cervical spine motion during airway management: a cinefluoroscopic study of the posteriorly destabilized third cervical vertebrae in human cadavers. Anesth Analg. 2000;91(5):1274–8.
15. Calder KB, Estores IM, Krassioukov A. Autonomic dysreflexia and associated acute neurogenic pulmonary edema in a patient with spinal cord injury: a case report and review of the literature. Spinal Cord. 2009;47(5):423–5. doi:10.1038/sc.2008.152. Epub 2009 Jan 13.
16. Catz A, Itzkovich M, Tesio L, Biering-Sorensen F, Weeks C, Laramee MT, et al. A multicenter international study on the Spinal Cord Independence Measure, version III: Rasch psychometric validation. Spinal Cord. 2007;45(4):275–91. doi:10.1038/sj.sc.3101960. Epub 2006 Aug 15.
17. CDC.gov [Internet]. Atlanta: Centers for Disease Control and Prevention; 2010. http://www.cdc.gov/traumaticbraininjury/scifacts.html. [Updated 2010 Nov 4].
18. Cleiman P, Nemeth J, Vetere P. A significant cervical spine fracture: think of the airway. J Emerg Med. 2012;42(2):e23–5. doi:10.1016/j.jemermed.2008.07.027. Epub 2009 Jan 6.
19. Crosby ET. Airway management in adults after cervical spine trauma. Anesthesiology. 2006;104(6):1293–318.
20. Dakwar E, Rifkin SI, Volcan IJ, Goodrich JA, Uribe JS. Rhabdomyolysis and acute renal failure following minimally invasive spine surgery: report of 5 cases. J Neurosurg Spine. 2011;14(6):785–8. doi:10.3171/2011.2.SPINE10369. Epub 2011 Mar 18.
21. Davies G, Deakin C, Wilson A. The effect of a rigid collar on intracranial pressure. Injury. 1996;27(9):647–9.
22. Davison DL, Terek M, Chawla LS. Neurogenic pulmonary edema. Crit Care. 2012;16(2):212. doi:10.1186/cc11226.
23. De Lorenzo RA, Olson JE, Boska M, Johnston R, Hamilton GC, Augustine J, et al. Optimal positioning for cervical immobilization. Ann Emerg Med. 1996;28(3):301–8.
24. Denton M, McKinlay J. Cervical cord injury and critical care. Contin Educ Anaesth Crit Care Pain. 2009;9(3):82–6.
25. Dharmavaram S, Jellish WS, Nockels RP, Shea J, Mehmood R, Ghanayem A, et al. Effect of prone positioning systems on hemodynamic and cardiac function during lumbar spine surgery: an echocardiographic study. Spine (Phila Pa 1976). 2006;31(12):1388–93. discussion 1394.
26. Dididze M, Green BA, Dalton Dietrich W, Vanni S, Wang MY, Levi AD. Systemic hypothermia in acute cervical spinal cord injury: a case-controlled study. Spinal Cord. 2013;51(5):395–400. doi:10.1038/sc.2012.161. Epub 2012 Dec 18.

27. Edgcombe H, Carter K, Yarrow S. Anaesthesia in the prone position. Br J Anaesth. 2008;100(2):165–83. doi:10.1093/bja/aem380.
28. Ehrenfeld JM, Cassedy EA, Forbes VE, Mercaldo ND, Sandberg WS. Modified rapid sequence induction and intubation: a survey of United States current practice. Anesth Analg. 2012;115(1):95–101. doi:10.1213/ANE.0b013e31822dac35. Epub 2011 Oct 24.
29. Fehlings MG, Vaccaro A, Wilson JR, Singh A, Cadotte WD, Harrop JS, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). PLoS One. 2012;7(2): e32037.
30. Fountas KN, Kapsalaki EZ, Nikolakakos LG, Smisson HF, Johnston KW, Grigorian AA, et al. Anterior cervical discectomy and fusion associated complications. Spine (Phila Pa 1976). 2007;32(21):2310–7.
31. Goldberg W, Mueller C, Panacek E, Tigges S, Hoffman JR, Mower WR, et al. Distribution and patterns of blunt traumatic cervical spinal injury. Ann Emerg Med. 2001;38(1):17–21.
32. Grissom TE, Varon AJ. Airway management controversies in trauma care. ASA Newslett. 2013;77(4):12–4.
33. Gulsen S, Yilmaz C, Calisaneller T, Caner H, Altinors N. Preoperative functional assessment of the recurrent laryngeal nerve in patients with cervical vertebra fracture: case report. Neurosurgery. 2009;64(1):E191–2; discussion E192. doi: 10.1227/01.NEU.0000336328.59216.08.
34. Hadley MN, Walters BC. Introduction to the guidelines for the management of acute cervical spine and spinal cord injuries. Neurosurgery. 2013;72 Suppl 2:5–16. doi:10.1227/NEU.0b013e3182773549.
35. Hadley MN, Walters BC, Aarabi B, Dhall SS, Gelb DE, et al. Clinical assessment following acute cervical spinal cord injury. Neurosurgery. 2013;72 Suppl 2:40–53. doi:10.1227/NEU.0b013e318276edda.
36. Hama A, Sagen J. Combination drug therapy for pain following chronic spinal cord injury. Pain Res Treat. 2012;2012:840486. doi:10.1155/2012/840486. Epub 2012 Mar 18.
37. Hauswald M, Sklar DP, Tandberg D, Garcia JF. Cervical spine movement during airway management: cinefluoroscopic appraisal in human cadavers. Am J Emerg Med. 1991;9(6):535–8.
38. Hindman BJ, Palecek JP, Posner KL, Traynelis VC, Lee LA, Sawin PD, et al. Cervical spinal cord, root, and bony spine injuries: a closed claims analysis. Anesthesiology. 2011;114(4):782–95. doi:10.1097/ALN.0b013e3182104859.
39. Hurlbert RJ, Hadley MN, Walters BC, Aarabi B, Dhall SS, Gelb DE, et al. Pharmacological therapy for acute spinal cord injury. Neurosurgery. 2013;72 Suppl 2:93–105. doi:10.1227/NEU.0b013e31827765c6.
40. Jia X, Kowalski RG, Sciubba DM, Geocadin RG. Critical care of traumatic spinal cord injury. J Intensive Care Med. 2013;28(1):12–23. doi:10.1177/0885066611403270. Epub 2011 Apr 11.
41. Jensen AG, Callesen T, Hagemo JS, Hreinsson K, Lund V, Nordmark J, et al. Scandinavian clinical practice guidelines on general anaesthesia for emergency situations. Acta Anaesthesiol Scand. 2010;54(8):922–50. doi:10.1111/j.1399-6576.2010.02277.x.
42. Jensen MP, Widerström-Noga E, Richards JS, Finnerup NB, Biering-Sørensen F, Cardenas DD. Reliability and validity of the International Spinal Cord Injury basic pain data set items as self-report measures. Spinal Cord. 2010;48(3):230–8. doi:10.1038/sc.2009.112. Epub 2009 Sep 29.
43. Kilburg C, Sullivan HG, Mathiason MA. Effect of approach side during anterior cervical discectomy and fusion on the incidence of recurrent laryngeal nerve injury. J Neurosurg Spine. 2006;4(4):273–7.
44. Kill C, Risse J, Wallot P, Seidl P, Steinfeldt T, Wulf H. Videolaryngoscopy with glidescope reduces cervical spine movement in patients with unsecured cervical spine. J Emerg Med. 2013;44(4):750–6. doi:10.1016/j.jemermed.2012.07.080. Epub 2013 Jan 22.
45. Kolb JC, Summers RL, Galli RL. Cervical collar-induced changes in intracranial pressure. Am J Emerg Med. 1999;17(2):135–7.
46. Kriskovich MD, Apfelbaum RI, Haller JR. Vocal fold paralysis after anterior cervical spine surgery: incidence, mechanism, and prevention of injury. Laryngoscope. 2000;110(9):1467–73.
47. Kudo T, Sato Y, Kowatari K, Nitobe T, Hirota K. Case Report Post Operative transient tetraplegia in two patients caused by cervical spondylotic myelopathy. Anaesthesia. 2011;66(3):213–6. doi:10.1111/j.1365-2044.2010.06562. Epub 2011 Jan 25.
48. Lall RR, Lall RR, Hauptman JS, Munoz C, Cybulski GR, Koski T, et al. Intraoperative neurophysiological monitoring in spine surgery: indications, efficacy, and role of the preoperative checklist. Neurosurg Focus. 2012;33(5):E10. doi:10.3171/2012.9.FOCUS12235.
49. Lanier WL, Warner MA. New perioperative cervical injury: medical and legal implications for patients and anesthesia providers. Anesthesiology. 2011;114(4):729–31. doi:10.1097/ALN.0b013e3182104884.
50. Leslie K, Wu CY, Bjorksten AR, Williams DL, Ludbrook G, Williamson E. Cardiac output and propofol concentrations in prone surgical patients. Anaesth Intensive Care. 2011;39(5):868–74.
51. Levi AD, Casella G, Green BA, Dietrich WD, Vanni S, Jagid J, et al. Clinical outcomes using modest intravascular hypothermia after acute cervical spinal cord injury. Neurosurgery. 2010;66(4):670–7. doi:10.1227/01.NEU.0000367557.77973.5F.
52. Li F, Gorji R, Allott G, Modes K, Lunn R, Yang ZJ. The usefulness of intraoperative neurophysiological monitoring in cervical spine surgery: a retrospective analysis of 200 consecutive patients. J Neurosurg

Anesthesiol. 2012;24(3):185–90. doi:10.1097/ANA.0b013e318255ec8f.

53. Liu EH, Goy RW, Tan BH, Asai T. Tracheal intubation with videolaryngoscopes in patients with cervical spine immobilization: a randomized trial of the Airway Scope and the GlideScope. Br J Anaesth. 2009;103(3):446–51. doi:10.1093/bja/aep164. Epub 2009 Jun 19.
54. Macias CA, Rosengart MR, Puyana JC, Linde-Zwirble WT, Smith W, Peitzman AB, Angus DC. The effects of trauma center care, admission volume, and surgical volume on paralysis after traumatic spinal cord injury. Ann Surg. 2009;249(1):10–7. doi:10.1097/SLA.0b013e31818a1505.
55. Manoach S, Paladino L. Manual in-line stabilization for acute airway management of suspected cervical spine injury: historical review and current questions. Ann Emerg Med. 2007;50(3):236–45. Epub 2007 Mar 6.
56. Manoach S, Paladino L. Laryngoscopy force, visualization, and intubation failure in acute trauma: should we modify the practice of manual in-line stabilization? Anesthesiology. 2009;110(1):6–7. doi:10.1097/ALN.0b013e318190b27b.
57. Mariappan R, Manninen P, Massicotte EM, Bhatia A. Circulatory collapse after topical application of vancomycin powder during spine surgery. J Neurosurg Spine. 2013;19(3):381–3.
58. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest. 2008;134(1):172–8. doi:10.1378/chest.07-2331.
59. Martyn JA, Richtsfeld M. Succinylcholine-induced hyperkalemia in acquired pathologic states: etiologic factors and molecular mechanisms. Anesthesiology. 2006;104(1):158–69.
60. Mayglothling J, Duane TM, Gibbs M, McCunn M, Legome E, Eastman AL, et al. Emergency tracheal intubation immediately following traumatic injury: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma Acute Care Surg. 2012;73(5 Suppl 4):S333–40. doi:10.1097/TA.0b013e31827018a5.
61. McLeod ADM, Calder I. Spinal cord injury and direct laryngoscopy—the legend lives on. Br J Anaesth. 2000;84(6):705–9.
62. Mehta S, Orenczuk K, McIntyre A, Willems G, Wolfe DL, Hsieh JT, et al. Neuropathic pain post spinal cord injury part 1: systematic review of physical and behavioral treatment. Top Spinal Cord Inj Rehabil. 2013;19(1):61–77. doi:10.1310/sci1901-61.
63. Middleton PM, Davies SR, Anand S, Reinten-Reynolds T, Marial O, Middleton JW. The pre-hospital epidemiology and management of spinal cord injuries in New South Wales: 2004–2008. Injury. 2012;43(4):480–5. doi:10.1016/j.injury.2011.12.010. Epub 2012 Jan 11.
64. Miller SM. Methylprednisolone in acute spinal cord injury: a tarnished standard. J Neurosurg Anesthesiol. 2008;20(2):140–2. doi:10.1097/01.ana.0000314442.40952.0d.
65. Muzevich KM, Voils SA. Role of vasopressor administration in patients with acute neurologic injury. Neurocrit Care. 2009;11(1):112–9. doi:10.1007/s12028-009-9214-z. Epub 2009 Apr 22.
66. Myburgh JA, Upton RN, Grant C, Martinez A. Epinephrine, norepinephrine and dopamine infusions decrease propofol concentrations during continuous propofol infusion in an ovine model. Intensive Care Med. 2001;27(1):276–82.
67. National Spinal Cord Injury Statistical Center. Facts and figures at a glance. Birmingham, AL: University of Alabama at Birmingham; 2013.
68. New York state in-hospital cervical spine clearance guidelines in blunt trauma [Internet]. New York: New York State Department of Health. 2013. http://www.health.ny.gov/professionals/ems/state_trauma/resources.htm.
69. Ney JP, van der Goes DN, Watanabe JH. Cost-benefit analysis: intraoperative neurophysiological monitoring in spinal surgeries. J Clin Neurophysiol. 2013;30(3):280–6. doi:10.1097/WNP.0b013e3182933d8f.
70. Nuwer MR, Emerson RG, Galloway G, Legatt AD, Lopez J, Minahan R, et al. Evidence-based guideline update: intraoperative spinal monitoring with somatosensory and transcranial electrical motor evoked potentials: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology and the American Clinical Neurophysiology Society. Neurology. 2012;78(8):585–9. doi:10.1212/WNL.0b013e318247fa0e.
71. Ostermeier A, Danz S, Unertl K, Grasshoff C. Risking or saving the patient's neck-relocation of an odontoid fracture by direct laryngoscopy and manual in-line stabilization during resuscitation. Resuscitation. 2011;82(1):137–8. doi:10.1016/j.resuscitation.2010.09.479. Epub 2010 Nov 2.
72. O'Toole JE. Timing of surgery after cervical spinal cord injury. World Neurosurg. 2013;pii:S1878–8750(13)00289–1.doi 10.1016/j.wneu.2013.02.024. [Epub ahead of print]
73. Palumbo MA, Aidlen JP, Daniels AH, Thakur NA, Caiati J. Airway compromise due to wound hematoma following anterior cervical spine surgery. Open Orthop J. 2012;6:108–13. doi:10.2174/1874325001206010108. Epub 2012 Mar 5.
74. Pastrana EA, Saavedra FM, Murray G, Estronza S, Rolston JD, Rodriguez-Vega G. Acute adrenal insufficiency in cervical spinal cord injury. World Neurosurg. 2012;77(3–4):561–3. doi: 10.1016/j.wneu.2011.06.041. Epub 2011 Nov 7.
75. Lee LA, Roth S, Todd MM, Posner KL, Polissar NL, Postoperative Visual Loss Study Group. Risk factors associated with ischemic optic neuropathy after spinal fusion surgery. Anesthesiology. 2012;116(1):15–24. doi:10.1097/ALN.0b013e31823d012a.
76. Rajkumar A, Bajekal R. Intraoperative airway obstruction due to dissection of a reinforced endotracheal tube

in a prone patient. J Neurosurg Anesthesiol. 2011;23 (4):377. doi:10.1097/ANA.0b013e31822cf882.

77. Ramasamy A, Midwinter M, Mahoney P, Clasper J. Learning the lessons from conflict: pre-hospital cervical spine stabilisation following ballistic neck trauma. Injury. 2009;40(12):1342–5. doi:10.1016/j.injury.2009.06.168. Epub 2009 Jul 17.
78. Redfors B, Shao Y, Omerovic E. Stress-induced cardiomyopathy in a patient with chronic spinal cord transection at the level of C5: endocrinologically mediated catecholamine toxicity. Int J Cardiol. 2012;159(3):e61–2. doi:10.1016/j.ijcard.2011.12.025. Epub 2012 Jan 10.
79. Robitaille A. Airway management in the patient with potential cervical spine instability: continuing professional development. Can J Anaesth. 2011;58 (12):1125–39. doi:10.1007/s12630-011-9597-0. Article in English, French. Epub 2011 Oct 27.
80. Robitaille A, Williams SR, Tremblay MH, Guilbert F, Thériault M, Drolet P. Cervical spine motion during tracheal intubation with manual in-line stabilization: direct laryngoscopy versus GlideScope videolaryngoscopy. Anesth Analg. 2008;106(3):935–41. doi:10.1213/ane.0b013e318161769e.
81. Roozmon P, Gracovetsky SA, Gouw GJ, Newman N. Examining motion in the cervical spine I: imaging systems and measurement techniques. J Biomed Eng. 1993;15(1):5–12.
82. Rudolph C, Schneider JP, Wallenborn J, Schaffranietz L. Movement of the upper cervical spine during laryngoscopy: a comparison of the Bonfils intubation fibrescope and the Macintosh laryngoscope. Anaesthesia. 2005;60(7):668–72.
83. Ryken TC, Hurlbert RJ, Hadley MN, Aarabi B, Dhall SS, Gelb DE, et al. The acute cardiopulmonary management of patients with cervical spinal cord injuries. Neurosurgery. 2013;72 Suppl 2:84–92. doi:10.1227/NEU.0b013e318276ee16.
84. Santoni BG, Hindman BJ, Puttlitz CM, Weeks JB, Johnson N, Maktabi MA, et al. Manual in-line stabilization increases pressures applied by the laryngoscope blade during direct laryngoscopy and orotracheal intubation. Anesthesiology. 2009;110 (1):24–31. doi:10.1097/ALN.0b013e318190b55.
85. Schriger DL, Larmon B, LeGassick T, Blinman T. Spinal immobilization on a flat backboard: does it result in neutral position of the cervical spine? Ann Emerg Med. 1991;20(8):878–81.
86. Siddall PJ, McClelland JM, Rutkowski SB, Cousins MJ. A longitudinal study of the prevalence and characteristics of pain in the first 5 years following spinal cord injury. Pain. 2003;103(3):249–57.
87. Theron A, Ford P. Management of acute cervical spine injury. Anaesthesia. 2008;24(1):30–4.
88. Thiboutot F, Nicole PC, Trépanier CA, Turgeon AF, Lessard MR. Effect of manual in-line stabilization of the cervical spine in adults on the rate of difficult orotracheal intubation by direct laryngoscopy: a randomized controlled trial. Can J Anaesth. 2009;56 (6):412–8. doi:10.1007/s12630-009-9089-7. Epub 2009 Apr 24.
89. Thiel D, Houten J, Wecksell M. Accidental tracheal extubation of a patient in the prone position. Anesthesia & Analgesia Case Reports. 2014;2(2):20–22.
90. Totten VY, Sugarman DB. Respiratory effects of spinal immobilization. Prehosp Emerg Care. 1999; 3(4):347–52.
91. Turkstra TP, Craen RA, Pelz DM, Gelb AW. Cervical spine motion: a fluoroscopic comparison during intubation with lighted stylet, GlideScope, and Macintosh laryngoscope. Anesth Analg. 2005; 101(4):1011.
92. Turkstra TP, Pelz DM, Jones PM. Cervical spine motion: a fluoroscopic comparison of the AirTraq Laryngoscope versus the Macintosh laryngoscope. Anesthesiology. 2009;111(1):97–101. doi:10.1097/ALN.0b013e3181a8649f.
93. Turkstra TP, Pelz DM, Shaikh AA, Craen RA. Cervical spine motion: a fluoroscopic comparison of Shikani Optical Stylet vs Macintosh laryngoscope. Can J Anaesth. 2007;54(6):441–7.
94. Wilson JR, Vaccaro A, Harrop JS, Aarabi B, Shaffrey C, Dvorak M, et al. The impact of facet dislocation on clinical outcomes after cervical spinal cord injury: results of a multicenter North American prospective cohort study. Spine (Phila Pa 1976). 2013;38 (2):97–103. doi:10.1097/BRS.0b013e31826e2b91.
95. Woller SA, Hook MA. Opioid administration following spinal cord injury: Implications for pain and locomotor recovery. Exp Neurol. 2013. pii: S0014–4886(13)00091–5. doi: 10.1016/j.expneurol.2013.03.008. [Epub ahead of print]
96. Yoo KY, Jeong CW, Kim SJ, Jeong ST, Shin MH, Lee J. Cardiovascular and arousal responses to laryngoscopy and tracheal intubation in patients with complete spinal cord injury. Br J Anaesth. 2009; 102(1):69–75. doi:10.1093/bja/aen317. Epub 2008 Nov 5.
97. Yoo KY, Jeong CW, Kim SJ, Jeong ST, Kwak SH, Shin MH, et al. Altered cardiovascular responses to tracheal intubation in patients with complete spinal cord injury: relation to time course and affected level. Br J Anaesth. 2010;105(6):753–9. doi:10.1093/bja/aeq267. Epub 2010 Oct 5.