



The role and timing of early decompression for cervical spinal cord injury: Update with a review of recent clinical evidence

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Summary¹ It remains controversial whether early decompression following spinal cord injury conveys a benefit in neurological outcome. The goal of this paper is to provide evidence-based recommendations regarding spinal cord decompression in patients with acute spinal cord injury. We performed a Medline search of experimental and clinical studies reporting on the effect of decompression on neurological outcome following spinal cord injury. Animal studies consistently show that neurological recovery is enhanced by early decompression. One randomized controlled trial showed no benefit to early (< 72 h) decompression, however, several recent prospective series suggest that early decompression (< 12 h) can be performed safely and may improve neurological outcomes. A recent meta-analysis showed that early decompression (< 24 h) resulted in statistically better outcomes compared to both delayed decompression and conservative management. Currently, there are no standards regarding the role and timing of decompression in acute spinal cord injury. We recommend urgent decompression of bilateral locked facets in patients with incomplete tetraplegia or in patients with spinal cord injury experiencing neurological deterioration. Urgent decompression in acute cervical spinal cord injury remains a reasonable practice option and can be performed safely.

Introduction

The role and timing of surgical decompression after an acute spinal cord injury (SCI) remains one of the most controversial topics pertaining to spinal surgery. Despite an enormous amount of interest and research in SCI and an increased understanding of the secondary injury cascade pathways involved after an acute SCI, the neurological prognosis for a patient with a severe SCI remains dismal. Given

that approximately 10,000–12,000 SCIs occur annually in the United States [1–4], and that recent statistics suggest that approximately 280,000 people are currently living with a SCI in North America (www.spinalcord.uab.edu/; www.icord.org/), SCI represents an important cause of morbidity and mortality. Furthermore, the majority of patients with SCI are young [3], and the economic and societal impact is enormous, both to the immediate family and to society at large. During 1995, in the United States alone, the total direct costs associated with all causes of SCI were \$7,736 billion [1]. Given this background, it behooves us to consider the evidence regarding decompression of acute SCI, particularly

¹ Abstracts in German, French, Italian, Spanish, Japanese, and Russian are printed at the end of this supplement.

given the frequency of this intervention in North America [5, 6].

It is now well recognized that acute SCI involves both primary and secondary injury mechanisms [7-10]. The primary mechanism involves the initial mechanical injury due to local deformation and energy transformation that occurs within the spinal cord at the moment of injury, which is irreversible. In the majority of cases, primary SCI is caused by rapid spinal cord compression due to bone displacement from a fracture-dislocation or burst fracture [11-13]. Other potential mechanisms include acute spinal cord distraction, acceleration-deceleration with shearing, and laceration from penetrating injuries [2, 14].

The concept of secondary mechanisms of injury following primary SCI was first postulated by Allen in 1911 [15]. There is now considerable evidence that the primary mechanical injury initiates a cascade of secondary injury mechanisms such as: vascular changes, including ischemia, loss of autoregulation, neurogenic shock, hemorrhage, loss of microcirculation, vasospasm, and thrombosis [8, 10, 16], electrolyte derangements, including increased intracellular calcium, increased extracellular potassium, and accumulation of intracellular sodium [8, 17, 21], accumulation of neurotransmitters, including serotonin, catecholamines, and extracellular glutamate [8, 18, 22], excitotoxicity [8, 23], arachidonic acid release, production of eicosanoids and free radicals, and lipid peroxidation [24-28], endogenous opioids [8, 29, 30], edema [31], inflammation [32], loss of energy metabolism, including adenosine triphosphate-dependent cellular processes [25], and apoptosis [8, 33-36]. Secondary injury is preventable, and may be reversible.

The increased understanding of the pathophysiology of acute SCI has led to clinically relevant neuroprotective therapies to attenuate the effects of the secondary injury. The National Acute Spinal Cord Injury Studies (NASCIS II and NASCIS III) have reported a modest beneficial effect of high-dose methylprednisolone if given within eight hours of injury in patients with SCI [37, 38], and suggested that treatment within three hours may be better than treatment initiated 3-8 hours after trauma [39]. These studies support the concept of targeting secondary mechanisms in acute SCI, and also emphasize the importance of the timing of intervention.

The development of these secondary injury events, which lead to tissue destruction during the first few hours after injury, is relevant to the surgical treatment of SCI as well. There is experimental evidence that persistent compression of the spinal cord is a potentially reversible form of secondary injury [14, 16, 40-56]. However, despite its widespread

use in patients with acute SCI in North America, the role of surgery in improving neurological recovery remains controversial because of the absence of well-designed and well-executed randomized controlled trials. The presence and duration of a therapeutic window, during which surgical decompression could mitigate the secondary mechanisms of SCI, remains unclear.

This paper will review the experimental and clinical evidence regarding the value of decompressive surgery in treating patients with acute, non-penetrating SCI, and compare these data with results of the conservative, non-operative management of SCI. This article represents an updated analysis of previous reports from our institution [57, 58].

Methods

We conducted a computer-assisted Medline search of the experimental and clinical literature from 1966 to 2004 dealing with the role of decompression in SCI using the medical subject headings (MeSH) of "decompression" and "spinal cord injury". Articles with English, German, and French abstracts were selected for review. This computerized literature review yielded a total of 960 studies, which were then pared down based on relevance to the issue of SCI management. This analysis was supplemented by a detailed examination of the reference lists from selected articles and standard spine textbooks. As summarized in Table 1, evidence from clinical trials was defined as Class I (well-designed and well-conducted randomized controlled trials), Class II (prospective cohort studies or controlled studies with well-defined comparison groups), or Class III (case series, retrospective reviews, and expert opinion) [59]. Based on this analysis, we suggested evidence-based recommendations regarding the role of acute decompression in SCI.

Study design	Class of evidence
Well-designed and well-executed randomized controlled clinical trials	I
Prospective, non-randomized studies	II
Case series; retrospective studies; expert opinion	III

Table 1: Classification of clinical evidence.

Results

A total of 65 articles (19 experimental studies in animal models [14, 16, 40-55, 60] and 46 clinical studies) were selected for detailed analysis (Tables 2-6). Of the clinical articles, nine [61-69] dealt with non-operative management, 31 [5, 6, 62, 63, 68, 69, 70-94]

with the role of early (< 4 weeks) surgical intervention (Tables 3 and 4), twelve [5, 70, 84, 85, 95-102] with the effect of closed reduction (Table 5), and seven [78, 103-108] with the role of delayed decompression (Table 6). Based on this analysis, we suggested evidence-based recommendations regarding the role of acute decompression in SCI (Table 7).

Species	Reference	Injury model	Timing of decompression	Conclusion
Primates	Kobrine et al 1978 [48], 1979 [49]	Extradural balloon	1-15 min	Early decompression promotes NR.
Cats	Brodkey et al 1972 [41]	Static weight	Minutes	Early removal of weight restores axonal conduction.
	Croft et al 1972 [44]	Static weight	5-20 min	Early decompression at milder severities of injury promotes NR.
	Thienprasit et al 1975 [55]	Extradural balloon	6 hr	Laminectomy ineffective in promoting NR.
Dogs	Bohlman et al 1979 [40]	Anterior piston	3-8 wk	Decompression promotes NR.
	Aki and Toya 1984 [16]	Static load 6-60g	30, 60 min	Vascular changes more severe with greater degrees of compression.
	Delamarter et al 1995 [45]	Circumferential cable	1 hr-1 wk	Decompression at 1 hr improves NR.
	Carlson et al 1997 [43]	Piston	5 min, 1 hr, 3 hr	Recovery of evoked potentials in 5 min and 1 hr groups.
	Carlson et al 2003 [42]	Piston	30 min, 3 hr	Early decompression promotes NR.
Rats	Dolan et al 1980 [14]	Clip compression		Neurological impairment increased with greater severities of injury.
	Guha et al 1987 [47]	Clip compression 2.3, 16.9, 53.0g	15 min, 1 hr, 2 hr, 4 hr	Early decompression at milder severities of injury promotes NR.
	Nystrom and Berglund 1988 [50]	Static load 20, 35, 50g	1 min, 5 min, 10 min	Neurological impairment increased with greater severities of injury.
	Zhang et al 1993 [60]	Static load 9, 35, 50g	5 min	Increased levels of lactate, inosine, hypoxanthine with SCI; reversal of metabolic changes with decompression.
	Dimar et al 1999 [46]	Extradural pacer and impactor	0-72 hr	Early decompression with minimal canal compromise promotes NR.

*NR: neurological recovery

Table 2: Experimental studies assessing the role of decompression in animal models of SCI.

Reference	# of patients (level)	Timing of intervention	Study design	Conclusions
Tator et al 1987 [87]	208 (all levels) 75 reduced or decompressed 41 fusion 92 non-operative	< 4 wk	Prospective, non-randomized	Surgery did not improve NR or change length of stay.
Duh et al 1994 [76]	487 (all levels) 303 decompressed early 38 late 105	Early < 25 hr Late > 200 hr	Prospective, non-randomized	Surgical decompression < 25 hr or > 200 hr may be associated with better NR.
Waters et al 1996 [90]	269 (all levels) 142 non-operative 127 decompressed	Average > 14 d	Prospective, non-randomized	Surgery of no benefit; however, all patients had delayed surgery.
Vaccaro et al 1997 [88]	62 (cervical) early 34 late 38	Early < 72 hr Late > 5 d	Prospective, randomized; however, 20 lost to follow-up	No difference in NR between early and late surgery groups.
Vale et al 1997 [89]	77 (all levels) 58 operated	11 < 24 hr 13 24-72 hr 34 > 72 hr	Prospective, non-randomized	No clear relationship between NR and timing of surgery.
Chen et al 1998 [63]	37 (cervical) 16 decompressed 21 non-operative	< 2 wk	Prospective, non-randomized	Surgery associated with improved NR and fewer complications.
Waters et al 1999 [91]	2204 (all levels)	88% admitted < 72 hr	Prospective, non-randomized	Surgery does not increase complication rates of SCI patients.
Ng et al 1999 [84]	26 (cervical)	7 had surgical decompression < 12 hr	Prospective, non-randomized	Surgical decompression within 8 hr of injury was feasible in 8%, and not associated with increased complication rates.
Pointillart et al 2000 [86]	106 (all levels) 58 (cervical)	49 had surgical decompression < 8 hr	Prospective, non-randomized	Early surgery did not improve NR.
Papadopoulos et al 2002 [85]	91 (cervical) 66 decompressed 25 non-operative	34 had surgical decompression < 10 hr	Prospective, non-randomized	Early decompression and surgery is feasible, may improve NR, and reduces hospital stay.

*NR: neurologic recovery

Table 3: Prospective clinical studies of surgical decompression in acute SCI.

Reference	# of patients (level)	Timing of intervention	Conclusions
Larson et al 1976 [78]	44 (thoracic, lumbar) < 1 wk 8 > 1 wk 36	Early < 1 wk Late > 1 wk	No difference in NR between early and late surgery; preferable to wait.
Maynard et al 1979 [80]	123 (cervical) 51 early 10 late 62 non-operative	Early < 4 wk Late > 4 wk	Early surgery not associated with improved NR.
Wagner and Chehrizi 1982 [6]	44 (cervical) 22 decompressed 20 non-operative	Early < 8 hr Late 8-48 hr	Early surgery no better than late surgery; surgery does not improve NR.
Bohlman et al 1985 [62]	218 (T1-T10)	Most late (> 1 wk)	No complete injuries improved with surgery; anterior approach best for NR in incomplete injuries.
McEvoy and Bradford 1985 [68]	53 (thoracic, lumbar) 31 decompressed 22 non-operative	Early < 2 wk Late > 2 wk	Surgery for incomplete neurological deficit may lead to NR.
McAfee et al 1985 [81]	70 (thoracic, lumbar)	Same day - 11 mo (average 60 d)	No relationship between time to decompression and NR; 32/47 improved at least 1 motor grade.
Aebi et al 1986 [70]	100 (cervical) 100 decompressed	75% reduced < 6 hr	Early reduction and surgery resulted in improvement in 31%.
Benzel and Larson 1986 [71]	105 (thoracic, lumbar)	All delayed (19-48 d)	Anterior decompression and fusion results in better NR than fusion alone or non-operative management.
Benzel and Larson 1986 [73]	35 (cervical; all complete) 25 anterior approach 10 posterior approach	1-4 wk	Anterior decompression improves nerve root recovery.
Benzel and Larson 1987 [72]	99 (cervical)	All delayed (17-52 d)	Surgery improves NR in incomplete SCI; no relationship between time to decompression and NR.
Donovan et al 1987 [75]	61 (cervical) 18 decompressed 43 non-operative	All < 3 wk	No difference in NR between decompressed and non-operative groups.
Wiberg and Hauge 1988 [93]	30 (thoracic, lumbar)	8 < 24 hr 2 1-7 d 20 > 7 d	Decompression improves NR; 2 patients with complete SCI operated within 10 hr showed NR.
Weinshel et al 1990 [92]	90 (cervical; ASIA A/B)	6 hr-60 days (average 13 d)	Decompression improves NR.
Murphy et al 1990 [83]	102 (cervical) 58 decompressed 44 non-operative	Early < 2 wk	Early surgery reduced length of stay and promoted earlier rehabilitation.
Wolf et al 1991 [94]	52 (cervical; bilateral locked facets)	40 reduced < 4 hr by traction; 23 had surgery < 24 hr	Early reduction of bilateral locked facets results in NR in complete and incomplete SCI.
Levi et al 1991 [79]	103 (cervical) 45 early 58 late	Early < 24 hr Late > 24 hr	Early surgery not associated with NR, but may benefit overall management.

Table 4: Retrospective case series of surgical decompression in acute SCI (continued on page 18)

Reference	# of patients (level)	Timing of intervention	Conclusions
Krengel et al 1993 [77]	14 (T2-T11 incomplete)	12 < 24 hr 1 36 hr 1 5 d	Early surgical reduction and decompression is safe and improves NR.
Petitjean et al 1995 [69]	49 (thoracic)	Early average 12 hr Late average 9 d	Decompression of no benefit in complete thoracic paraplegia.
Botel et al 1997 [74]	255 (all levels) 178 decompressed 51.4% early 10.5% late	Early < 24 hr Late > 2 wk	No NR in complete SCI; no association of NR to timing of decompression.
Mirza et al 1999 [82]	30 (cervical)	15 < 72 hr 15 > 72 hr	Decompression improves NR and does not increase complication rates.
Tator et al 1999 [5]	585 (all levels)	23.5% had surgery < 24 hr	65% of patients in North America with SCI have surgery; no consensus on timing of intervention.

*NR: neurological recovery

Table 4: Retrospective case series of surgical decompression in acute SCI.

Reference	# of patients	Treatment	Outcome
Burke and Berryman 1971 [96]	76	Closed reduction under anesthesia; 50% admitted < 8 hr	Early reduction improved NR in incomplete SCI.
Dall 1972 [97]	75	Timing of reduction not specified	Anatomical reduction of fracture-dislocations does not improve NR.
Harris et al 1980 [99]	145	Traction protocol not specified	Reduction of cervical dislocations does not improve NR.
Sonntag 1981 [101]	15	11 had closed reduction; 4 had open reduction	Root recovery in 2 cases; transient root palsy in 1 case.
Aebi et al 1986 [70]	100	75% reduced < 6 hr	Early reduction results in NR in 31%.
Brunette and Rockswold 1987 [95]	1	C3-C4 reduced < 90 min	Improvement from ASIA A to ASIA E by rapid reduction of C3-C4 dislocation.
Star et al 1990 [102]	53	39 required > 50 lb	68% had NR; no deterioration.
Hadley et al 1992 [98]	54	10 reduced < 8 hr	Reduction < 8 hr resulted in NR in all 10 patients.
Lee et al 1994 [100]	210	Rapid traction $\leq$ 150 lb	Rapid reduction improves NR.
Ng et al 1999 [84]	26	11 reduced by traction at average < 12 hr	Traction is the most practical method of achieving urgent decompression after SCI.
Tator et al 1999 [5]	173	Traction used in only 47%	Traction associated with 8.1% incidence of neurological deterioration; no consensus as to timing, role, and upper limit of traction.
Papadopoulos et al 2002 [85]	91	32 reduced by traction alone at average < 8 hr	Early reduction results in NR.

*NR: neurologic recovery

Table 5: Studies of closed reduction in acute SCI.

Reference	# of patients	Timing of surgery	Outcome
Larson et al 1976 [78]	25 (all levels)	All > 4 wk; 15 > 6 mo	Improved NR with surgery in complete and incomplete SCI.
Bohlman and Freehafer 1975 [105]	36 (all levels) 32 cervical 4 thoracolumbar	Average 22 mo	8/27 patients with incomplete SCI had motor recovery; 29/36 patients had nerve root recovery.
Maiman et al 1984 [107]	20 (thoracic, lumbar)	1 mo-5 yr	85% attained NR.
Transfeldt et al 1992 [108]	49 (thoracic, lumbar)	> 3 mo	46.5% of incomplete SCI had NR.
Anderson and Bohlman 1992 [103]	51 (cervical; motor complete)	1 mo-8 yr (average 15 mo)	Improved segmental (1-2 levels) recovery in 25 cases; improved distal cord function in 1 case.
Bohlman and Anderson 1992 [104]	58 (cervical; motor incomplete)	1 mo-9 yr (average 13 mo)	Improved NR in 46 cases.
Bohlman et al 1994 [106]	25 (thoracic, lumbar)	Average 4.5 yr	84% had NR; proportion incomplete unclear.

*NR: neurological recovery

Table 6: Retrospective case series of late (> 4 weeks) decompression following SCI.

Level of recommendation	Class of evidence	Details
Standards	I	There are no standards regarding the role and timing of decompression in acute SCI.
Guidelines	II	Early surgery (< 24 hr) can be done safely in patients with SCI. There are limited data to support a recommendation for urgent reduction of bilateral locked facets in a patient with incomplete tetraplegia. There are limited data to support a recommendation for urgent decompression in a patient with SCI experiencing neurological deterioration.
Options	III	Decompression is recommended at the level of a reasonable practice option in acute cervical SCI; when possible, (excluding patients with life-threatening multi-system trauma) it is recommended that urgent decompression be performed with 24 hr of SCI.

Table 7: Evidence-based recommendations for decompression after acute SCI.

Experimental studies of decompression in acute SCI in animal models

There is compelling evidence from laboratory studies in animal models that persistent compression of the spinal cord is a potentially reversible form of secondary injury. The severity of SCI in animal models is related to the force of compression, duration of compression, displacement, impulse, and kinetic energy [14, 47, 53, 109-111]. Numerous experimental studies of decompression after SCI have been performed in various animal models including primates, dogs, cats, and rodents [14, 16,

40-55, 60]. These studies have consistently shown that neurological recovery is enhanced by early decompression (Table 2).

The most convincing experimental evidence that spinal cord decompression after SCI is beneficial was provided by Dimar et al in 1999 [46]. Using a weight drop model followed by placement of an epidural spacer to simulate persistent compression, a thoracic SCI was performed in rats. The effect of decompression at 0, 2, 6, 24, and 72 hours after SCI was then assessed by quantitative analysis of locomotor recovery, lesion volume, and electrophysiology. Neurological recovery was inversely

related to the duration of compression, with statistically significant differences seen in all experimental groups. Carlson et al also reported similar results in dogs [42]. A piston was used to create a SCI, and the effect of decompression at 30 or 180 minutes was assessed by somatosensory evoked potentials, locomotor function, and lesion volume. Functional recovery was significantly better, and lesion volume was significantly smaller in those animals undergoing early decompression.

Role of conservative management in acute SCI

To evaluate the possible role of surgery in the management of SCI in the appropriate context, it is essential to examine the outcomes of conservative, non-operative treatment [61-69]. Guttman advocated the use of postural techniques combined with bed rest to achieve reduction and spontaneous fusion of the spine [112, 113]. Operative management was avoided because laminectomy was associated with a higher incidence of neurological complications and worse clinical outcomes [61, 112-116]. Frankel, who adhered to these principles, reported on a cohort of 612 patients with closed spinal injuries [65]. Only four patients developed delayed instability. Importantly, 29% of Frankel A patients (complete motor and sensory paralysis below the level of the injury) improved at least one grade during their hospital stay. These data provide the benchmark against which other interventions can be compared.

Spontaneous neurological improvement with non-operative management has also been reported by others [61, 114-115, 117-121]. Some authors have reported that neither spinal surgery nor anatomical realignment of the spinal column improved neurological outcome in patients sustaining acute SCI, with the possible exception of bilateral locked facets [97, 99, 114]. However, all non-operative management studies to date have been limited to non-controlled, retrospective analyses of clinical databases (Class III evidence). Furthermore, surgeons are now aware that laminectomy with fusion is contraindicated in most cases of acute SCI because it usually provides inadequate decompression of the spinal cord, and may exacerbate the underlying spinal instability [74, 122].

While meticulous non-operative management remains an essential component in the care of patients with SCI, modern spine surgery has advanced significantly over the past two decades. Furthermore, non-operative management is not without its attendant risks; up to 10% of patients with incomplete cervical

SCI deteriorate neurologically while being managed in an exclusively non-operative manner [66].

Role of decompression in the management of acute SCI

Our review of the literature identified ten prospective, controlled studies of surgical decompression in acute SCI (Table 3) [63, 76, 84-91]. Tator et al reported a prospective, non-randomized case-control study of 208 patients with acute SCI or cauda equina injury [87]. Operative management was associated with a lower overall mortality rate despite a higher thromboembolic complication rate. There was no difference in neurological recovery between the two groups.

Duh et al, using data from NASCIS II (Class II evidence), reported that patients undergoing acute surgery (< 25 hours after injury) had improved outcomes compared to a conservatively managed control cohort, although the results were not statistically significant [76]. Interestingly, results of surgery were similar in the early (< 25 hours) and delayed (> 200 hours) groups. More recently, Papadopoulos et al evaluated 91 patients with acute cervical SCI to assess the feasibility and outcome of an immediate decompression treatment protocol [85]. All except one patient were admitted within nine hours of their injury. They reported that 39/66 patients in the protocol group improved, including some patients presenting with a complete SCI, compared to 6/25 in the control group. This study was classified as Class II evidence due to the lack of randomization.

In contrast, the prospective studies by Vale et al [89], Vaccaro et al [88], and Waters et al [90] were unable to document a beneficial effect of surgical decompression. It is noteworthy, however, that all patients underwent delayed operative management in the study by Waters et al. Moreover, although the study by Vaccaro et al was a prospective, randomized trial, 20 of the 62 patients were lost to follow-up and "early" surgery was defined as being within 72 hours after SCI. In view of the large number of patients lost to follow-up, we have considered this study to provide Class II evidence.

A number of authors have advocated early reduction (4-10 hours) and operative fixation of spinal fractures in patients with acute SCI [70, 93, 94, 98]. These studies suggest that early decompression may enhance neurological recovery in selected patients with SCI. Most of these studies lack randomization or appropriate controls, however, and thus represent Class III evidence only.

The clinical benefits of early reduction of fracture-dislocations of the spine by closed techniques

or open surgery are difficult to assess in the absence of Class I data (Tables 3-5) [70, 82, 84, 92, 95, 101, 102, 123, 124]. While reports of significant neurological improvement in some cervical cases decompressed by early traction are encouraging, they do not provide sufficient evidence to support standards or guidelines [85, 94, 95, 98]. Moreover, a number of studies did not find any neurological benefit with reduction [90, 97, 125], with the possible exception of patients with bilateral facet dislocation [103]. Of particular relevance is the study by Burke and Berryman in which 76 patients with unilateral or bilateral dislocations of the cervical spine were treated with closed reduction under general anesthesia [96]. 50% of the patients were admitted within eight hours of injury, and the authors concluded that early reduction improved the neurological recovery of patients with incomplete SCI. Based on these data and a number of other Class II studies (Table 5) [5, 70, 84, 85, 95, 97-102], we can support a recommendation for urgent reduction of bilateral locked facets in patients with incomplete tetraplegia.

Aebi et al undertook a retrospective review of 100 patients with cervical spine injuries and attempted to find an association between neurological recovery and the timing of fracture reduction by closed or open techniques (Class III evidence) [70]. A manual or surgical reduction was performed within the first six hours after the accident in only 25% of the cases, and within the first 24 hours in 57%. Overall, 31% of the 100 patients recovered, and 75% of the recoveries were in patients reduced within the first six hours. Cotler et al examined the safety and efficacy of early reduction and undertook a prospective study of early reduction by traction in 24 patients (Class II evidence) [123]. They found no neurological deterioration in any of the patients, most of whom were successfully reduced by closed techniques within 24 hours of injury. Papadopoulos et al prospectively examined 91 patients with cervical SCI, 32 of whom had immediate spinal cord decompression by traction alone. They suggested that patients who were decompressed by closed reduction alone (mean time to decompression 6.0 hours) had better neurological outcomes than those requiring surgical decompression (mean time to decompression 12.6 hours) [85]. This qualifies as Class II evidence.

Despite the potential appeal of aggressive, closed reduction of locked cervical facets, Tator et al documented an 8.1% rate of neurological deterioration with attempts at closed reduction in 585 cases [5]. These data are sobering and emphasize the difficulty in interpreting accounts of the beneficial effects of rapid closed reduction by traction in the absence of Class I data.

In contrast to the aforementioned studies of early decompression, Larson et al advocated operating a week or more after SCI to allow medical and neurological stabilization of the injured patient [78]. This approach remains the practice in many institutions, particularly in light of early reports suggesting an increased rate of medical complications with early surgery (< 5 days after SCI) [126]. Interestingly, a number of authors have documented recovery of neurological function after delayed decompression of the spinal cord (months to years) after the injury (Table 6) [78, 103, 104, 106, 108, 127]. Although these studies are retrospective in design (Class III evidence), the improvement in neurological function with delayed decompression in patients with cervical or thoracolumbar SCI who have plateaued in their recovery is noteworthy, and suggests that compression of the cord is an important contributing cause of neurological dysfunction.

Effect of surgery on complications and length of stay after SCI

The issue of whether surgery, especially early surgery, increases the rate of complications in patients with SCI has been one that has generated considerable controversy and debate. Many patients with SCI with high tetraplegia or significant associated systemic injuries are critically ill because of cardiorespiratory compromise. Many authors have argued against surgery, especially early intervention in these critically ill patients [112-114, 120, 126]. However, modern techniques of spine surgery as well as advances in critical care and neuroanesthesia have allowed these patients to undergo surgery with minimal differences in complication rates between operative and non-operative cases [71, 87, 89, 122, 128, 129, 130-132]. Indeed, Duh et al showed that those operated on in the first 24 hours had a lower rate of complications than those undergoing operative intervention at later timepoints [76]. Waters et al, in a prospective study of 2,204 cases, found that there was no difference in the complication rates of cases managed by non-operative or surgical techniques [90, 91]. In a study from our unit, the only difference in morbidity between the surgical and non-surgical cases was a slight increase in the incidence of deep venous thrombosis in the surgical group [87]. Furthermore, the length of stay in the two groups did not differ. In the prospective, randomized trial by Vaccaro et al, there was no significant difference in length of acute postoperative intensive care stay or length of inpatient rehabilitation between the early and late groups [88]. This finding has been reiterated by others as well [63,

82]. Accordingly, there is Class II evidence to support the safety of surgery, including operative treatment within the first 24 hours.

Meta-analysis of the current literature of the role of decompression in acute SCI

La Rosa et al recently published a meta-analysis of the literature addressing the issue of early decompression and its role in acute SCI [133]. They reviewed all of the published clinical studies up to the year 2000, and were able to extract data on 1,687 patients. Patients were divided into three treatment groups: early decompression (< 24 hours), delayed decompression (> 24 hours), and conservative management. Statistically, early decompression resulted in better outcomes compared to both delayed decompression and conservative management. However, the author performed an analysis of homogeneity, and only data regarding patients with incomplete SCI who underwent early decompression were reliable. They concluded that early decompression can only be considered as a practice option [133]. Their conclusions are in concordance with ours.

Current studies of the role of decompression in acute SCI

In an attempt to provide a definitive answer regarding the role of early decompression, our institution has initiated a multicenter, prospective, randomized trial to evaluate the effect of early (≤ 12 hours post-injury) versus late (24 hours) decompressive surgery for cervical SCI (Surgical Timing in Acute Spinal Cord Injury Study; STASCIS). While such aggressive treatment protocols demand expedient care and cooperation between spine surgeons and radiologists, they are feasible [84, 85]. A total of 450 patients are required for this study, which is currently open for enrollment.

Conclusion

There is strong experimental evidence from animal models that decompression of the spinal cord improves recovery after SCI. However, it is difficult to determine a time window for the effective application of decompression in the clinical setting from these animal models. Studies of secondary injury mechanisms including ischemia, free radical-mediated lipid peroxidation, and calcium-mediated cytotoxicity, suggest that early intervention within hours

of SCI is critical to obtain a neuroprotective effect. Whether a similar time window applies to surgical treatment is as yet unclear, but seems likely. To date, the clinical studies that have examined the role of surgical decompression in SCI are limited to Class II and III evidence, except for one study. Surgery remains a valid practice option, although there are no conclusive data showing a benefit over conservative management approaches. There is Class II evidence suggesting that early surgical intervention is safe and effective, and even delayed decompression may convey a neurological benefit. Clearly, what is needed to definitely answer the question regarding the timing of surgery following SCI is a well-designed, prospective, randomized controlled, multicenter trial producing Class I data. Such a study is currently in progress. In general, surgery should be attempted as early as clinically feasible, once all appropriate diagnostic studies have been performed and the patient is medically stable. This can often be done within 24 hours of admission.

References

1. DeVivo MJ (1997) Causes and costs of spinal cord injury in the United States. *Spinal Cord*; 35(12):809-813.
2. Kraus JF, Franti CE, Riggins RS, et al (1975) Incidence of traumatic spinal cord lesions. *J Chronic Dis*; 28(9):471-492.
3. Sekhon LH, Fehlings MG (2001) Epidemiology, demographics, and pathophysiology of acute spinal cord injury. *Spine*; 26(24 Suppl):S2-12.
4. Tator CH (1995) Epidemiology and general characteristics of the spinal cord injury patient. *Benzel E and Tator CH (eds), Contemporary Management of Spinal Cord Injury. Neurosurgical Topics*. Park Ridge, IL: American Association of Neurological Surgeons.
5. Tator CH, Fehlings MG, Thorpe K, et al (1999) Current use and timing of spinal surgery for management of acute spinal surgery for management of acute spinal cord injury in North America: results of a retrospective multicenter study. *J Neurosurg Spine*; 91(1):12-18.
6. Wagner FC, Jr., Chehrizi B (1982) Early decompression and neurological outcome in acute cervical spinal cord injuries. *J Neurosurg*; 56(5):699-705.
7. Amar AP, Levy ML (1999) Pathogenesis and pharmacological strategies for mitigating secondary damage in acute spinal cord injury. *Neurosurgery*; 44(5):1027-1039; discussion 1039-1040.
8. Fehlings MG, Sekhon L (2000) Cellular, ionic and biomolecular mechanisms of the injury process. *Benzel E and Tator CH (eds), Contemporary Management of Spinal Cord Injury: From Impact to Rehabilitation*. Chicago, IL: American Association of Neurological Surgeons.
9. Tator CH (1996) Pathophysiology and pathology of spinal cord injury. *Wilkins RH and Rengachary SS (eds), Neurosurgery*. 2nd ed. New York: McGraw-Hill, 2847-2859.

10. Tator CH, Fehlings MG (1991) Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. *J Neurosurg*; 75(1):15-26.
11. Bunge RP, Puckett WR, Becerra JL, et al (1993) Observations on the pathology of human spinal cord injury. A review and classification of 22 new cases with details from a case of chronic cord compression with extensive focal demyelination. *Adv Neurol*; 59:75-89.
12. Kakulas BA (1984) Pathology of spinal injuries. *Cent Nerv Syst Trauma*; 1(2):117-129.
13. Tator CH (1983) Spine-spinal cord relationships in spinal cord trauma. *Clin Neurosurg*; 30:479-494.
14. Dolan EJ, Tator CH, Endrenyi L (1980) The value of decompression for acute experimental spinal cord compression injury. *J Neurosurg*; 53(6):749-755.
15. Allen A (1911) Surgery for experimental lesions of spinal cord equivalent to crush injury of fracture dislocation of spinal column: a preliminary report. *JAMA*; 57:878-880.
16. Aki T, Toya S (1984) Experimental study on changes of the spinal-evoked potential and circulatory dynamics following spinal cord compression and decompression. *Spine*; 9(8):800-809.
17. Agrawal SK, Fehlings MG (1996) Mechanisms of secondary injury to spinal cord axons in vitro: role of Na⁺, Na⁺-K⁺-ATPase, the Na⁺-H⁺ exchanger, and the Na⁺-Ca²⁺ exchanger. *J Neurosci*; 16(2):545-552.
18. Agrawal SK, Fehlings MG (1997) Role of NMDA and non-NMDA ionotropic glutamate receptors in traumatic spinal cord axonal injury. *J Neurosci*; 17(3):1055-1063.
19. Agrawal SK, Nashmi R, Fehlings MG (2000) Role of L- and N-type calcium channels in the pathophysiology of traumatic spinal cord white matter injury. *Neuroscience*; 99(1):179-188.
20. Schwartz G, Fehlings MG (2002) Secondary injury mechanisms of spinal cord trauma: a novel therapeutic approach for the management of secondary pathophysiology with the sodium channel blocker riluzole. *Prog Brain Res*; 137:177-190.
21. Young W, Koreh I (1986) Potassium and calcium changes in injured spinal cords. *Brain Res*; 365(1):42-53.
22. Osterholm JL, Mathews GJ (1972) Altered norepinephrine metabolism, following experimental spinal cord injury. 2. Protection against traumatic spinal cord hemorrhagic necrosis by norepinephrine synthesis blockade with alpha methyl tyrosine. *J Neurosurg*; 36(4):395-401.
23. Faden AI, Simon RP (1988) A potential role for excitotoxins in the pathophysiology of spinal cord injury. *Ann Neurol*; 23(6):623-626.
24. Anderson DK, Hall ED (1993) Pathophysiology of spinal cord trauma. *Ann Emerg Med*; 22(6):987-992.
25. Anderson DK, Means ED, Waters TR, et al (1980) Spinal cord energy metabolism following compression trauma to the feline spinal cord. *J Neurosurg*; 53(3):375-380.
26. Demopoulos HB, Flamm ES, Pietronigro DD, et al (1980) The free radical pathology and the microcirculation in the major central nervous system disorders. *Acta Physiol Scand Suppl*; 492:91-119.
27. Hall ED, Yonkers PA, Horan KL, et al (1989) Correlation between attenuation of posttraumatic spinal cord ischemia and preservation of tissue vitamin E by the 21-aminosteroid U74006F: evidence for an in vivo antioxidant mechanism. *J Neurotrauma*; 6(3):169-176.
28. Hung TK, Albin MS, Brown TD, et al (1975) Biomechanical responses to open experimental spinal cord injury. *Surg Neurol*; 4(2):271-276.
29. Faden AI, Jacobs TP, Holaday JW (1982) Comparison of early and late naloxone treatment in experimental spinal injury. *Neurology*; 32(6):677-681.
30. Faden AI, Jacobs TP, Holaday JW (1982) Neuropeptides and spinal cord injury. *Adv Biochem Psychopharmacol*; 33:131-138.
31. Wagner FC, Jr., Stewart WB (1981) Effect of trauma dose on spinal cord edema. *J Neurosurg*; 54(6):802-806.
32. Dietrich WD, Chatzipanteli K, Vitarbo E, et al (2004) The role of inflammatory processes in the pathophysiology and treatment of brain and spinal cord trauma. *Acta Neurochir Suppl*; 89:69-74.
33. Casha S, Yu WR, Fehlings MG (2001) Oligodendroglial apoptosis occurs along degenerating axons and is associated with FAS and p75 expression following spinal cord injury in the rat. *Neuroscience*; 103(1):203-218.
34. Crowe MJ, Bresnahan JC, Shuman SL, et al (1997) Apoptosis and delayed degeneration after spinal cord injury in rats and monkeys. *Nat Med*; 3(1):73-76.
35. Emery E, Aldana P, Bunge MB, et al (1998) Apoptosis after traumatic human spinal cord injury. *J Neurosurg*; 89(6):911-920.
36. Lou J, Lenke LG, Ludwig FJ, et al (1998) Apoptosis as a mechanism of neuronal cell death following acute experimental spinal cord injury. *Spinal Cord*; 36(10):683-690.
37. Bracken MB, Holford TR (1993) Effects of timing of methylprednisolone or naloxone administration on recovery of segmental and long-tract neurological function in NASCIS 2. *J Neurosurg*; 79(4):500-507.
38. Bracken MB, Shepard MJ, Collins WF, et al (1990) 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*; 322(20):1405-1411.
39. Bracken MB, Shepard MJ, Holford TR, et al (1997) 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*; 277(20):1597-1604.
40. Bohlman HH, Bahniuk E, Raskulinec G, et al (1979) Mechanical factors affecting recovery from incomplete cervical spinal cord injury: a preliminary report. *Johns Hopkins Med J*; 145(3):115-125.
41. Brodkey JS, Richards DE, Blasingame JP, et al (1972) Reversible spinal cord trauma in cats. Additive effects of direct pressure and ischemia. *J Neurosurg*; 37(5):591-593.
42. Carlson GD, Gorden CD, Oliff HS, et al (2003) Sustained spinal cord compression: part I: time-dependent effect on long-term pathophysiology. *J Bone Joint Surg Am*; 85(1):86-94.
43. Carlson GD, Minato Y, Okada A, et al (1997) Early time-dependent decompression for spinal cord injury: vascular mechanisms of recovery. *J Neurotrauma*; 14(12):951-962.
44. Croft TJ, Brodkey JS, Nulsen FE (1972) Reversible spinal cord trauma: a model for electrical monitoring of spinal cord function. *J Neurosurg*; 36(4):402-406.

45. Delamarter RB, Sherman J, Carr JB (1995) Pathophysiology of spinal cord injury. Recovery after immediate and delayed decompression. *J Bone Joint Surg Am*; 77(7):1042-1049.
46. Dimar JR, 2nd, Glassman SD, Raque GH, et al (1999) The influence of spinal canal narrowing and timing of decompression on neurologic recovery after spinal cord contusion in a rat model. *Spine*; 24(16):1623-1633.
47. Guha A, Tator CH, Endrenyi L, et al (1987) Decompression of the spinal cord improves recovery after acute experimental spinal cord compression injury. *Paraplegia*; 25(4):324-339.
48. Kobrine AI, Evans DE, Rizzoli H (1978) Correlation of spinal cord blood flow and function in experimental compression. *Surg Neurol*; 10(1):54-59.
49. Kobrine AI, Evans DE, Rizzoli HV (1979) Experimental acute balloon compression of the spinal cord. Factors affecting disappearance and return of the spinal evoked response. *J Neurosurg*; 51(6):841-845.
50. Nystrom B, Berglund JE (1988) Spinal cord restitution following compression injuries in rats. *Acta Neurol Scand*; 78(6):467-472.
51. Tarlov IM (1954) Spinal cord compression studies. III. Time limits for recovery after gradual compression in dogs. *AMA Arch Neurol Psychiatry*; 71(5):588-597.
52. Tarlov IM (1957) *Spinal cord compression; mechanism of paralysis and treatment*. Springfield, IL: Charles C. Thomas.
53. Tarlov IM (1955) Spinal cord injuries; early treatment. *Surg Clin North Am*; 35:591-607.
54. Tarlov IM, Klinger H (1954) Spinal cord compression studies. II. Time limits for recovery after acute compression in dogs. *AMA Arch Neurol Psychiatry*; 71(3):271-290.
55. Thienprasit P, Bantli H, Bloedel JR, et al (1975) Effect of delayed local cooling on experimental spinal cord injury. *J Neurosurg*; 42(2):150-154.
56. Yoshida H, Okada Y, Maruiwa H, et al (2003) Synaptic blockade plays a major role in the neural disturbance of experimental spinal cord compression. *J Neurotrauma*; 20(12):1365-1376.
57. Fehlings MG, Sekhon LH, Tator C (2001) The role and timing of decompression in acute spinal cord injury: what do we know? What should we do? *Spine*; 26(24 Suppl):S101-110.
58. Fehlings MG, Tator CH (1999) An evidence-based review of decompressive surgery in acute spinal cord injury: rationale, indications, and timing based on experimental and clinical studies. *J Neurosurg Spine*; 91(1):1-11.
59. Woolf SH (1993) Practice guidelines: a new reality in medicine. III. Impact on patient care. *Arch Intern Med*; 153(23):2646-2655.
60. Zhang Y, Hillered L, Olsson Y, et al (1993) Time course of energy perturbation after compression trauma to the spinal cord: an experimental study in the rat using microdialysis. *Surg Neurol*; 39(4):297-304.
61. Bedbrook GM (1979) Spinal injuries with tetraplegia and paraplegia. *J Bone Joint Surg Br*; 61(3):267-284.
62. Bohlman HH, Freehafer A, Dejak J (1985) The results of treatment of acute injuries of the upper thoracic spine with paralysis. *J Bone Joint Surg Am*; 67(3):360-369.
63. Chen TY, Dickman CA, Eleraky M, et al (1998) The role of decompression for acute incomplete cervical spinal cord injury in cervical spondylosis. *Spine*; 23(22):2398-2403.
64. Donovan WH, Cifu DX, Schotte DE (1992) Neurological and skeletal outcomes in 113 patients with closed injuries to the cervical spinal cord. *Paraplegia*; 30(8):533-542.
65. Frankel HL, Hancock DO, Hyslop G, et al (1969) The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. I. *Paraplegia*; 7(3):179-192.
66. Katoh S, el Masry WS, Jaffray D, et al (1996) Neurologic outcome in conservatively treated patients with incomplete closed traumatic cervical spinal cord injuries. *Spine*; 21(20):2345-2351.
67. Kinoshita H, Nagata Y, Ueda H, et al (1993) Conservative treatment of burst fractures of the thoracolumbar and lumbar spine. *Paraplegia*; 31(1):58-67.
68. McEvoy RD, Bradford DS (1985) The management of burst fractures of the thoracic and lumbar spine. Experience in 53 patients. *Spine*; 10(7):631-637.
69. Petitjean ME, Moussellard H, Pointillart V, et al (1995) Thoracic spinal trauma and associated injuries: should early spinal decompression be considered? *J Trauma*; 39(2):368-372.
70. Aebi M, Mohler J, Zach GA, et al (1986) Indication, surgical technique, and results of 100 surgically-treated fractures and fracture-dislocations of the cervical spine. *Clin Orthop*; (203):244-257.
71. Benzel EC, Larson SJ (1986) Functional recovery after decompressive operation for thoracic and lumbar spine fractures. *Neurosurgery*; 19(5):772-778.
72. Benzel EC, Larson SJ (1987) Functional recovery after decompressive spine operation for cervical spine fractures. *Neurosurgery*; 20(5):742-746.
73. Benzel EC, Larson SJ (1986) Recovery of nerve root function after complete quadriplegia from cervical spine fractures. *Neurosurgery*; 19(5):809-812.
74. Botel U, Glaser E, Niedeggen A (1997) The surgical treatment of acute spinal paralysed patients. *Spinal Cord*; 35(7):420-428.
75. Donovan WH, Kopaniky D, Stolzmann E, et al (1987) The neurological and skeletal outcome in patients with closed cervical spinal cord injury. *J Neurosurg*; 66(5):690-694.
76. Duh MS, Shepard MJ, Wilberger JE, et al (1994) The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. *Neurosurgery*; 35(2):240-248; discussion 248-249.
77. Krengel WF, 3rd, Anderson PA, Henley MB (1993) Early stabilization and decompression for incomplete paraplegia due to a thoracic-level spinal cord injury. *Spine*; 18(14):2080-2087.
78. Larson SJ, Holst RA, Hemmy DC, et al (1976) Lateral extracavitary approach to traumatic lesions of the thoracic and lumbar spine. *J Neurosurg*; 45(6):628-637.
79. Levi L, Wolf A, Rigamonti D, et al (1991) Anterior decompression in cervical spine trauma: does the timing of surgery affect the outcome? *Neurosurgery*; 29(2):216-222.

80. Maynard FM, Reynolds GG, Fountain S, et al (1979) Neurological prognosis after traumatic quadriplegia. Three-year experience of California Regional Spinal Cord Injury Care System. *J Neurosurg*; 50(5):611-616.
81. McAfee PC, Bohlman HH, Yuan HA (1985) Anterior decompression of traumatic thoracolumbar fractures with incomplete neurological deficit using a retroperitoneal approach. *J Bone Joint Surg Am*; 67(1):89-104.
82. Mirza SK, Krengel WF, 3rd, Chapman JR, et al (1999) Early versus delayed surgery for acute cervical spinal cord injury. *Clin Orthop*; (359):104-114.
83. Murphy KP, Opitz JL, Cabanela ME, et al (1990) Cervical fractures and spinal cord injury: outcome of surgical and nonsurgical management. *Mayo Clin Proc*; 65(7):949-959.
84. Ng WP, Fehlings MG, Cuddy B, et al (1999) Surgical treatment of acute spinal cord injury pilot study #2: evaluation of protocol for decompressive surgery within 8 hours after injury. *Neurosurg Focus*; 6:article 3.
85. Papadopoulos SM, Selden NR, Quint DJ, et al (2002) Immediate spinal cord decompression for cervical spinal cord injury: feasibility and outcome. *J Trauma*; 52(2):323-332.
86. Pointillart V, Petitjean ME, Wiart L, et al (2000) Pharmacological therapy of spinal cord injury during the acute phase. *Spinal Cord*; 38(2):71-76.
87. Tator CH, Duncan EG, Edmonds VE, et al (1987) Comparison of surgical and conservative management in 208 patients with acute spinal cord injury. *Can J Neurol Sci*; 14(1):60-69.
88. Vaccaro AR, Daugherty RJ, Sheehan TP, et al (1997) Neurologic outcome of early versus late surgery for cervical spinal cord injury. *Spine*; 22(22):2609-2613.
89. Vale FL, Burns J, Jackson AB, et al (1997) Combined medical and surgical treatment after acute spinal cord injury: results of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg*; 87(2):239-246.
90. Waters RL, Adkins RH, Yakura JS, et al (1996) Effect of surgery on motor recovery following traumatic spinal cord injury. *Spinal Cord*; 34(4):188-192.
91. Waters RL, Meyer PR, Jr., Adkins RH, et al (1999) Emergency, acute, and surgical management of spine trauma. *Arch Phys Med Rehabil*; 80(11):1383-1390.
92. Weinshel SS, Maiman DJ, Baek P, et al (1990) Neurologic recovery in quadriplegia following operative treatment. *J Spinal Disord*; 3(3):244-249.
93. Wiberg J, Hauge HN (1988) Neurological outcome after surgery for thoracic and lumbar spine injuries. *Acta Neurochir (Wien)*; 91(3-4):106-112.
94. Wolf A, Levi L, Mirvis S, et al (1991) Operative management of bilateral facet dislocation. *J Neurosurg*; 75(6):883-890.
95. Brunette DD, Rockswold GL (1987) Neurologic recovery following rapid spinal realignment for complete cervical spinal cord injury. *J Trauma*; 27(4):445-447.
96. Burke DC, Berryman D (1971) The place of closed manipulation in the management of flexion-rotation dislocations of the cervical spine. *J Bone Joint Surg Br*; 53(2):165-182.
97. Dall DM (1972) Injuries of the cervical spine: II. Does anatomical reduction of the bony injuries improve the prognosis for spinal cord recovery? *S Afr Med J*; 46:1083-1090.
98. Hadley MN, Fitzpatrick BC, Sonntag VK, et al (1992) Facet fracture-dislocation injuries of the cervical spine. *Neurosurgery*; 30(5):661-666.
99. Harris P, Karmi MZ, McClement E, et al (1980) The prognosis of patients sustaining severe cervical spine injury (C2-C7 inclusive). *Paraplegia*; 18(5):324-330.
100. Lee AS, MacLean JC, Newton DA (1994) Rapid traction for reduction of cervical spine dislocations. *J Bone Joint Surg Br*; 76(3):352-356.
101. Sonntag VK (1981) Management of bilateral locked facets of the cervical spine. *Neurosurgery*; 8(2):150-152.
102. Star AM, Jones AA, Cotler JM, et al (1990) Immediate closed reduction of cervical spine dislocations using traction. *Spine*; 15(10):1068-1072.
103. Anderson PA, Bohlman HH (1992) Anterior decompression and arthrodesis of the cervical spine: long-term motor improvement. Part II-Improvement in complete traumatic quadriplegia. *J Bone Joint Surg Am*; 74(5):683-692.
104. Bohlman HH, Anderson PA (1992) Anterior decompression and arthrodesis of the cervical spine: long-term motor improvement. Part I-Improvement in incomplete traumatic quadriparesis. *J Bone Joint Surg Am*; 74(5):671-682.
105. Bohlman HH, Freehafer A, Dejak J (1975) Late anterior decompression of spinal cord injuries. *J Bone Joint Surg Am*; 57:1025.
106. Bohlman HH, Kirkpatrick JS, Delamarter RB, et al (1994) Anterior decompression for late pain and paralysis after fractures of the thoracolumbar spine. *Clin Orthop*; (300):24-29.
107. Maiman DJ, Larson SJ, Benzel EC (1984) Neurological improvement associated with late decompression of the thoracolumbar spinal cord. *Neurosurgery*; 14(3):302-307.
108. Transfeldt EE, White D, Bradford DS, et al (1990) Delayed anterior decompression in patients with spinal cord and cauda equina injuries of the thoracolumbar spine. *Spine*; 15(9):953-957.
109. Hung TK, Chang GL, Chang JL, et al (1981) Stress-strain relationship and neurological sequelae of uniaxial elongation of the spinal cord of cats. *Surg Neurol*; 15(6):471-476.
110. Panjabi MM (1987) Experimental spinal cord trauma. A biomechanical viewpoint. *Paraplegia*; 25(3):217-220.
111. Rivlin AS, Tator CH (1978) Effect of duration of acute spinal cord compression in a new acute cord injury model in the rat. *Surg Neurol*; 10(1):38-43.
112. Guttman L (1963) Initial treatment of traumatic paraplegia and tetraplegia. Harris P (ed), *Spinal Injuries Symposium*. Edinburgh: Royal College of Surgeons, 80-92.
113. Guttman L (1976) *Spinal Cord Injuries: Comprehensive Management and Research*. 2nd ed. Oxford: Blackwell.
114. Bedbrook GM, Sakae T (1982) A review of cervical spine injuries with neurological dysfunction. *Paraplegia*; 20(6):321-333.
115. Bedbrook GM, Sedgley GI (1980) The management of spinal injuries-past and present. *Int Rehabil Med*; 2(2):45-61.
116. Collins WF (1983) A review and update of experiment and clinical studies of spinal cord injury. *Paraplegia*; 21(4):204-219.

117. Comarr AE, Kaufman AA (1956) A survey of the neurological results of 858 spinal cord injuries; a comparison of patients treated with and without laminectomy. *J Neurosurg*; 13(1):95-106.
118. Ditunno JF, Jr., Sipski ML, Posuniak EA, et al (1987) Wrist extensor recovery in traumatic quadriplegia. *Arch Phys Med Rehabil*; 68(5 Pt 1):287-290.
119. Tator CH, Duncan EG, Edmonds VE, et al (1995) Neurological recovery, mortality and length of stay after acute spinal cord injury associated with changes in management. *Paraplegia*; 33(5):254-262.
120. Wilmot CB, Hall KM (1986) Evaluation of the acute management of tetraplegia: conservative versus surgical treatment. *Paraplegia*; 24(3):148-153.
121. Wu L, Marino RJ, Herbison GJ, et al (1992) Recovery of zero-grade muscles in the zone of partial preservation in motor complete quadriplegia. *Arch Phys Med Rehabil*; 73(1):40-43.
122. Fehlings MG, Cooper PR, Errico TJ (1994) Posterior plates in the management of cervical instability: long-term results in 44 patients. *J Neurosurg*; 81(3):341-349.
123. Cotler JM, Herbison GJ, Nasuti JF, et al (1993) Closed reduction of traumatic cervical spine dislocation using traction weights up to 140 pounds. *Spine*; 18(3):386-390.
124. Sussman BJ (1976) Letter: Early management of spinal cord trauma. *J Neurosurg*; 44(6):766-767.
125. Harvey C, Rothschild BB, Asmann AJ, et al (1990) New estimates of traumatic SCI prevalence: a survey-based approach. *Paraplegia*; 28(9):537-544.
126. Marshall LF, Knowlton S, Garfin SR, et al (1987) Deterioration following spinal cord injury. A multicenter study. *J Neurosurg*; 66(3):400-404.
127. Brodkey JS, Miller CF, Jr., Harmody RM (1980) The syndrome of acute central cervical spinal cord injury revisited. *Surg Neurol*; 14(4):251-257.
128. Ahn JH, Ragnarsson KT, Gordon WA, et al (1984) Current trends in stabilizing high thoracic and thoracolumbar spinal fractures. *Arch Phys Med Rehabil*; 65(7):366-369.
129. Levi L, Wolf A, Belzberg H (1993) Hemodynamic parameters in patients with acute cervical cord trauma: description, intervention, and prediction of outcome. *Neurosurgery*; 33(6):1007-1016; discussion 1016-1007.
130. Rizzolo SJ, Vaccaro AR, Cotler JM (1994) Cervical spine trauma. *Spine*; 19(20):2288-2298.
131. Rosner MJ, Elias Z, Coley I (1984) New principles of resuscitation for brain and spinal injury. *N C Med J*; 45(11):701-708.
132. Wilberger JE (1991) Diagnosis and management of spinal cord trauma. *J Neurotrauma*; 8 Suppl 1:S21-28; discussion S29-30.
133. La Rosa G, Conti A, Cardali S, et al (2004) Does early decompression improve neurological outcome of spinal cord injured patients? Appraisal of the literature using a meta-analytical approach. *Spinal Cord*; 42(9):503-512.

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