

Acute, severe traumatic spinal cord injury: monitoring from the injury site and expansion duraplasty

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CLINICS CARE POINTS

- Intraspinial pressure monitoring at the injury site may be used to guide management after spinal cord injury, analogous to intracranial pressure monitoring for brain injury.
- Aiming to optimize spinal cord perfusion pressure makes more sense than targeting blood pressure, because spinal cord perfusion pressure accounts for intraspinal pressure.
- The monitoring studies showed that the injured cord swells and is compressed against dura, which causes compartmentalization at the injury site.
- Based on the observation that the swollen, injured cord is compressed against dura, a randomized trial termed DISCUS is being set up to investigate expansion duraplasty as a novel treatment.

ABSTRACT

The management of acute SCI has evolved from being considered a hopeless condition by the ancient Egyptians, through attempts to restore normal spinal alignment by the ancient Greeks, followed by bony decompression in the 19th century, and 20th century realisation that draining urine, avoiding pressure ulcers and treating chest infections dramatically improves outcome. Now in the 21st century we discuss two evolving management options that hold promise to further improve outcome; pressure monitoring from the injured cord and expansion duraplasty.

Probes surgically implanted at the injury site can transduce intraspinal pressure, spinal cord perfusion pressure and cord metabolism. Results suggest that the optimum spinal cord perfusion pressure varies between patients, thus supporting individualized management. Furthermore, intraspinal pressure is not adequately reduced by bony decompression alone because the swollen, injured cord is compressed against the dura. Expansion duraplasty may be necessary to effectively decompress the injured cord. A randomized controlled trial called DISCUS, is underway to investigate expansion duraplasty as a novel treatment for acute, severe traumatic cervical spinal cord injury.

INTRODUCTION

This review will focus on two promising management options for acute, severe traumatic spinal cord injury (SCI), monitoring from the injury site and expansion duraplasty. Injury site monitoring for SCI is analogous to injury site monitoring for traumatic brain injury (TBI). We discuss how our understanding of SCI has evolved and introduce novel concepts identified by monitoring from the injured cord. We apply these concepts to answer questions regarding management with emphasis on duraplasty as a novel treatment.

HISTORY

In the Edwin Smith papyrus (c.3000 B.C.), the ancient Egyptians advocated conservative management for SCI patients who had limb paralysis because their outcome was universally fatal [1]. Hippocrates (c.460 – 370 B.C.) attempted to reduce the fractured spine using traction devices [2], whereas Paul of Aegena (c.626 – 690 A.D.) performed laminectomy using wine to wash the wound and compression dressings to control bleeding [3]. The 19th century London surgeon Astley Cooper carried out decompressions for SCI [4]. He often operated with no warning; many of his patients were terrified and refused to consent, but Cooper believed that it was the surgeon’s duty to “get the job done”. The dismal results of such operations led the 19th century Scottish surgeon/neurologist Charles Bell to propose that all of the cord damage happens at the time of injury rendering ongoing cord compression irrelevant; he concluded that surgical decompression was both dangerous and useless [4]. Bell realized that death after SCI was caused by retention of urine resulting in renal damage and sepsis. This was a major milestone in the treatment of SCI, and Bell’s views became widely accepted. Donald Munro (1889 – 1973) in Boston emphasised the importance of not only managing urinary retention, but also preventing pressure ulcers, treating chest infections and immobilizing patients to limit deformity [4, 5]. During his period at Stoke Mandeville

Hospital in the U.K., Ludwig Guttman (1899 – 1980) formulated the following guidelines: patients should be cared for in specialized units, receive immediate attention by appropriate specialists, undergo continuous monitoring of multiple body systems with thorough documentation, be provided with appropriate aftercare including rehabilitation and vocational retraining, all under the jurisdiction of the public health service and with the cooperation of the Ministry of Pensions and employer [4, 5]. Guttman emphasized motivating patients and founded the Paralympics. His integrated treatment program became the mainstay of SCI management in the mid to late 20th century, refocusing priorities to psychological rehabilitation and prolonged survival while shifting the emphasis away from surgery. However, over the last 40 years surgery has re-gained popularity in attempts to mitigate secondary injury by rapid decompression [6-8]. The effect of early decompression on neurological outcome remains difficult to quantify.

LESSONS FROM TRAUMATIC BRAIN INJURY

Since the brain and spinal cord are composed of the same cell types, SCI must share common pathological mechanisms with traumatic brain injury (TBI). Techniques for invasive monitoring in the setting of TBI have evolved to allow for quantification of intracranial pressure (ICP), cerebral perfusion pressure (CPP) and autoregulation [9, 10]. CPP is the pressure driving blood flow to the brain. Autoregulation provides for constant cerebral blood flow (CBF) regardless of CPP. In the normal state this provided a continuous supply of oxygen and the brain's main energy substrate - glucose.

After TBI, the brain swells against the non-expansile dura and skull. Initially, compensatory mechanisms (reduction in intracranial venous and cerebrospinal fluid (CSF) volumes) buffer rise in ICP. As the compensatory mechanisms become exhausted autoregulation fails, ICP

rises, and CPP falls (Monro-Kellie doctrine). Thus, CBF decreases causing drop in brain tissue oxygen (P_{btO_2}) and glucose.

In patients suffering severe TBI, ICP is monitored using an intraparenchymal pressure probe and compared to MAP obtained by an indwelling arterial catheter. This allows the CPP to be calculated ($CPP=MAP-ICP$) [11]. Tissue metabolites and P_{btO_2} may be monitored using microdialysis (MD) catheters [12] and tissue oxygen electrodes [11]. It is possible to apply these same concepts and techniques to the injured spinal cord, substituting Spinal Cord Perfusion Pressure (SCPP) for CPP, Intraspinal Pressure (ISP) for ICP, and spinal cord tissue oxygen partial pressure (P_{sctO_2}) rather than P_{btO_2} .

MONITORING FROM THE INJURY SITE IN SCI

Intraspinal Pressure. After severe SCI, Intraspinal Pressure (ISP) may be monitored by surgically implanting an intradural extramedullary pressure transducer at the injury site (Fig.

1) [13-15]. The ISP signal has interesting properties:

- 1) At the injury site, subdural ISP is comparable to intraparenchymal pressure.
- 2) Normal ISP is comparable to ICP with three peaks (P1 percussion, P2 tidal, P3 dicrotic) and comparable Fourier transforms (prominent cardiac/respiratory components).
- 3) ISP at the injury site is higher than ISP above or below.
- 4) As ISP rises, ISP pulse amplitude also rises and P2 rises above P1 or P3.

These ISP properties support the notion that at injury site the cord swells circumferentially, initially displacing CSF and ultimately becoming compressed against dura. Spinal cord swelling against the dura after SCI is also evident on MRI [16] and can be conceptualized as

resulting in four discrete compartments: (1) CSF above; (2) CSF below; (3) injury site; and (4) extradural compartment (Fig. 2) [17]. This explains why ISP cannot be reliably determined by monitoring lumbar CSF pressure and why ISP is not effectively reduced by draining CSF [18]. Within its fluctuations, the ISP signal contains “hidden information” about the injury site that may be revealed by a visibility graph analysis [19] and non-linear dynamic [20] signal analysis.

Risks of Intraspinal Pressure monitoring: Based on a series of 42 SCI patients with ASIA Impairment Scale (AIS) grades A – C (Table 1) [21], the most common complications of ISP monitoring are CSF-related, and either spontaneously resolve or can be managed by adding extra sutures to the wound. No adverse neurological events have been reported.

Spinal Cord Perfusion Pressure. SCPP is a more accurate way to monitor cord perfusion at the injury site rather than making inferences from mean arterial blood pressure (MAP). This argument is analogous to using CPP rather than MAP in TBI. The widespread use of MAP to manage SCI patients arises because of the invasive nature of measuring ISP requiring open laminectomy and durotomy for implantation. There have been attempts to monitor ISP via a lumbar CSF catheter [22, 23]. Though inserting a lumbar catheter is technically simpler than placing probes at the injury site, in SCI lumbar CSF pressure differs from ISP as the injured cord is compressed against dura [18]. Ideally current guidelines of maintaining MAP at 85 – 90 mmHg for seven days after SCI [24] should be replaced by SCPP guidelines, which would better reflect degree of spinal cord swelling and local (rather than systemic) perfusion. As can be surmised from the formula $SCPP = MAP - ISP$, SCPP may be increased by adjusting inotrope dose to increase the MAP [13].

Implications for wound care. Laminectomies in SCI patients with a swollen spinal cord compressed against dura allow easy transmission of external forces directly to the spinal cord. External compression of the wound caused increased pressure within the spinal cord, causing potentially catastrophic rise in ISP and fall in SCPP (Fig. 3) [13, 21]. Placing gauze pads on either side of the wound or including cross-links in the rod–pedicle screw construct may prevent external forces from compressing the injured cord.

OPTIMIZING CORD PERFUSION

A consequence of spinal cord swelling against unforgiving dura is that spinal cord tissue must obey the Monro-Kellie doctrine with a pressure-volume relation analogous to that of brain [15], i.e. exhaustion of compensatory reserve and loss of autoregulation as the tissue swells. The more impaired the spinal cord, the more deranged its autoregulation. In TBI, autoregulation may be quantified using the pressure reactivity index PRx (running correlation coefficient between ISP and MAP); we term the corresponding SCI parameter spinal PRx (sPRx). $sPRx \leq 0$ indicates intact autoregulation and $sPRx > 0$ deranged autoregulation [13, 25]. sPRx rises as ISP increases; i.e. as the cord swells, autoregulation is lost [13]. The sPRx *versus* SCPP relationship is U-shaped (Fig. 4A); the ideal goal is for an SCPP that optimizes autoregulation, termed SCPP_{opt}. Thus, monitoring and analysis of the sPRx *versus* SCPP curve allows determination of SCPP_{opt} and suggests it generally to be in the range of ~90 mmHg.

Cord hyper-perfusion may be detrimental. As SCPP decreases below SCPP_{opt}, autoregulation is deranged due to cord ischemia. Interestingly, as SCPP increases above SCPP_{opt}, autoregulation is also impaired, which suggests that cord hyper-perfusion is also

detrimental [13]. The mechanism by which hyper-perfusion impairs cord function is unknown but possibilities include:

- 1) *Steal phenomenon*. Hyper-perfusion increases overall spinal cord blood flow (SCBF), but heterogeneously: well-perfused regions become more perfused whereas under-perfused regions less perfused [26].
- 2) *Cord edema*. Hyper-perfusion may exacerbate cord edema at the injury site.
- 3) *Cord hemorrhage*. Hyper-perfusion may cause haemorrhages in the injured cord.

Individualized management. Fig. 4B shows sPR_x versus SCPP plots for two patients (rather than the pooled patient data in Fig. 4A). Interestingly, $SCPP_{opt}$ varies widely between patients [27], which leads to the concept of individualized management based on $SCPP_{opt}$. $SCPP_{opt}$ is likely influenced by several factors that vary between patients, including the extent of damage to spinal cord and blood vessels, pre-existing conditions (hypertension, smoking, diabetes) and the level of injury (because some sites require lower SCBF than others). The concept of individualized $SCPP_{opt}$ may be refined further [27]. First, it is likely there is a range of physiological SCPPs with intact autoregulation, i.e. $SCPP_{opt}$ is a range not a single value. Second, $SCPP_{opt}$ may vary with time in each patient. The latter argues for continuous $SCPP_{opt}$ monitoring, computed and updated every minute (Fig. 4D).

MICRODIALYSIS MONITORING

In addition to invasive ISP monitoring, additional probes may be positioned inside the dura to monitor the partial pressure of oxygen in spinal cord tissue (P_{sctO_2}) and to sample interstitial fluid by microdialysis (MD) [28] (Fig. 5). MD catheters placed on the surface of an organ provide similar readings as intraparenchymal catheters for pig heart [29], liver [30],

esophagus [31] and small bowel [32]. We thus elected to place the MD catheter on the surface of the injured cord, under the dura and arachnoid [28].

The metabolic changes detected by an MD catheter at the injury site correlates with injury severity, SCPP, and injury site metabolism [28, 33]. Inserting two MD catheters, one at the injury site and another below, provides markedly different metabolic profiles; the distal catheter of course demonstrating more normal metabolism than the injury site catheter [17, 28, 33]. These studies indicate that MD monitoring from the injury site is feasible and may provide important information about injury metabolism amenable to intervention.

Information from MD monitoring. Below, we illustrate how MD may be useful:

- 1) *Fever.* Most SCI patients develop fever early after injury [17]. Data obtained from 759 hours of MD monitoring in 44 SCI patients revealed that fever is associated with metabolic stress at the injury site [17]. The fever burden in the first two weeks after SCI predicts AIS grade improvement, independent of other prognosticators, validated in two SCI patient cohorts from different centres. Thus, the hypothesis that eliminating fever improves neurological outcome merits further investigation.
- 2) *Hypothermia.* We used MD monitoring to determine the impact on the injured cord of local hypothermia in five patients with acute, severe thoracic SCI [34]. Cooling altered injury site metabolism (increased tissue glucose, lactate, LPR, glutamate; decreased glycerol) and reduced cord inflammation (reduced tissue IL1 β , IL8, MCP, MIP1 α , MIP1 β). Compared with the pre-cooling baseline, re-warming significantly worsened cord physiology (increased ICP, decreased SCPP), metabolism (increased lactate, LPR; decreased glucose, glycerol) and inflammation (increased IL1 β , IL8, IL4, IL10, MCP, MIP1 α). We concluded that, after SCI, hypothermia was potentially

beneficial by reducing cord inflammation, but re-warming was detrimental due to increased cord swelling, ischemia and inflammation.

- 3) *Pharmacotherapy.* We administered a bolus of dexamethasone intravenously to three SCI patients and monitored its concentration in the serum and injury site microdialysate. Increasing SCPP by ~10 mmHg, increased the entry of dexamethasone into the injury cord ~3-fold suggesting roles for both MD and SCPP in optimizing drug delivery to the injury site [28].
- 4) *Clinical trials.* The above findings suggest that ISP and MD monitoring from the injury site may aid randomized controlled trials (RCTs) examining hypothermia/re-warming [34] and neuroprotective drugs [28]. One needs to first determine conditions that maximize benefits and minimize adverse effects of hypothermia/re-warming on cord metabolism and inflammation, e.g. by slowing the re-warm speed, and to optimize drug penetration at the injury site. Such monitoring was not part of NABIS II [35], Cool Kids [36] or EuroTherm [37], that evaluated hypothermia/re-warming in TBI, or of NASCIS [38], that evaluated methylprednisolone in SCI.

INJURY SITE MONITORING AND OUTCOME

Though definitive proof to establish injury site monitoring improves outcome may depend on RCTs, mounting evidence suggests that ISP and SCPP directed management may be beneficial:

- 1) *Correlations.* In 45 SCI patients with AIS grade A – C, there was strong correlation between mean ISP and mean SCPP on admission in predicting long-term neurological improvement. [39]. AIS C SCI patients exhibited strong correlations between fluctuations in ISP and SCPP, and variations in limb motor scores in the first week after injury [13, 39].

- 2) *Intervention to increase SCPP.* Increasing SCPP in some patients, improves the amplitude of motor evoked responses at or below the injury site [13], limb motor score [13], sensory level [40] and urinary function (manuscript in preparation).
- 3) *Causality analysis.* Establishing causation in medicine is based on the totality of evidence that includes strong association, biological mechanism, consistency of findings, temporal sequence and dose-response [41]. Clive Granger developed a mathematical definition of causality based on the notion that causes precede and help predict effect [42]. To test the hypothesis that increasing SCPP improves limb motor score, we considered the time series SCPP *versus* time and limb motor score *versus* time. We first used earlier limb motor scores to predict future limb motor scores. Then, we used earlier limb motor scores plus earlier SCPP values to predict future limb motor scores. If the inclusion of earlier SCPP values improves the prediction of future limb motor scores, compared with using earlier limb motor scores alone, then SCPP Granger-causes limb motor score. Granger analysis applied to 19 AIS grade C SCI patients, revealed causal relations between ICP, SCPP, LPR and limb motor score, summarized in Fig. 6 [43].

EXPANSION DURAPLASTY

Reducing ISP may be beneficial. An alternative to increasing SCPP after SCI (by augmenting ABP using inotropes) is to reduce ISP, which may be equally or perhaps even more beneficial:

- 1) *Increased SCPP.* Because $SCPP = MAP - ISP$, reducing ISP increases SCPP.
- 2) *Reduced inotrope requirements.* Reducing ISP means lower ABP achieves the same SCPP, thus reducing inotrope requirements. Inotropes cause cardiogenic complications in SCI, especially in patients older than 60 years [44, 45].

- 3) *Shorter stay in Intensive Care Unit (ICU).* In an MRI study of 65 SCI patients, dural cord compression resolved slowly ($t_{1/2} \sim 9$ days) [16], ie. without ISP reduction, SCI patients require prolonged ICU stay to optimize SCPP. Reducing ISP may thus reduce ICU stay.
- 4) *High ISP may be detrimental.* The higher the ISP, the more impaired spinal cord autoregulation becomes [13, 15], and at least theoretically - the higher the risk of blood vessel rupture and intraparenchymal hemorrhage.

Reducing partial pressure of arterial CO₂ or administering intravenous mannitol, reduces ICP but has no impact on ISP [13]; only expansion duraplasty is known to reduce ISP [46].

Expansion duraplasty. Duraplasty involves opening the dorsal dura longitudinally and suturing an elliptical patch of artificial dura to the dural edge (Fig. 7), aiming to increase the space around the injured cord to reduce ISP and enhance cord perfusion. Several lines of evidence suggest that expansion duraplasty may be beneficial after SCI:

- 1) *Exploratory studies:* We assessed the effect of duraplasty on ISP and SCPP after SCI [46]. Compared with bony decompression in 11 patients, bony plus dural decompression in 10 patients reduced ISP by ~ 10 mmHg and increased SCPP by ~ 15 mmHg. The injured cord expanded into the additional intradural space that had been created by the duraplasty. Beneficial effects of duraplasty after SCI have also been reported in another study [47]. This may be considered analogous to fasciotomies for compartment syndrome in traumatized limbs.
- 2) *ISP monitoring.* A key finding in our injury site monitoring studies, is that ISP remains high with low SCPP even after antero-posterior bony decompression [13, 48] thus suggesting the dura as a source of ongoing cord compression.

- 3) *MRI scans.* In 65 SCI patients without bony compression, dural restriction was evident on MRI as lack of CSF around the injured cord [16]. The degree of spinal cord swelling against the dura was associated with increasing AIS grade.
- 4) *Animal studies.* Reducing ISP by duraplasty or by deletion of the astrocyte water channel aquaporin-4 limiting cord edema, improved outcome in numerous rodent [49-54] and dog [55] SCI studies.
- 5) *Analogy with TBI.* The dura is non-elastic [56]. It is well-established that the dura restricts brain swelling; decompression for TBI is based on removing bony and dural restrictions, shown to lower mortality in the RESCUEicp RCT [57]. We propose that expansion duraplasty for SCI is also analogous to decompressive craniectomy for TBI.
- 6) *Syringomyelia.* Another potential benefit of early duraplasty may be to prevent delayed syringomyelia [58]. Syrinx arises when scarring tethers the injured cord to the dura, which causes delayed neurological deterioration [59]. In rodent SCI models, early duraplasty reduced cord inflammation and scarring resulting smaller syrinx formation [50].

Risks of expansion duraplasty. Expansion duraplasty has been reported in two series of patients with acute SCIs without serious morbidity or mortality [46, 47]. Duraplasty is also performed in Chiari malformation to decompress the cranio-cervical junction. The commonest complications are CSF-related (Table 2). The incidence of wound infection is very low (<1 %) and easily treated with antibiotics. Septic meningitis is rare.

Reducing risks of duraplasty. Several technical nuances may minimize the risks of infection, CSF leak and pseudomeningocele after duraplasty for SCI:

- 1) Careful integration of the dural graft into native dura with running sutures.
- 2) Avoid fibrin glues which expand and can cause cord compression
- 3) Use running locking sutures for fascial and skin apposition, providing two layers of watertight closure.
- 4) Place purse-string sutures around probes to tighten the skin against the probe.
- 5) Apply a waterproof film dressing (e.g. Ioban®) over the wound for one week.
- 6) Postoperative antibiotic prophylaxis.
- 7) Place wound drains open to gravity for a week.
- 8) Nurse patients with cervical SCI at 45 degrees upright for a week to reduce CSF pressure.

RCT of expansion duraplasty in SCI. An RCT, termed DISCUS (Duraplasty for Injured cervical Spinal Cord with Uncontrolled Swelling) is being planned to test whether expansion duraplasty + bony decompression improves outcome compared with bony decompression alone in AIS A – C cervical SCI patients. The trial will begin recruiting in 2021 and aims for 222 patients to be randomized 1:1 in each trial arm. The primary outcome is change in motor score at 6 months compared to baseline. The estimated sample size provides 85% power and 5% significance (two-sided) to detect at least 11-point improvement in the change in total limb motor score at 6 months in the intervention arm, allowing 15 % patient loss to follow-up. DISCUS includes monitoring of ISP +/- MD from the injury site as optional extra.

CONCLUSIONS

Invasive monitoring from the injury site in SCI patients is feasible. A variety of data can be gathered that may be useful to guide management. A key finding is that after SCI, spinal cord swelling is restricted by the dura resulting in high intraspinal pressure and reduced cord

perfusion. An RCT termed DISCUS is being set up to evaluate expansion duraplasty as a novel treatment for SCI. Invasive monitoring from the injury site will also be examined in the DISCUS trial.

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FIGURE LEGENDS

Fig. 1. Monitoring setup. **A.** A tunneler pulls the pressure probe through the skin into the wound. **B.** The dura is perforated with a 90° bent needle one level below the injury. **C.** The pressure probe is inserted through the dural perforation. **D.** The incision is closed and the probe is secured to skin. **E.** CT checks probe position. **F.** Monitoring trolley kept behind patient bed in ICU. Trolley carries laptop, ICP box and monitoring system. Taken from [13].

Fig. 2. Compartmentalization after SCI. MRI (*left*) and schematic (*right*) of the four compartments that form after SCI. 1. Intrathecal above injury, 2. Extrathecal, 3. Intrathecal at injury site (cord compressed against dura), 4. Intrathecal below injury. Adapted from [60].

Fig. 3. Effect of wound compression on ISP and SCPP. **A.** Compressing skin incision in (*left*) laminectomized (Lami) and (*right*) non-laminectomized (No lami) patients with corresponding ISP and SCPP traces. **B.** Δ ISP and Δ SCPP from wound compression (Lami n = 10, No lami n = 7). Mean \pm standard error, $P < 0.001^{***}$. Adapted from [13].

Fig. 4. SCPP_{opt} concept. **A.** sPRx vs. SCPP using pooled data from 45 patients. Dotted line shows pooled SCPP_{opt}. **B.** sPRx vs. SCPP for two patients (a, b). Dotted line shows each patient's SCPP_{opt} computed from entire monitoring period **C.** Individual SCPP_{opt} values for 45 patients. Nil: SCPP_{opt} not computable. Mean \pm standard error. **D.** Continuous SCPP vs. time. sPRx zones: Red (loss of autoregulation), Green (preserved autoregulation) and Yellow (transition from preserved to impaired autoregulation). Line is actual SCPP. Adapted from [27].

Fig. 5. MD monitoring at injury site. **A. i.** Insertion of ISP probe and MD catheter under dura and arachnoid. **ii.** Magnified view of dural entry site. Arrowheads show arachnoid. **iii.** Sutured dural entry site. **iv.** Prone patient with cervical SCI at end of surgery. LMS, lateral mass screw. **B.** Postoperative CT scan of cervical spine. Adapted from [28].

Fig. 6. Granger causality relations in SCI. Each arrow indicates the direction of information flow, i.e. causal influence. “+” or “-” indicate the correlation between the variables. For further details of about causality analysis see [43].

Fig. 7. Expansion duraplasty for SCI. **A.** (*left*) Exposed dura after laminectomy. (*middle*) Durotomy held open with forceps showing injured spinal cord and ISP probe. (*right*) Sutured dural patch. **B.** Pre-operative T2 MRI showing high signal at site of SCI. **C.** Post-operative (*left*) CT showing ISP probe and (*right*) T2 MRI showing duraplasty. Adapted from [46].

Table 1. Risks of ISP monitoring from the injury site. For further discussion of complications see [21].

COMPLICATION	PATIENTS		COMMENTS
	NO.	%	
CSF leak	3 / 42	7	Resolved by wound re-suturing
Wound infection or breakdown	0 / 42	0	
Meningitis (septic)	0 / 42	0	
Pseudomeningocele	8 / 42	19	All resolved at 6-month MRI
Probe displacement	1 / 42	2	
Neurological deterioration	0 / 42	0	
Hematoma	0 / 42	0	

Table 2. Complications of expansion duraplasty for SCI.

COMPLICATION	PATIENTS		COMMENT
	NO.	%	
<i>Duraplasty for SCI</i> [46, 47]			
CSF leak	4 / 26	10	Treated successfully with lumbar drain
Wound infection	0 / 26	0	
Meningitis (septic)	0 / 26	0	
Pseudomeningocele	5 / 26	19	All resolved at 6-month MRI

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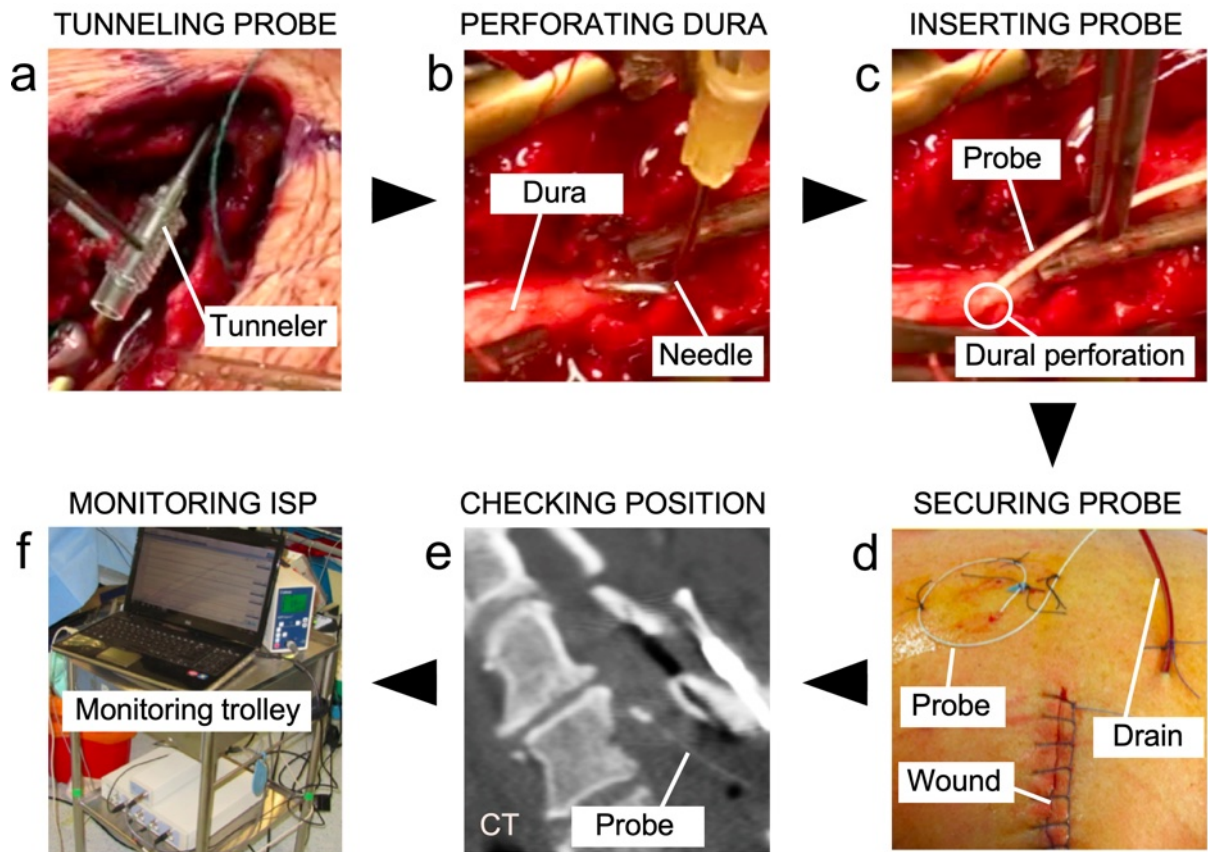


FIGURE 1

FOUR COMPARTMENTS

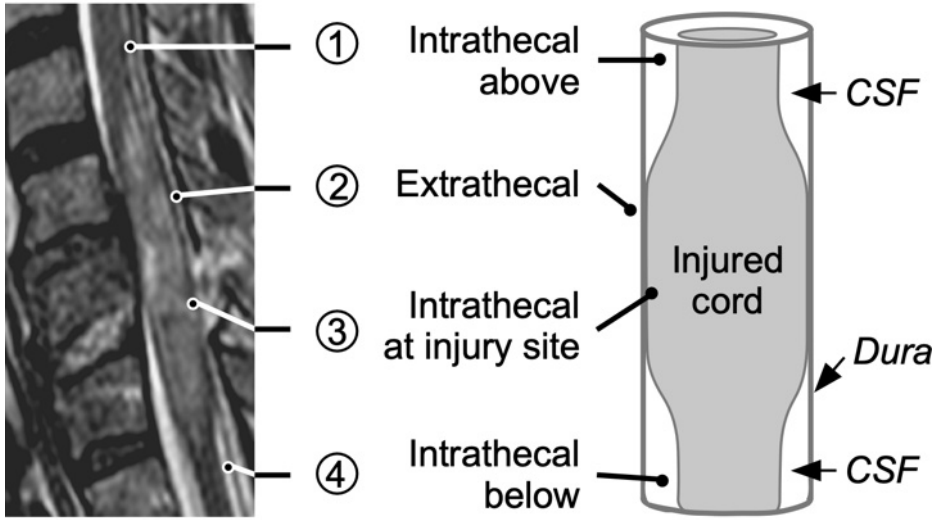


FIGURE 2

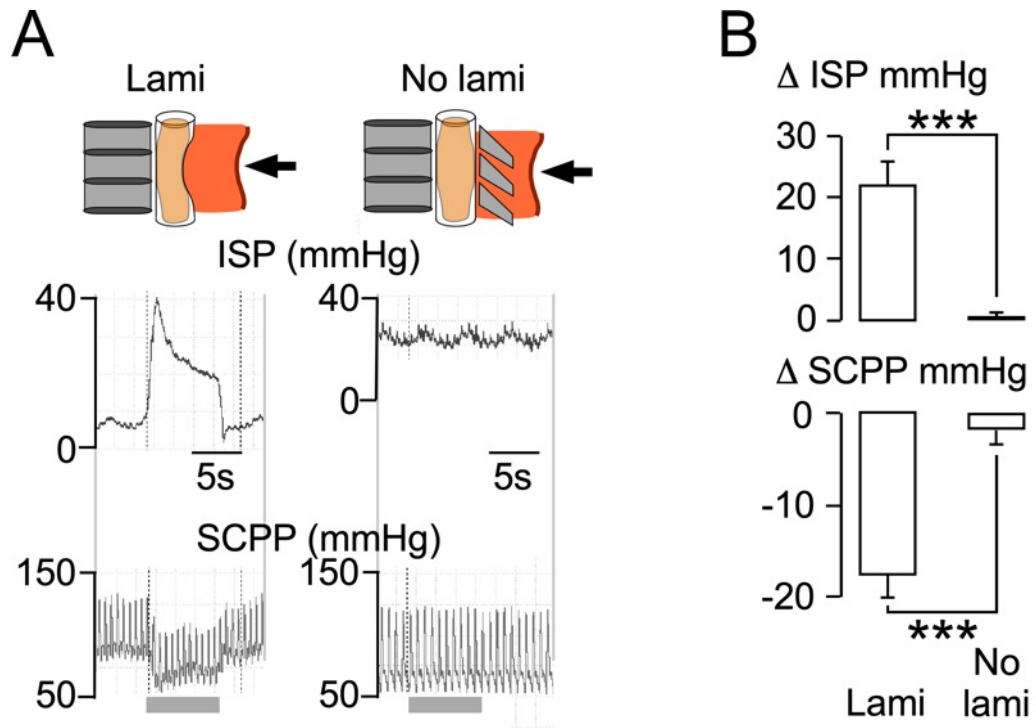


FIGURE 3

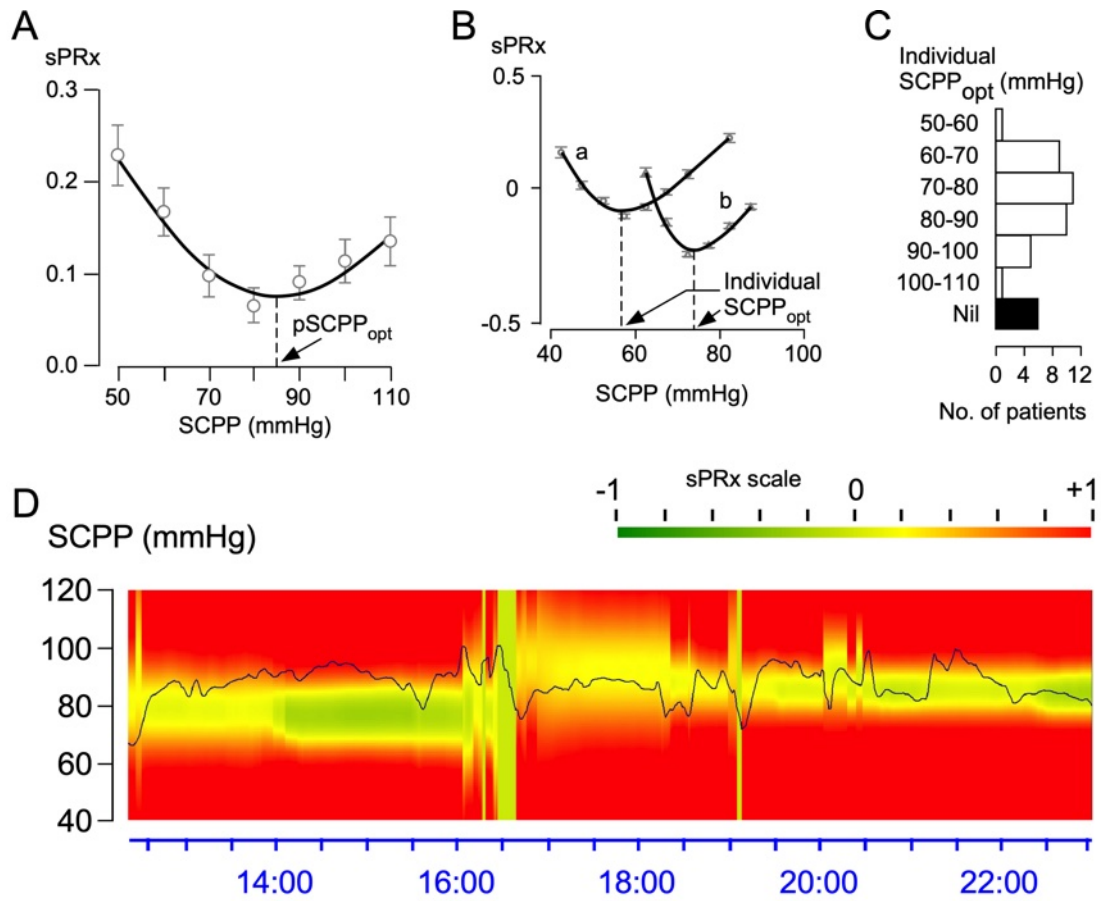


FIGURE 4

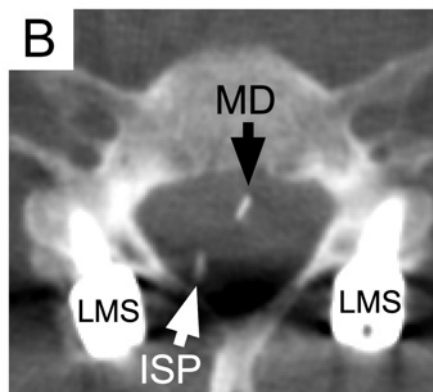
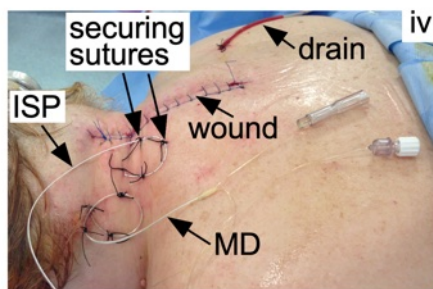
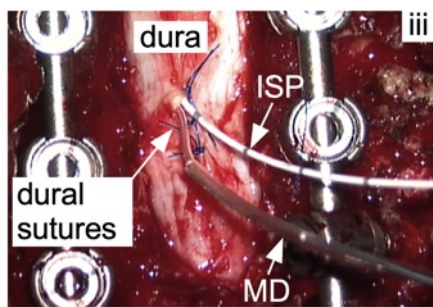
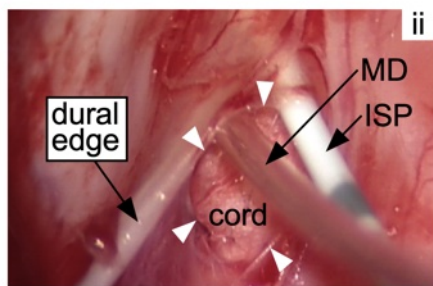
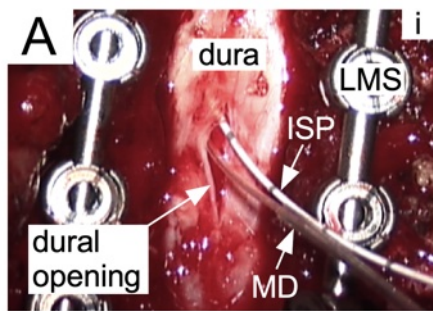


FIGURE 5

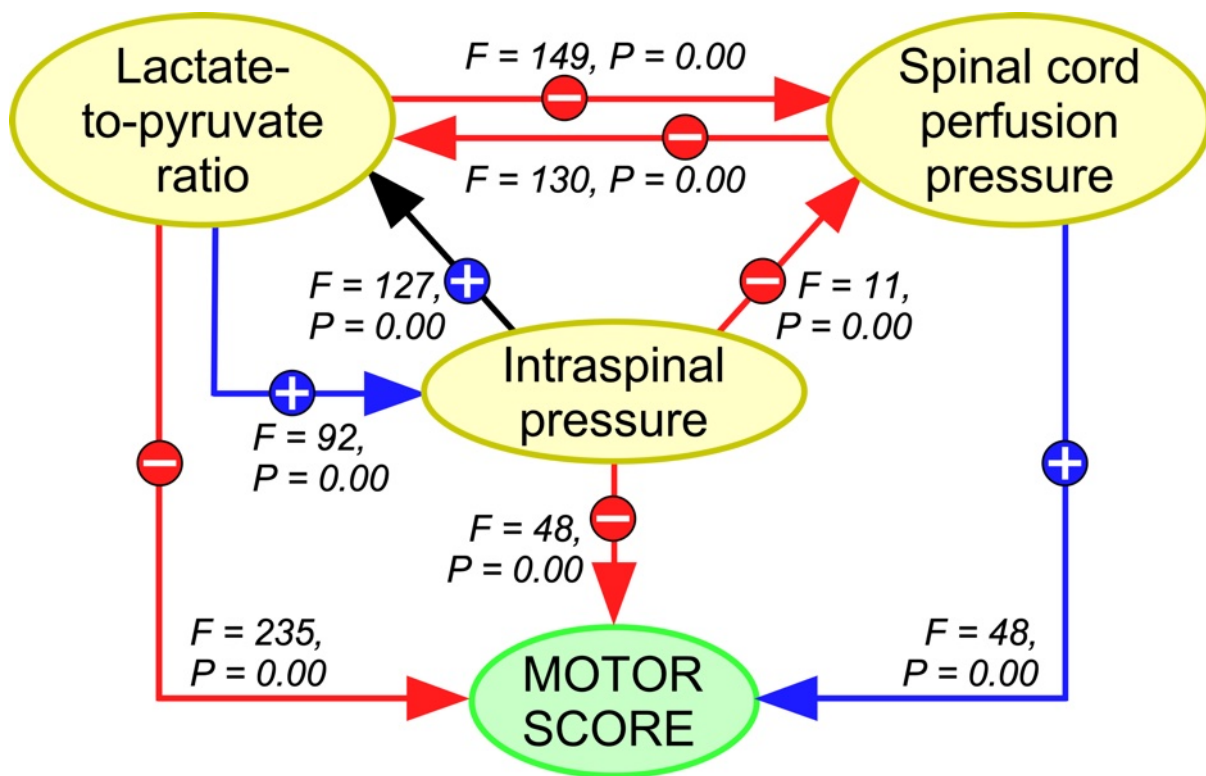


FIGURE 6

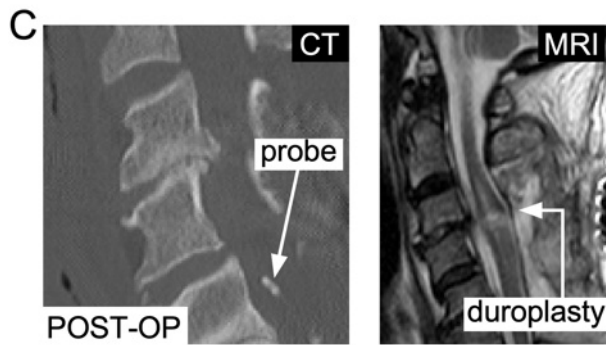
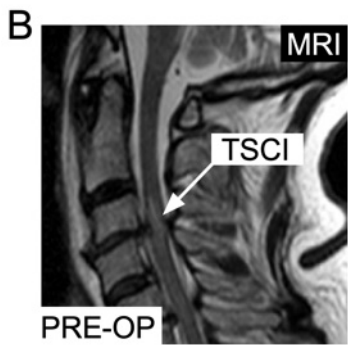
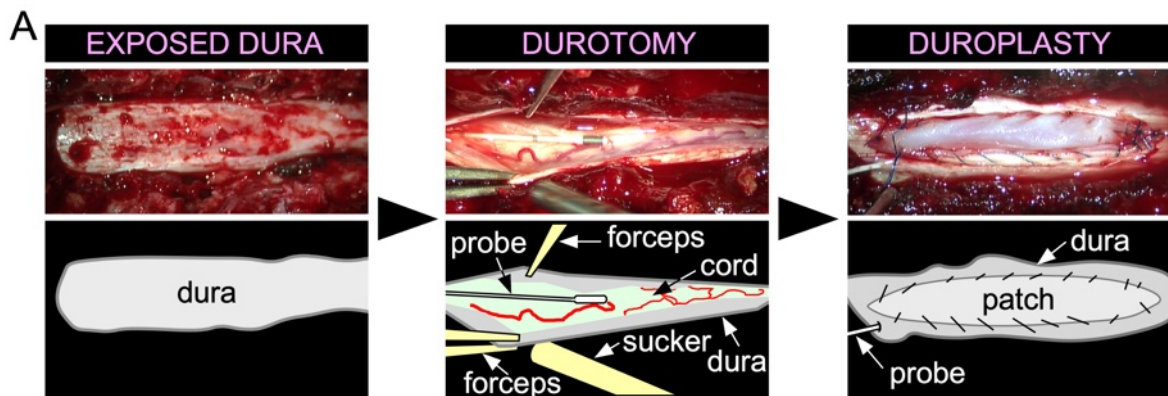


FIGURE 7