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An evidence-based review of surgical decompression for acute spinal cord injury: rationale, indications, and timing based on experimental and clinical studies

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The authors conducted an evidence-based review of the literature to evaluate critically the rationale and indications for and the timing of decompressive surgery for the treatment of acute, nonpenetrating spinal cord injury (SCI).

The experimental and clinical literature concerning the role of, and the biological rationale for surgical decompression for acute SCI was reviewed. Clinical studies of nonoperative management of SCI were also examined for comparative purposes. Evidence from clinical trials was categorized as Class I (well-conducted randomized prospective trials), Class II (well-designed comparative clinical studies), or Class III (retrospective studies).

Studies in which animal models of SCI were used consistently demonstrated a beneficial effect of early surgical decompression, although it is difficult to apply these data directly to the clinical setting. The clinical studies provided suggestive (Class III and limited Class II) evidence that decompressive procedures improve neurological recovery after SCI. However, no clear consensus can be inferred from the literature as to the optimum timing of decompressive surgery. Many authors have advocated delayed treatment to avoid medical complications, although there is good evidence from recent Class II trials that early decompressive surgery can be performed safely without added morbidity or mortality.

There is biological evidence from experimental studies in animals that early surgical decompression may improve neurological recovery after SCI, although the relevant interventional timing in humans remains unclear. To date, the role of surgical decompression in patients with SCI is only supported by Class III and limited Class II evidence. Accordingly, decompressive surgery for SCI can only be considered a practice option. Furthermore, analysis of the literature does not allow definite conclusions to be drawn regarding appropriate timing of intervention. Hence, there is a need to conduct well-designed experimental and clinical studies of the timing and neurological results of surgical decompression for the treatment of acute SCI.

Key Words * surgical decompression * acute spinal cord injury * review

With an average incidence of 11,000 cases per year in North America, spinal cord injury (SCI) is an important cause of morbidity and mortality,[63,91] particularly among teenagers and young adults. The main

causes of acute SCI are traffic accidents, sports and recreational activities, accidents at work, falls in the home, and violence,[63,91] Because there is a lack of effective treatments for restoring neurological function below the level of the injury means the vast majority of SCI victims face many years of lost independence and continued medical expenses. Indeed, the financial cost of care for acute SCI is enormous.[97] For example, in 1975, Kraus, et al.,[63] estimated an "annual cost to the United States for support and treatment of all persons with a spinal cord injury of two billion dollars," and in 1990, Stripling[86] estimated that this figure had risen to four billion dollars annually.

The results of recent studies of pharmacotherapy, such as the use of methylprednisolone as recommended on the basis of the National Acute Spinal Cord Injury Studies (NASCIS-2 and NASCIS-3)[21-23] have shown improved recovery in patients with SCI. Unfortunately, the improved neurological recovery observed to date has been modest, with only slight improvement in the functional capability in these patients.

Recent advances in the safety and efficacy of spinal cord surgical decompressive procedures offer significant potential for repairing some of the neurological damage caused by injuries to the spine.[4,14,33,96,110] Despite the widespread use of surgery in patients with acute SCI in North America, the role of this intervention in improving neurological recovery remains controversial because of the lack of well-designed and -executed randomized controlled trials. In the present paper, we review the experimental and clinical evidence with regard to the value of surgical decompression in treating patients with acute nonpenetrating SCI and compare these data with the results of conservative, nonoperative management of SCI. This evidence-based literature analysis was conducted as part of the Surgical Trial in the Acute Spinal Cord Injury Study project, an undertaking supported by the Joint Section of Neurotrauma and Critical Care and the Joint Section of Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. The goal of the Surgical Trial in Acute Spinal Cord Injury Study is ultimately to plan and conduct a randomized controlled trial to investigate the appropriate timing and clinical indications for decompressive surgery in the injured spinal cord.

CLINICAL MATERIAL AND METHODS

We conducted a computer-assisted Medline search of the experimental and clinical literature from 1966 to 1998 that concerned the role of decompressive surgery in the treatment of SCI. Only articles with English-language abstracts were selected for review. This computerized literature review was supplemented by a detailed examination of the reference lists from the selected articles. Fifty-nine papers (16 experimental studies in animal models and 43 clinical studies) were selected for detailed analysis (Tables 1-5). Evidence from clinical trials was classified as Class I (well-designed and -conducted randomized controlled trials), Class II (prospective cohort studies or controlled studies with well-defined comparison groups) and Class III (case series, retrospective reviews, and expert opinion).

BIOLOGICAL RATIONALE FOR EARLY TREATMENT OF PATIENTS WITH ACUTE SCI

Concept of Secondary Injury

There is increasing evidence that the pathophysiology of acute SCI involves both primary and secondary injury mechanisms,[6,56,92,93,99,106,114,115] In the majority of traumatic SCIs, the mechanism of injury involves rapid cord compression due to bone displacement from a fracture-dislocation or burst fracture.[27,58,95] Acute spinal cord distraction, acceleration-deceleration with shearing, and transection from penetrating injuries are other potential mechanisms of injury.[39,63] There is considerable evidence that the primary mechanical injury initiates a cascade of secondary injury mechanisms such as: 1) vascular changes including reduction in blood flow, loss of autoregulation, neurogenic shock, hemorrhage, loss of microcirculation, vasospasm and thrombosis (see Stripling[86] and Tator[94] for reviews); 2) electrolyte

shifts including increased intracellular calcium, increased extracellular potassium, and increased sodium permeability;[2,117] 3) neurotransmitter accumulation such as serotonin or catecholamines[77] and extracellular glutamate,[3] the latter producing excitotoxicity;[46] 4) arachidonic acid release, free radical production especially oxygen-free radicals,[37] eicosanoid production, especially prostaglandins, and lipid peroxidation;[54,56] 5) endogenous opioids;[44,45] 6) edema formation;[105] 7) inflammation;[142] and 8) loss of energy metabolism, especially decreased adenosine triphosphate production.[8] These theories of secondary injury have been the subject of several recent reviews.[7,30,43,99,116]

The improved understanding of the pathophysiology of acute SCI has led to use of novel pharmacological strategies to attenuate the effects of the secondary injury. In the NASCIS-2 study a modest beneficial effect of high-dose methylprednisolone was demonstrated if given within 8 hours postinjury in patients with complete and incomplete SCIs,[22] which emphasizes the importance of the timing of treatment. Furthermore, the NASCIS-3 study provided some evidence that treatment within 3 hours may have been superior to treatment begun between 3 and 8 hours postinjury.[23] The findings of these studies provide validity to the concept that secondary SCI mechanisms can be attenuated in the clinical setting. It is unclear, however, whether the "time window" for methylprednisolone is directly applicable to surgical decompression of the spinal cord.

There is experimental evidence that persistent compression of the spinal cord is a potentially reversible form of secondary injury. The severity of SCI in experimental models appears to vary depending on several factors, including force of compression, duration of compression, displacement, impulse and kinetic energy.[39,50,57,75,78,80,89] The evolution of these pathophysiological processes leading to progressive pathological changes during the first few hours postinjury is important with respect to the surgical and nonsurgical treatment of SCI. As we will describe in the following detail, the severity of the pathological changes and the degree of recovery are directly related to the duration of acute compression as demonstrated by experimental studies in which longer compression times produced less demonstrable clinical recovery.[21,36,39,50,76,87]

Studies of Surgical Decompression in Experimental Models

Biomechanics of spinal cord injury and classification of models. Several experimental studies of decompressive procedures performed after SCI have been conducted using these models,[16,25,29,34,36,39,50,61,62,76,87,88,90] and most have demonstrated that neurological recovery is enhanced by early decompressive surgery (Table 1).

TABLE 1
EFFECT OF DECOMPRESSION IN ANIMAL MODELS OF ACUTE SCI

Authors & Year	Animal Studied	Model of Injury	Duration of Compression	Results
Tarlov, 1954, Tarlov, 1957, Tarlov and Klinger, 1954	canine	extradural balloon	mins to days	decomp improved neuro-recovery
Brodkey, et al., 1972	cat	static weight	mins	early removal of weight restores axonal conduction
Croft, et al., 1972	cat	static weight	5–20 mins	early decomp at milder severities of injury improved recovery
Thienprasit, et al., 1975	cat	extradural balloon	6 hrs	laminectomy ineffective in promoting neurorecovery
Kobrine, et al., 1978, 1979	monkey	extradural balloon	1–15 mins	early decomp promotes neuro-recovery
Bohlman, et al., 1979	canine	ant piston	3–8 wks	decomp promotes neurorecovery
Dolan, Tator, et al., 1980	rat	clip	secs–15 mins	decomp improves neurorecovery
Aki & Toya, 1984	canine	static loads 6–60g	30 & 60 mins	vascular changes more severe with greater degrees of comp
Guha, et al., 1987	rat	clip	1–4 hrs	decomp by 2 hrs improves recovery with milder injuries
Nystrom & Berglund 1988	rat	static weight	1–19 mins	early decomp promotes recovery with more severe SCI
Zhang, et al., 1993	rat	static load 9, 35, & 50 g	5 mins	increased levels of lactate, inosine, hypoxanthine with SCI; reversal of metabolic changes with decomp
Delamarter, et al., 1995	canine	circumferential cable	1 hr–1 wk	decomp at 1 hr improves neurorecovery
Carlson, et al., 1997	canine	piston	5 mins, 1 hr, & 3 hrs	recovery of evoked potentials in 5 mins and 1 hr group
* ant = anterior; comp = compression; decomp = decompression				

However, to understand the rationale of spinal cord decompressive surgery in the context of acute SCI and to interpret the results of experimental studies correctly, a brief review of the biomechanics of SCI and the relevant experimental models is appropriate. The spinal cord is viscoelastic and behaves as a linear elastic material under small strains (strain = force/area).[57] Indeed, Somerson and Stokes[83] have shown in compression injuries causing less than 1.0 mm deformation of the rodent spinal cord that the cord behaves like a spring, with a linear relationship between the applied force and the resultant displacement. In contrast, at displacements greater than 1.0 mm, the spinal cord displays nonlinear characteristics. Consequently, the relationship between the degree of spinal cord displacement from a space-occupying lesion and the loss of neurological function may also be nonlinear. Nevertheless, clinical outcome and morphometric characteristics of the spinal cord lesion do appear to vary depending on a number of factors, including force, duration of compression, displacement, impulse, and kinetic energy.[39,50,57,75,78,80]

In one review we found that several experimental models have been developed that mimic human SCI.[47] Compression models can be classified as either kinetic or static according to the biomechanics of the applied forces. Kinetic compression models involve rapid compression of the cord in less than 1 second. Indeed, most kinetic models compress the cord in less than 100 milliseconds. With kinetic compression, the applied load compresses the cord with increasing velocity (acceleration > 0) to the point of maximal cord compression. In contrast, static compression models use forces that slowly compress the cord at approximately constant velocity.

Kinetic Models of Spinal Cord Injury. Of the many types of kinetic compression models, which most closely simulate the majority of traumatic human SCIs, the studies that relate the duration of compression to

the recovery of function have been limited to the extradural balloon compression technique in several species and the clip compression model in rodents. The advantage of the latter technique is that the force of clip closure can be calibrated precisely, and the duration of the compression can be altered over a wide range of times.

Tarlov and Klinger[87,88,90] have used the extradural balloon compression technique to injure the lumbar spinal cord and cauda equina in dogs. With acute SCI, the degree of recovery varied with the size of the balloon ("medium" or "large") and with the duration of compression. For example, with a large-sized balloon, full recovery was seen with decompressive surgery after 1 minute, partial recovery was seen after 5 minutes, and no recovery was demonstrated if compression was maintained longer. With medium-sized balloons, recovery was seen if decompressive surgery was performed within 1 hour of injury. The shortcomings of this study were the variable placement of the balloon and the lack of precise quantitation of the injury force or balloon pressure.

Kobrine and colleagues[61,62] conducted a series of experiments in monkeys in which spinal cord compression at the T-6 level was produced by an inflatable extradural balloon. In the slow balloon inflation group, there was return of function based on return of the spinal cord evoked potential even after periods of compression that lasted several minutes.[61] However, after rapid balloon inflation and acute spinal cord compression, only the animals that underwent compression for 1 minute had return of the response by 1 hour. In contrast, animals that underwent acute compression for 3, 5, 7, or 15 minutes showed no recovery by 1 hour.[62] No recordings were obtained after 1 hour, and thus it is not known if some recovery would have occurred after that timepoint. The authors concluded that recovery of spinal cord function following spinal cord compression depended on the "rapidity and length of time of compression." [62] Thus, with an extradural balloon compression method in dogs and cats, the duration of compression was found to be a significant determinant of neurological recovery but only when decompression was performed after a period of minutes.

In the laboratory of one of the authors (C.H.T.), Dolan, et al.,[39] used an extradural clip compression model of the T-1 cord in rats to provide a more quantitative assessment of injury force. The springs of modified aneurysm clips were machined to provide closing forces of 16, 71, or 178 g. Durations of compression times between 3 and 900 seconds were examined. We found that clinical neurological recovery, as assessed by the inclined plane technique,[81] varied exponentially according to the force of injury and linearly with the duration of compression. The results of this study demonstrated that the major determinant of recovery was the initial force of injury. However, the duration of compression (and thus time to decompressive intervention) was a significant determinant of clinical recovery, even in cases of severe injury forces. The main shortcoming of this study was that the maximum duration of compression examined (900 seconds) was too short to be of clinical relevance. Subsequently, our laboratory extended the duration of compression to 4 hours in the study reported by Guha, et al.,[50] with injury forces of 2.3, 16.9, or 53.0 g and durations of compression of 15, 60, 120, or 240 minutes. Again, the major determinant of recovery was the force of injury, although the time to decompression also affected clinical recovery. The beneficial effects of early decompressive surgery were most apparent in the groups receiving smaller-force injuries (2.3 and 16.9 g). For example, decompression performed at 2 hours produced significantly more recovery than when performed at 4 hours.

In conclusion, the results of these studies confirm the efficacy of surgical decompressive procedures in attenuating the deleterious effects of acute SCI. However, it is difficult to extrapolate the results of these studies to clinical practice. Intuitively, it appears likely that the time window in humans is much longer than in the species studied experimentally.

Static Models of Spinal Cord Injury. Static models, which involve a gradual compression of the spinal cord, are useful to model the effect of spinal cord displacement, as well as strain and duration of compression. However, because the load is applied slowly to the cord, these models do not accurately simulate the biomechanical aspects of the majority of human SCI. Nystrom and Berglund[76] have used a static model of SCI in rats to examine the role of injury force and duration of compression on clinical neurological recovery. Weights of 20, 35, or 50 g were applied to the midthoracic cord of rats for periods of 1, 5, or 19 minutes, and clinical neurological recovery was assessed by using the inclined plane technique.[81] In the 20-g injury group, the duration of compression was not a significant determinant of neurological recovery. However, when the 35- and 50-g weights were applied, neurological recovery varied directly according to the duration of the compression period. Unfortunately, the durations of compression studied were too brief to be clinically meaningful, although the results suggest that the timing of decompressive intervention may play a significant role in outcome after acute SCI. Tarlov[88] also examined the role of decompressive surgery in a model of gradual extradural balloon inflation. Functional recovery varied both as a function of injury "force" (determined by the size of the balloon) and duration of compression. For example, if the extradural balloon was inflated over 75 minutes to the point of paralysis, excellent neurological recovery was attained if deflation was performed within 9 hours. In contrast, if the period of balloon inflation was extended to 48 hours, full neurological recovery was seen if decompression was performed within 7 days.

CLINICAL STUDIES

Role of Conservative Management in Acute Spinal Cord Injury

To evaluate the possible role of surgery in the management of SCI, it is important, for comparative purposes, to examine the results of conservative, nonoperative treatment (Table 2).

TABLE 2 NEUROLOGICAL RECOVERY IN PATIENTS WITH SCI UNDERGOING NONOPERATIVE MANAGEMENT*				
Authors & Year	No. of Patients (level treated)	Study Design	Class of Evi- dence	Results
Guttman, 1963	396 (all levels) nonop	retro case series	III	30 died; 3 neurologically worse; 120 improved > 1 grade
Frankel, et al., 1969	612 (all levels) nonop	retro case series	III	only 4/612 developed delayed in- stability; 29% of Frankel A pts improved > 1 grade
Maynard, et al., 1979	123 (cervical), 57 nonop	retro case series	III	nonop showed similar results to surgical treatment
Harris, et al., 1980	145 (cervical), 48 op, 97 nonop	retro case series	III	neither surgery nor realignment of spinal column improved neuro outcome
Bedbrook, et al., 1979	253 (all levels) nonop in all but 171	retro case series	III	13% of Frankel A pts recovered spontaneously
Donovan, et al., 1987	61 (cervical) 43 nonop	retro case series	III	31/43 conservatively treated pts improved ≥ 1 Frankel
Duh, et al., 1994	487 (all levels) 189 nonop	pro series w/controls; nonran	II	mean change score of 13.2 in nonop pts < group undergoing surgery w/in 25 hrs (mean change score 17.8)
Kato, et al., 1996	63 (incomplete cervical SCI)	retro case series	III	neuro deterioration in 6/63; neuro improvement in 44 pts
* neuro = neurological; nonran = nonrandomized; pro = prospective; retro = retrospective.				

This approach has been advocated by those who adhere to the tenets of Sir Ludwig Guttmann, founder of the

Stoke-Mandeville Hospital in England. Guttman used postural techniques combined with bedrest to achieve reduction and spontaneous fusion of the spine. Operative approaches were rarely performed because of a higher incidence of neurological complications and impaired recovery after laminectomy procedures.[11,12,31,51,52] For example, Frankel, et al.,[48] reported on a cohort of 612 patients who suffered "closed spinal injuries" and who were treated by these techniques. Only four of these patients developed delayed instability and required operative fusion. However, detailed descriptions of the fractures and the criteria for determining spinal cord instability or failure of nonoperative management were not provided. Importantly, 29% of Frankel A patients (with complete motor and sensory paralysis below the level of the injury) improved at least one grade during the course of their hospital stay.

In patients undergoing conservative therapy, the spontaneous improvement in neurological status has been replicated in several subsequent studies.[8,31,38,98,111,113] Accordingly, the comparative beneficial results of surgical treatment need to be weighed against the limited spontaneous recovery that occurs after SCI. Indeed, some authors have reported that neither spinal cord surgery or anatomical realignment of the spinal column improved neurological outcome in patients with acute SCI with the possible exception of those patients with bilateral locked facets.[35,55,70] To date, studies of nonoperative management are limited to noncontrolled, retrospective analyses of clinical databases and, accordingly, provide Class III evidence. Furthermore, it is now well recognized that laminectomy as the sole surgical technique is contraindicated in most cases of acute SCI because it usually fails to produce adequate decompression of the spinal cord and often causes spinal instability that itself can lead to neurological deterioration.

Although meticulous, conservative care remains the cornerstone of SCI management, modern surgical techniques have evolved considerably since the era of Guttman. Furthermore, there are major limitations to using an exclusive policy of nonoperative management to treat patients with SCI. For example, there is evidence that neurological deterioration can occur in up to 10% of patients with incomplete cervical SCI who undergo an exclusively conservative management protocol.[59]

Role of Decompressive Surgery in the Management of Acute SCI

The evidence regarding the role of decompressive procedures in the management of SCI is summarized in Tables 3, 4, and 5.

TABLE 3 EFFECT OF DECOMPRESSION IN PATIENTS WITH ACUTE SCI*					
Authors & Year	No. of Patients (level treated)	Timing of Surgery	Study Design	Class of Evidence	Results
Larson, et al., 1976	44 (thoracic & lumbar): < 1 wk, 8 pts; > 1 wk, 36 pts	early < 1 wk; late > 1 wk	retro case series	III	no difference in neurological recovery between "early" & late groups; preferable to wait
Maynard, et al., 1979	123 (cervical); 51 early; 10 late; 62 nonop	early < 4wks; late > 4wks	retro case series	III	surgery w/ 4 wks not associated w/ improved neurorecovery
Wagner & Chehrizi, 1982	44 (cervical): 24 op; 20 nonop	early < 8 hrs; late 9–48 hrs	retro case series	III	decom w/ 8 hrs not better than 9–48 hrs; surgery does not improve recovery
Bohlman, et al., 1985	218 (T1–10)	most delayed (> 1 week post-SCI)	retro case series	III	pt w/ complete injury recovered w/ surgery; ant decom best for neurorecovery in incomplete injuries
Aebi, et al., 1986	100 (cervical): all surgical	75% reduced w/ 6 hrs	retro case series	III	early reduc & surgery improved 31/100 cases
Benzel & Larson, 1986	105 (thoracic & lumbar)	all delayed surgery (19–48 days)	retro case series	III	ant decom & stabil resulted in superior neurorecovery to a nonop approach or only stabil
Benzel & Larson	35 (cervical, all	1–4 wks	retro case	III	ant decom improves nerve root

Benzel & Larson, 1986	53 (cervical, all complete) 25 ant; 10 post	1-4 wks	retro case series	III	ant decom improves nerve root recovery
Benzel & Larson, 1987	99 (cervical)	17-52 days	retro case series	III	surgery improved neurofunction in incomplete SCI; no relationship between time to decom & neuro-recovery
Donovan, et al., 1987	61 (cervical): 18 op; 43 nonop	w/ 3 wks	retro case series	III	no difference in outcome between op & nonop cases
Tator, et al., 1987	208 (all levels): 75 decom or reduced; 41 fusion; 92 nonop	w/ 4 wks	pro, nonran	II	surgery did not improve neurorecovery or length of stay
Wiberg & Hauge 1988	30 (thoracic & lumbar)	8 pts w/ 24 hrs; 2, 1-7 days; 20, > 7 days	retro case series	III	decom improved neurorecovery; 2 pts w/ complete SCI op w/ 10 hrs showed recovery
Murphy, et al., 1990	102 (cervical): 58 op; 44 nonop	early < 2 wks	retro case series	III	early surgery reduced LOH & promoted earlier rehab
Weinshel, et al., 1990	90 (cervical ASIA A & B)	6 hrs-60 days (av 13 days)	retro case series	III	decom improved neurorecovery in motor complete cervical SCI
Wolf, et al., 1991	52 (cervical; bilat locked facets)	40 pts reduced < 4 hrs by traction; 23 op < 24 hrs	retro case series	III	early reduc of bilat locked facets resulted in neurorecovery in complete & incomplete SCI
Levi, et al., 1991	103 (cervical): early 45; late 58	early < 24hrs; late > 24hrs	retro case series	III	early surgery may benefit overall management but not associated w/ improved neurorecovery
Krengel, et al., 1993	14 (T2-11 incomplete)	12 pts < 24hrs 1 pt 36 hrs 1 pt 5 days	retro case series; historical controls	III	early surgical reduc & decom is safe & improves neuro function; ? superior to historical controls
Duh, et al., 1994	487 (all levels): 303 op; early 38 late 105	early < 25hrs; late > 200hrs	pro, nonran	II	surgical decom < 25 hrs or > 200hrs may be associated w/ better recovery
Petitjean, et al., 1995	49 (thoracic): 65% associated w/ head injury	early av 12hrs; late av 9 days	retro case series	III	spinal decom has no benefit in complete thoracic paraplegia
Waters, et al., 1996	269 (all levels): 142 nonop; 127 op	av > 14 days	pro, nonran	II	no benefit to surgery; however, all pts underwent delayed treatment
Botel, et al., 1997	255 (all levels): 178 op; 51.4% early; 10.5% late	early < 24 hrs; late > 2 wks	retro case series	III	no neurorecovery in complete SCI; no clear relationship of neurorecovery to timing
Vaccaro, et al., 1997	62 (cervical): early 34; late 28	early < 72hrs; late > 5 days	pro ran; 20 pts lost to FU	II	no difference in neurological outcome
Vale, et al., 1997	77 (all levels): 58 op	11 pts < 24hrs; 13 24-72hrs; 34 > 72 hrs	pro, nonran	II	no clear relationship between neuro outcome & timing of surgery

* ant = anterior; ASIA = American Spinal Injury Association; av = average; FU = follow up; LOH = length of hospitalization; pos = posterior; reduc = reduction; rehab = rehabilitation; stabil = stabilization.

Most studies, with a few notable exceptions, are retrospective case series with historical controls (Class III evidence). From these studies, there is no clear consensus as to the appropriate timing of surgical intervention, nor is there compelling evidence that surgical decompression influences patient neurological outcome after SCI. For example, Aebi et al.,[1] Wiberg and Hauge,[109] Hadley, et al.,[53] and Wolf, et al.,[112] have advocated early spinal cord reduction procedures (4-10 hours) and operative fixation of spinal fractures associated with SCI. Suggestive evidence is presented in these studies that early decompressive surgery in selected patients may enhance neurological recovery. However, these studies were uncontrolled, and the beneficial effects need to be considered in the context of spontaneous recovery, which can occur in nonoperatively managed patients with SCI.[48,59]

The benefits of early surgical reduction procedures to treat spinal dislocations by either open or closed techniques are difficult to evaluate in the absence of randomization (Table 4).[1,26,32,49,107] Accounts of

impressive neurological recovery in some patients who underwent early cervical decompression by traction must be considered anecdotal[26] (Table 4).

TABLE 4 EFFECT OF CLOSED REDUCTION ON NEUROLOGICAL RECOVERY AFTER ACUTE CERVICAL SCI*					
Authors & Year	No. of Patients	Intervention	Study Design	Class of Evidence	Results
Burke & Berryman, 1971	76	CR; 50% admitted w/ 8 hrs	retro case series	III	early CR improved neuro outcome in incomplete SCI
Dall, 1972	75	timing of reduc NS	retro case series	III	anatomical reduc of fracture-dislocations did not improve neurorecovery
Harris, et al., 1980	145	protocol for traction NS	retro case series	III	reduc of cervical dislocations did not improve neurooutcome
Sonntag, 1981	15	CR in 11; open in 4	retro case series	III	transient root palsy in 1; root recovery in 2
Aebi, et al., 1986	100	75% reduced w/ 6 hrs	retro case series	III	early reduc resulted in neuroimprovement in 31/100
Brunette & Rockswold, 1987	1	C3-4 reduced by 90 mins	case report	III	improvement from ASIA A to ASIA E by rapid reduc of C3-4 dislocation
Star, et al., 1990	53	39/53 required > 50 lbs	retro case series	III	68% of pts improved neurologically; no deterioration
Hadley, et al., 1992	54	10 reduced w/ 8 hrs	retro case series	III	reduc w/ 8 hrs in 10 cases resulted in neuro improvement
Lee, et al., 1994	210	rapid traction up to 150 lbs	retro case series	III	rapid realignment improves neuro outcome
* CR = closed reduction; NS = not specified.					

Moreover, the authors of several studies have found no neurological benefit associated with reduction procedures,[35,55,107] with the possible exception of patients with bilateral facet dislocation.[10] Burke and Berryman[28] have described 76 patients in whom unilateral or bilateral dislocations of the cervical spine were treated by performing closed reduction after induction of general anesthesia, often with manipulation; 50% of the patients were admitted to their center within 8 hours of injury. These authors concluded that early surgical reduction procedures improved the neurological recovery of patients with incomplete SCI.

Aebi, et al.,[1] have examined the records retrospectively of 100 patients with cervical spine injuries and attempted to relate neurological recovery to the timing of the reduction procedure by closed manual traction or open surgical reduction. A manual or surgical reduction procedure was performed within the first 6 hours postinjury 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 in whom reduction was performed manually or surgically within the first 6 hours (Table 94). Cotler's group[32,73] studied the safety and effectiveness of early reduction procedures, and performed a prospective study of early reduction by traction in 24 patients. They found no neurological deterioration in any of the patients, most of whom underwent successful reduction procedures within 24 hours of injury, although the exact interval in hours between injury and intervention was not provided. All of the patients were awake during the procedure, although a muscle relaxant was administered to some patients.

In contrast to the aforementioned studies in which decompressive procedures were performed in the early stage after injury, Larson and coworkers[65] have advocated the surgery be performed a week or more after SCI to allow medical and neurological stabilization of the injured patient (Table 3). This remains the practice in many institutions, particularly in light of early reports, the results of which suggest an increased rate of medical complications when surgery is performed to early after injury (< 5 days after SCI).[71] Interestingly,

a number of authors (summarized in Table 5) have documented recovery of neurological function after performing delayed decompressive surgery in the spinal cord (months to years) postinjury.[9,15,17,19,24,65,101]

TABLE 5 EFFECT OF LATE DECOMPRESSIVE SURGERY ON NEUROLOGICAL RECOVERY AFTER SCI*					
Authors & Year	No. of Patients (level treated)	Timing of Surgery	Study Design	Class of Evidence	Results
Larson, et al., 1976	25 (all levels)	all > 4 wks; 15 > 6 mos	retro case series	III	improved neurorecovery with surgery in complete and incomplete SCI
Bohlman & Freehafer, 1979	36 (32 cervical, 4 thoracolumbar)	av 22 mos	retro case series	III	8/27 pts w/incomplete SCI had motor recovery, 29/36 had nerve root recovery
Brodkey, et al., 1980	83	wks-4 yrs	retro case series	III	improved neurorecovery in incomplete SCI
Maiman, et al., 1984	20 (thoracolumbar)	1 mo-5 yrs	retro case series	III	17/20 pts made neurorecovery
Transfeldt, et al., 1990	49 (thoracolumbar)	> 3 mos	retro case series	III	46.5% of incomplete SCI improved neurologically
Anderson & Bohlman, 1992	51 (cervical: motor complete)	1 mo-8 yrs (av 15 mos)	retro case series	III	improved segmental (1 or 2 levels) recovery in 25/51; improved distal cord function in 1 pt
Bohlman, Anderson, 1992	58 (cervical; motor incomplete)	1 mo-9 yrs (av 13 mos)	retro case series	III	improved neurorecovery in 46 incomplete SCI pts
Bohlman, et al., 1994	25 (thoracolumbar)	av 4.5 yrs	retro case series	III	21/25 patients improved neurologically; proportion incomplete unclear
*Late decompressive surgery was defined as > 4 weeks postinjury.					

Although these studies were retrospective in design, the improvement in neurological function after delayed decompressive surgery in patients with cervical or thoracolumbar SCI whose recovery has plateaued is noteworthy; it suggests that compression of the cord is an important contributing cause of neurological dysfunction.

In our literature review we found five prospective, controlled studies of surgical decompression in acute spinal cord injury (Table 3).[1,12,13,18,20,40,41,64,65,68,72,74,79,96,102-104,107-109,112] In a prospective, nonrandomized case control study of 208 patients with acute spinal cord or cauda equina injury, Tator and colleagues[96] compared the results of surgery (56% of patients) with nonoperative management (44% of patients). Operative management was associated with a lower overall mortality rate (6.1%) than nonoperative treatment (15.2%) despite a higher rate of thromboembolic complications in the surgical group. Overall, there was no difference between surgically and nonsurgically treated patients in length of stay or neurological recovery.

In an analysis of the NASCIS-2 database (Class II evidence), Duh, et al.,[41] reported that patients who underwent acute surgery (< 25 hours after injury) achieved statistically insignificant improvement (mean neurological change score of 17.8) when compared with a control cohort of patients who were treated nonoperatively (mean change score of 13.2). Interestingly, results of surgery were similar in the early (< 25 hours postinjury) and delayed (> 200 hours postinjury) groups. In contrast, in a series of prospective studies Vale, et al.,[103] Vaccaro, et al.,[102] and Waters, et al.,[107] could not document a beneficial effect of decompressive surgery. 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 that performed within 72 hours after SCI. In view of the large number of patients lost to follow-up review, we have considered the study by Vaccaro, et al., to provide Class II evidence.

THE EFFECT OF SURGERY ON THE COMPLICATION RATE AND THE LENGTH OF STAY AFTER SCI

There has been controversy about whether surgery, especially early surgery, increases the rate of complications in patients with SCI. Many SCI patients with high cervical complete injuries or significant associated injuries to the limbs or viscera suffer critical illness due to either hemodynamic or respiratory difficulties. Early investigators such as Guttman[51,52] and Bedbrook and Sekae[11] and, more recently, Wilmot and Hall[111] and Marshall, et al.,[71] have warned against surgery, (especially early surgery) in these critically ill patients. However, modern methods of respiratory and hemodynamic resuscitation[60,67,82,103] have allowed these patients to undergo surgery with minimal differences in complication rates between operative and nonoperative cases.[96,111] Indeed, Wilberger's recent study[110] showed that those patients in whom operations were performed within the first 24 hours had a lower rate of complications than those who underwent surgery at later times. In a previously mentioned study from our center,[96] the only difference in morbidity rates between the surgical and nonsurgical cases was a slight increase in the incidence of deep venous thrombosis in the surgically treated group. The length of stay in the two groups did not differ.[96] In the randomized trial in which Vaccaro, et al.,[102] examined the timing of the surgery, there was no significant difference in length of acute postoperative intensive care stay or length of inpatient rehabilitation between the early and late groups. Thus, with respect to complications, there is no compelling evidence that early surgery increases the rate of complications.

CONCLUSIONS

There is strong experimental evidence from animal models to indicate that decompressive surgery in the spinal cord improves recovery after SCI. However, it is difficult to determine a time window for the effective application of surgical decompressive intervention in the clinical setting from these animal models. Results of these studies on secondary injury mechanisms including ischemia, free radical-mediated lipid peroxidation, and calcium-mediated cytotoxicity suggest that early intervention within hours after SCI is critical to attain a neuroprotective effect. Whether the same time window applies to surgical treatment is as yet unclear. To date, the clinical studies that have examined the role of decompressive surgery in the treatment of SCI are limited to Class II and Class III evidence, except for one study in which the timing of decompression was examined. Surgery remains a valid practice option, although there is no conclusive data showing a benefit over conservative management approaches. There is limited Class II evidence suggesting that either early (< 25 hours) or delayed (> 200 hours) surgical intervention is safe and equally effective. Clearly, to define better the role of surgery in the management of acute SCI, randomized controlled prospective trials are required.

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