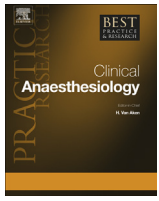
Best Practice & Research Clinical Anaesthesiology 30 (2016) 27–39

Contents lists available at ScienceDirect

Best Practice & Research Clinical Anaesthesiology

journal homepage: www.elsevier.com/locate/bean



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Acute spinal cord injury

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Keywords:
spinal cord injuries
spinal cord compression
cervical cord
trauma, nervous system
methylprednisolone
GM-1 ganglioside
anesthesia

Acute spinal cord injury (SCI) is a devastating event with high mortality and, among survivors, a high degree of morbidity due to both motor and sensory deficits. The damage that occurs with a SCI is recognized to be pathophysiologically biphasic: the initial insult to neural tissue can be followed by a secondary process of progressive ischemia that may worsen the severity of dysfunction. The extent of this secondary insult is potentially modifiable, and a variety of interventions have been studied in an attempt to improve motor and sensory outcomes. What follows is a brief review of some such procedural and pharmacologic interventions, including early decompressive surgery, use of methylprednisolone, and blood pressure and respiratory management, which have been proposed to improve outcomes after SCI.

Introduction

The annual incidence of traumatic spinal cord injury (SCI) varies throughout the world [1], but in all countries, many patients with acute SCI present for surgery shortly after their injury – whether to stabilize or decompress the spinal cord, or to manage concurrent non-spinal traumatic injuries. Published retrospective studies and case series have suggested that over 70% of the patients with SCI will undergo surgical intervention on the spine within a week of injury [2,3], and practice guidelines published subsequent to these studies advocate early surgical treatment [4]. Anesthesia providers should therefore have a good understanding of current recommendations for the pharmacologic management and cardiopulmonary support of such patients.

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http://dx.doi.org/10.1016/j.bpa.2015.11.003
1521-6896/

The severity of SCI is commonly described in studies by the American Spinal Injury Association Impairment Scale (AIS) [5], which uses an alphabetical grade to describe the completeness of injury at a given spinal nerve level. Higher grades represent more complete injuries, with AIS A referring to a complete lack of motor or sensory function below the neurological level of injury (see Table 1). Neurologic levels are defined by the relationship of segmental nerve roots to numbered vertebra in the bony spinal column, and can be identified on clinical exam by the area of skin innervated by the sensory nerve axons and the function of muscles innervated by motor nerve axons of a given level (see Fig. 1). With the exception of a central cord lesion, an injury at a given spinal level will tend to create deficits at all levels below the injury. Thus, a complete (AIS A) SCI at the C3 level will demonstrate a loss of sensation in all parts of the body below the supraclavicular fossa and a loss of motor function in all extremities, the abdomen, and most of the thorax. An incomplete (AIS B–D) SCI at the C3 level will display at least some residual motor or sensory function in all parts of the body. This nomenclature has been considered throughout the rest of this review.

Mechanisms of injury

The most common causes of acute SCI are motor vehicle accidents, falls, directed violence such as gunshot wounds, and sporting accidents [6–8]. underlying spinal diseases such as rheumatoid arthritis or cervical spondylosis/stenosis may make some patients more susceptible to certain forms of traumatic SCI, but many patients are relatively young (average age of injury in the United States was 37 in 2010) [6] without other major preexisting comorbidities at the time of injury. Injury to the bony vertebral column with both fracture and dislocation at a vertebral joint is a common precipitant of nerve injury [8]. Ligamentous tearing, herniation of the intervertebral disk, or less common mechanisms such as high-voltage electrical injury or decompression injury from scuba diving accidents could lead to SCI even in the absence of bony injury [9]. The cord may rarely be completely transected, but most injuries leave at least some intact axons that cross the level of injury [7]. These spared axons provide the theoretical potential for some degree of functional neurologic recovery.

Basic science studies have shown that an initial incomplete injury can rapidly progress to a more complete injury due to a process of inflammatory or ischemic edema, hemorrhage, or other causes of hypoxia or progressive compression [7]. Based on an early work with animal models, spinal cord edema is believed to reach a maximum about 3 days after initial injury, and probably continues for a few weeks [10]. Spinal cord ischemia can extend above the level of initial injury; hence, surgical and pharmacological therapy of SCI in this time period is meant to avoid or minimize such progression in the level and grade of injury.

Indication for surgical intervention

A meta-analysis of human studies from 2004 [11] and more recent multicenter prospective cohort studies [12,13] have suggested that decompressive surgery (including open reduction of fractures or

Table 1
ASIA Impairment Scale (AIS). The AIS is meant to encourage precision in the definition of neurologic injury levels and to standardize the reporting of the extent of incomplete injury [5]. The neurological level of injury is defined as the most rostral spinal level that demonstrates "unimpaired" function. Muscle function is rated from 0-total paralysis to 5-normal with full range of motion against significant resistance.

A = **Complete**. No sensory or motor function is preserved in the sacral segments S4–5.
B = **Sensory Incomplete**. Sensory function, including that of the sacral segments S4–5 (light touch or pin prick at S4–5 or deep anal pressure), is preserved, AND no motor function is preserved more than three levels below the motor level on either side of the body.
C = **Motor Incomplete**. Some motor function is preserved below the neurological level of injury but at least half of the key muscle functions below the level of injury have a muscle grade <3 (e.g., are profoundly weak or plegic).
D = **Motor Incomplete**. Motor function is preserved below the neurological level of injury, with at least half of key muscle functions having a muscle grade ≥3 (e.g., movement against gravity is possible).
E = **Normal**. Recovery to normal sensation and motor function in all segments after a patient had prior deficits. Someone without an initial SCI does not receive an AIS grade.