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The concepts of Intra Spinal Pressure (ISP), Intra Thecal Pressure (ITP), and Spinal Cord Perfusion Pressure (SCPP) in acute, severe traumatic spinal cord injury: Narrative review

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ABSTRACT

There is increasing interest in monitoring pressure from the injured spinal cord to guide the management of patients with acute, severe traumatic spinal cord injuries (TSCI). This is analogous to monitoring intracranial pressure and cerebral perfusion pressure in traumatic brain injury (TBI). Here, we explore key concepts in this field and novel therapies that are emerging from these ideas. We argue that the Monro-Kellie doctrine, a fundamental principle in TBI, may also apply to TSCI as follows: The injured cord swells, initially displacing surrounding cerebrospinal fluid (CSF) that prevents a rise in spinal cord pressure; once the CSF space is exhausted, the spinal cord pressure at the injury site rises. The spinal Monro-Kellie doctrine allows us to define novel concepts to guide the management of TSCI based on principles employed in the management of TBI such as intraspinal pressure (ISP), intrathecal pressure (ITP), spinal cord perfusion pressure (SCPP), spinal pressure reactivity index (sPRx), and optimum SCPP ($SCPP_{opt}$). Draining lumbar CSF and expansion duroplasty are currently undergoing clinical trials as novel therapies for TSCI. We conclude that there is acknowledgement that blood pressure targets applied to all TSCI patients are inadequate. Current research aims to develop individualised management based on ISP/ITP and SCPP monitoring. These techniques are experimental. A key controversy is whether the spinal cord pressure is best measured from the injury site (ISP) or from the lumbar cerebrospinal fluid (ITP).

1. Introduction

The management of acute, severe traumatic spinal cord injury (TSCI) markedly differs from the management of acute, severe traumatic brain injury (TBI). This is surprising considering that the brain and the spinal cord are comprised of the same cell types similarly arranged, which implies that they respond to trauma in the same way. Here, we argue that basic principles developed to aid the management of acute, severe TBI in the intensive care unit (ICU), such as intracranial pressure (ICP), cerebral perfusion pressure (CPP), and decompressive craniectomy (DC) also apply in TSCI. We discuss research attempting to define analogous concepts in TSCI and provide ideas for future research.

2. Current management

2.1. Traumatic brain injury

Patients with severe TBI have CT imaging of the brain and are urgently transferred to an ICU for monitoring ICP and CPP (Rabelo et al., 2021). TBI management focusses on optimising each patient's ICP and CPP to prevent secondary brain damage. ICP and CPP vary between patients, and the causes for high ICP and low CPP (e.g. intracranial haematoma, cerebral contusion, and diffuse brain oedema) also vary. Different treatments are employed including osmotic diuretics, removal of haematoma, and DC. CT imaging and ICP/CPP monitoring thus allow individualised management of TBI patients, by neurosurgeons and ICU physicians specialising in neurotrauma.

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2.2. Traumatic spinal cord injury

TSCI patients are managed by orthopaedic surgeons or neurosurgeons. Many TSCI patients are not admitted to ICU. A CT scan of the spine is done to define the bony damage. Unlike TBI, where the CT provides useful information about the injured brain (e.g. haematoma, effacement of ventricles, effacement of basal cisterns, midline shift), in TSCI an MRI is required to visualise the spinal cord, which is cumbersome to obtain as an emergency. Surgical decisions focus on how to fix the spine (e.g. anterior *vs.* posterior *vs.* 360° approach) and how many levels to fix. The optimal timing of surgery (early ≤24 h *vs.* late >24 h) has been vigorously debated with most surgeons currently advocating early decompression and fixation (Ter et al., 2024). What constitutes effective spinal cord decompression in TSCI has received little attention (Aarabi et al., 2019). Most surgeons request a CT after spinal fixation to visualise the metalwork and to demonstrate restoration of normal spinal alignment. CT provides little information about the adequacy of spinal cord decompression, in contrast to brain CT in TBI that clearly shows whether the brain has been well-decompressed. Thus, the surgical emphasis in TSCI is on bony fixation, whereas in TBI it is on effective decompression of the injured brain.

The haemodynamic management after TSCI is also poorly defined (Evaniew et al., 2024). The lack of monitoring from the injury site, analogous to ICP and CPP monitoring in TBI, means that doctors are unaware of the status of the injury site, thus making it impossible for them to initiate meaningful treatment to prevent secondary spinal cord damage. Management guidelines often mention maintaining mean arterial pressure (MAP) 85–90 mmHg for a week after TSCI injury (Saadeh et al., 2017), but with weak evidence that this is beneficial and with some evidence that it is detrimental in the elderly (Werndle et al., 2016; Inoue et al., 2014; Readdy et al., 2015). Surely, what matters in TSCI is not the MAP, but the spinal cord perfusion pressure (SCPP), a concept analogous to the CPP in TBI (Werndle et al., 2016). To illustrate the superiority of SCPP over MAP, consider patient with a swollen and compressed spinal cord (high ISP) who will require a higher MAP to maintain adequate spinal cord perfusion than a patient with a well-decompressed spinal cord (low ISP).

3. Pressure-volume relationship at the injury site

A basic concept in TBI is the Monro-Kellie doctrine, which states that the sum of the volumes of brain, CSF, and intracranial blood is constant and thus an increase in one should cause a decrease in one or both of the other two (Rabelo et al., 2021). After TBI, the brain swells initially displacing venous blood and CSF, which are low-pressure compartments, thus preventing a rise in ICP. When the CSF and venous blood spaces become exhausted, the brain then becomes compressed generating a steeply rising ICP with increased brain swelling. This concept explains the exponential pressure-volume relationship of the intracranial system.

It is likely that the Monro-Kellie doctrine also applies in TSCI (Fig. 1). The injured spinal cord swells thus displacing venous blood and surrounding CSF, to prevent a rise in spinal cord pressure. Once the CSF and venous blood spaces become exhausted, the swollen spinal cord becomes compressed against the surrounding theca and spinal cord pressure at the injury site will begin to rise. After TSCI, there is often external compression of the theca (e.g. by bone, haematoma, disc etc) which reduces the compensatory reserve space around the injured spinal cord. The Monro-Kellie doctrine likely still applies in TSCI even in the absence of extrathecal compression, e.g. following bony decompression including laminectomy. In the absence of extrathecal compression, there is more compensatory reserve space around the injured spinal cord; however, the swollen injured spinal cord will ultimately become compressed against the non-stretchable theca. The theca has a high elastic modulus, axially, of 5–20 MPa (Pearcy et al., 2022), which means that changes in pressure within the physiologically relevant range i.e. 0 – 50 mmHg (0–0.0067 MPa) would not produce any appreciable stretching of the theca. These arguments apply to pressure changes that happen within a few days. The theca may stretch, and the adjacent bone may remodel, in conditions of chronically elevated pressure, which are not relevant here. This argument means that it is possible to define the concept of Intra Spinal Pressure (ISP) in TSCI analogous to ICP for TBI.

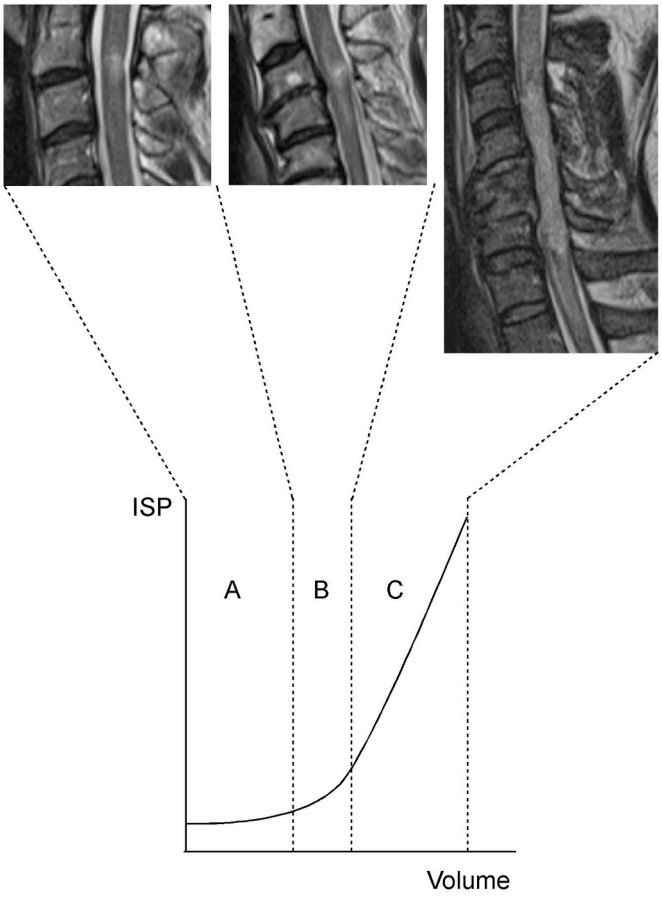

Fig. 1. Monro-Kellie doctrine applied to TSCI. **A.** CSF around injury site. Further swelling of spinal cord displaces CSF and does not increase ISP at the injury site. **B.** Injury site in contact with dura. Further swelling of spinal cord begins increase ISP at the injury site. **C.** Injury site in compressed against dura. Further swelling of spinal cord causes exponential increase ISP at the injury site.

What contributes to elevated pressure of CNS tissue at the injury site in brain *vs.* spinal cord? In TBI, brain oedema and various types of intracranial haematoma (extradural, subdural, intraparenchymal) are the main causes of elevated ICP. In TSCI, the elevated ISP arises from cord compression external to the dura by bony fragments, disc material and haematoma. What is less well appreciated, and by analogy to TBI, the elevated ISP also arises from the swollen spinal cord (by cord oedema, intramedullary haematoma) becoming compressed against the surrounding dura (Saadoun et al., 2016).

4. Monitoring spinal cord pressure

4.1. Intra spinal pressure (ISP)

ISP, defined as the pressure within the spinal cord at the injury site, is technically difficult to measure safely, because inserting a pressure probe into the injured cord will potentially exacerbate cord damage. Some groups monitor ISP by placing a pressure probe between the injured cord and the dura at the injury site (Dhaliwal et al., 2023; Werndle et al., 2014). When the injured spinal cord is compressed against surrounding structures, this technique measures ISP, which

differs from cerebrospinal fluid pressure (CSFP) above the injury site, CSFP (Zeiler et al., 2018, 2019) below, and extradural pressure, i.e. there is compartmentalisation at the injury site (Fig. 2). When the injured spinal cord is not compressed, this technique measures CSFP which, with the patient, lying horizontally, is the same throughout the CSF spaces. When there is CSF around the injury site, the pressure inside the spinal cord at the injury site may not equal CSFP, if the pia is intact. In normal spinal cord, the pia is tough enough to allow high pressure to be generated inside the cord without spinal cord swelling, by ~70 mm Hg (cervical) and ~50 mm Hg (thoracic) (Harwell et al., 2016). Experiments in rodents indicate that, after severe TSCI, the pia becomes damaged and no longer restricts spinal cord swelling or spinal cord compression against the dura (Khaing et al., 2017, 2020, 2021).

There are limitations to inserting a pressure probe intradurally to measure ISP (Phang et al., 2016a). First is the risk of exacerbating spinal cord damage whilst inserting the probe. Second, is the risk of pseudomeningocele and/or CSF leak through the surgical wound. Third, the probe can only be inserted under direct vision using a microscope in the operating room. Data from 42 patients who had ISP monitoring (Phang et al., 2016a) show that the technique is safe: complications were probe displacement in 1/42 patients (2.4 %), CSF leakage that required wound re-suturing in 3/42 (7.1 %), and asymptomatic pseudomeningocele in 8/42 patients (19.0 %) that resolved within 6 months in all patients. ISP measurements have provided invaluable information to define the physiology of spinal cord swelling and spinal cord perfusion after TSCI.

4.2. Biophysics of the ISP signal

The ISP signal is remarkably similar to ICP (Werndle et al., 2014; Varsos et al., 2015) with three peaks: P1 (percussion wave generated from arterial pulsation), P2 (tidal wave, represents spinal cord compliance), and P3 (dicrotic wave, produced by aortic valve closure). Fourier analysis reveals frequency peaks for heart rate, respiration, and harmonics. With increasing ISP, P2 rises with the ISP waveform ultimately becoming rounded and losing the individual peaks. These changes in the ISP waveform shape with increasing ISP further support the notion that the Monroe-Kellie doctrine, initially developed in TBI, also applies to TSCI at the injury site. This means that we can define the concept of compensatory reserve, i.e. how much space there is around the injured spinal cord to allow cord swelling without generating high ISP. In TBI, compensatory reserve is measured using the RAP index, computed as the correlation coefficient (R) between the ICP pulse amplitude AMP (A) and the mean ICP (P) (Zeiler et al., 2018, 2019). RAP = 0 on the linear part of the pressure-volume curve, +1 on the ascending exponential part, and ≤0 as ICP increases further related to the critical closing of cerebral vessels. By analogy with RAP in TBI, we defined sRAP in TSCI as the spinal compensatory reserve index and showed that, as ICP rises and as SCPP falls, compensatory reserve is exhausted (Werndle et al., 2014).

4.3. Autoregulation and optimum spinal cord perfusion

A basic concept in neurotrauma is cerebral autoregulation, which is the ability of the brain to maintain constant cerebral blood flow (CBF) independent of changes in arterial blood pressure (ABP). Intact autoregulation means that an increase in ABP causes vasoconstriction, whereas a decrease in ABP causes vasodilation, to maintain constant CBF. Vasoconstriction will decrease the ICP whereas vasodilatation will increase the ICP. Thus, with intact autoregulation, there is a negative correlation between the ABP and the ICP signals. When autoregulation is impaired in TBI, CBF, and thus ICP, increases (or decreases) as ABP increases (or decreases) and, therefore, there is a positive correlation between ABP and ICP. The running correlation coefficient between ICP and ABP, termed 'Pressure Reactivity index' or PRx is widely used to quantify cerebral autoregulation (Donnelly et al., 2019; Czosnyka et al., 2017).

An analogous concept may also be defined in TSCI, i.e. the running correlation coefficient between ISP and ABP, termed the 'spinal PRx' or sPRx (Werndle et al., 2014). Fig. 3 shows plots of ISP *vs.* PRx and SCPP *vs.* PRx for two patients with acute, severe TSCIs. As ISP rises, autoregulation becomes increasingly disrupted in both patients. Each patient has a different optimum SCPP ($SCPP_{opt}$) defined as the minimum of the sPRx *vs.* SCPP curve. Reducing the SCPP below $SCPP_{opt}$, causes spinal cord hypoperfusion with more disrupted autoregulation. Increasing the SCPP above $SCPP_{opt}$, causes spinal cord hyperperfusion, again with more disrupted autoregulation. $SCPP_{opt}$ not only varies between patients, but $SCPP_{opt}$ also varies with time in each patient (Chen et al., 2017). The concept of $SCPP_{opt}$ may be further refined to be a range rather than a single value, defined as the range of SCPP that maintains sPRx <0.2. Targeting SCPP within a range is more practical, than trying to achieve a single SCPP value.

4.4. Intra thecal pressure (ITP)

Another way of measuring pressure is by inserting a lumbar catheter (Squair et al., 2017; Kwon et al., 2009). This pressure, termed intrathecal pressure (ITP), is the CSFP in the lumbar subarachnoid space. Advantages of this technique, compared with ISP monitoring, are familiarity with inserting a lumbar catheter, ease of insertion, insertion done in the ICU without surgery, and no risk of damaging the injured cord. The lumbar catheter may also be used to drain CSF to reduce the ITP, potentially increasing spinal cord perfusion. The idea of monitoring ITP comes from vascular surgery, to monitor spinal cord perfusion in thoracoabdominal aneurysm repair and drain CSF to reduce spinal cord ischemia (Blakeslee-Carter et al., 2024).

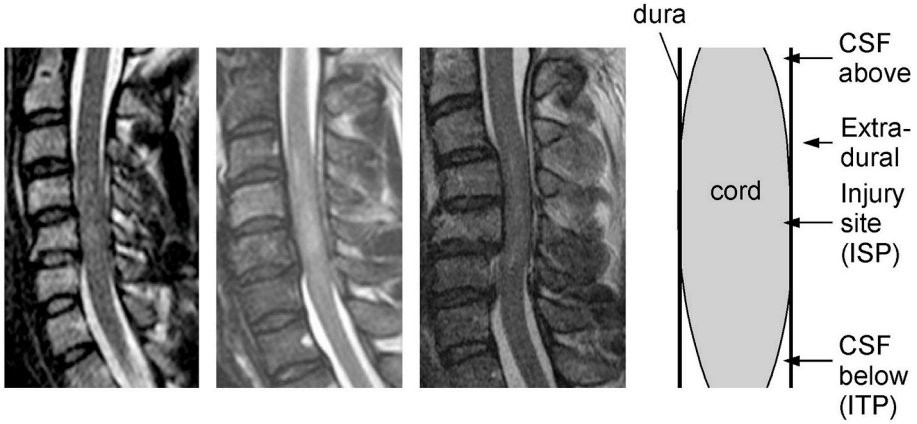

Fig. 2. Compartmentalisation at injury site. T2 sagittal MRIs of 3 patients showing swollen spinal cords compressed against dura. Compression creates 4 compartments, each having a different pressure: CSF above, extradural, injury site (ISP), and CSF below (ITP).

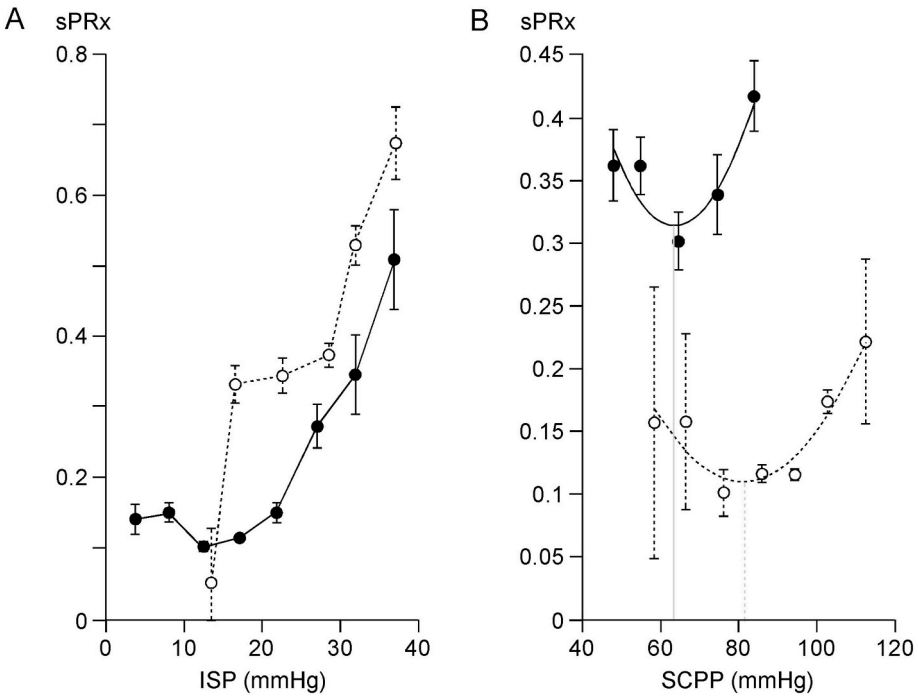

Fig. 3. Spinal cord pressure reactivity index (sPRx). **A.** sPRx *vs.* ISP, and **B.** sPRx *vs.* SCPP, for two patients (white dots, black dots) with different $SCPP_{opt}$ at 66 and 82 mmHg.

When there is CSF around the injured spinal cord, ISP (measured at the injury site using an intradural, extramedullary probe) = ITP. In this situation SCPP, calculated as mean arterial pressure (MAP) minus ISP (measured at the injury site using an intradural, extramedullary probe), is the same as SCPP calculated as MAP – ITP. If the pia mater is intact, the pressure inside the injured spinal cord may be higher than CSFP; however, CSFP contributes to the pressure inside the spinal cord and thus draining CSF should reduce spinal cord pressure, as done during thoracoabdominal aneurysm repair (Blakeslee-Carter et al., 2024).

When the injured spinal cord is compressed, ISP (measured at the injury site using an intradural, extramedullary probe) ≠ ITP. In this case, SCPP computed as MAP – ISP ≠ SCPP computed as MAP – ITP. Therefore, draining CSF from the lumbar subarachnoid space is expected to reduce ITP and increase SCPP (computed as MAP – ITP), but not to have an appreciable effect on ISP or SCPP (computed as MAP – ISP). In 12 patients with acute, severe TSCI, there was no correlation between mean 24-h ISP (measured at the injury site using an intradural, extramedullary probe) and simultaneously measured mean 24-h ITP (Hogg et al., 2020a). Examination of the ISP and ITP traces revealed periods when ITP = ISP and periods when ITP ≠ ISP. This finding suggests that, following surgery, spinal cord compression against the dura is dynamic, and that ITP may be an inadequate estimate of ISP.

Unlike ISP, ITP cannot be used to compute other physiological parameters of the injured spinal cord such as pressure reactivity and optimum SCPP, defined below. Monitoring from the lumbar CSF compartment precludes multi-modality monitoring including microdialysis (Phang et al., 2016b) and spinal cord tissue oxygen ($p_{sct}O_2$) (Visagan et al., 2022), because these probes need to contact spinal cord tissue at the injury site to provide meaningful readings.

5 Novel treatments

5.1. Effect of draining lumbar CSF

Currently, the Canadian/American Spinal Cord Perfusion Pressure and Biomarker Study (CASPER, Clinicaltrials.gov NCT03911492) trial is investigating whether 'the active management of SCPP (computed at MAP – ITP) with maintenance at ≥ 65 mmHg results in better neurologic recovery than conventional hemodynamic management that aims solely to maintain the MAP'. As discussed above, this definition of SCPP is not necessarily the same as SCPP = MAP – ISP. Draining 10 mL CSF from the lumbar subarachnoid space in 12 patients had variable effect on ISP, sometimes decreasing ISP, sometimes increasing ISP, sometimes without changing ISP, but overall having little influence on ISP following several CSF drainages per patient (Hogg et al., 2020a). Draining 10 mL CSF from the lumbar subarachnoid space also had a variable effect on $p_{sct}O_2$, sometimes decreasing $p_{sct}O_2$, sometimes increasing $p_{sct}O_2$, sometimes without change in $p_{sct}O_2$, but overall having little influence on $p_{sct}O_2$ following several CSF drainages per patient (Visagan et al., 2022). These findings suggest that draining lumbar CSF may not effectively improve spinal cord physiology at the injury site. This may be because, unlike TBI where draining CSF from a lateral ventricle may create more intracranial space thus reducing ICP, in TSCI there is no CSF around the swollen cord compressed against the theca and therefore draining CSF does not effectively reduce ISP or increase $p_{sct}O_2$.

5.2. Expansion duroplasty (or duraplasty)

After TBI, the swollen brain becomes compressed against dura and skull. Therefore, DC requires not only removing skull, but also opening the dura to effectively reduce ICP (Schirmer et al., 2008). Decompressing the injured spinal cord merely refers to restoring normal spinal alignment and removing components compressing the spinal cord from outside the dura such as bone fragments, disc protrusions or haematoma. The concept that spinal cord compression may arise because of 'compression from inside the dura', i.e. the cord is expanded and compressed against the dura is not widely appreciated. It is interesting to speculate why the notion of 'compression from inside the dura' has not developed in TSCI.

1. The injured cord cannot be adequately visualised on CT imaging, and most surgeons do not obtain MRIs postoperatively to visualise compression of the injured cord against the dura.

2. The most common surgical pathologies that affect the spinal cord such as disc protrusion, canal stenosis, and ossification of the posterior longitudinal ligament are compressions from structures outside the dura. This introduces bias when thinking about TSCI, that spinal cord compression is also solely from external factors.
3. TSCI is commonly associated with extradural spinal cord compression from bony fragments, which are the most striking feature on the pre-operative MRI making it impossible to visualise 'compression from inside'.
4. Many TSCI patients are managed by orthopaedic surgeons rather than neurosurgeons who may not be very familiar with the TBI concepts of ICP, and CPP.

Recently, some surgeons have used ultrasound imaging intra-operatively to ensure adequate decompression of the spinal cord by showing CSF surrounding the cord (Chryssikos et al., 2023; Ali et al., 2023). Ultrasound is non-invasive, easy to use, and provides clear images of the injured cord. A potential problem is that intraoperative ultrasound may provide false reassurance in case of delayed spinal cord swelling with cord compression against the dura. The analogy with TBI is an early CT showing no brain compression but effacement of basal cisterns after a few days as brain oedema increases, and brain contusions blossom (Carnevale et al., 2018; Ratan et al., 2023). ISP monitoring for several days after TSCI is required to define the evolution of spinal cord swelling after TSCI. Another problem is that intraoperative USS is only feasible following laminectomy and thus impossible to perform with an anterior approach.

The logical extension of the concept of spinal cord compression by dura is surgical decompression not only by laminectomy, but also by duroplasty to reduce ISP, analogous to DC in TBI. Several case series report generally positive findings with duroplasty after TSCI (Phang et al., 2015; Zhu et al., 2019; Garg et al., 2022). A pilot study has shown that the procedure is safe, and effectively reduces ISP, increases SCPP and improves sPRx (Phang et al., 2015). Duroplasty is a simple surgical technique (Phang et al., 2015; Zhu et al., 2019; Garg et al., 2022). After laminectomy, the theca is incised longitudinally under the microscope. An oval-shaped dural patch is then sutured along the thecal edges. The longitudinal extent of the thecal opening is judged from the preoperative MRI to span the extent of cord injury. The width of the patch is about 2 cm. Fig. 4 shows an example of expansion duroplasty in a TSCI patient. Duroplasty is often performed during foramen magnum decompression for Chiari malformation (Yahanda and Limbrick, 2023; Chotai and Medhkour, 2014) but it has not been widely used in TSCI and thus the technique is not standardised in this context. Unanswered questions are whether to open the arachnoid, whether to divide the dentate ligament, whether to place a tenting suture, and whether to use fibrin sealant that may limit CSF leak but cause delayed cord compression. Commonly used dural substitutes are acellular bovine collagen, and endogenous fascia lata (Khurana et al., 2024). In experienced hands the procedure adds about 10–30 min to the operating time and the cost of an artificial duroplasty patch is around €100. The main complication is non-compressive pseudomeningocele evident on postoperative MRI that generally disappears within 6 months postoperatively.

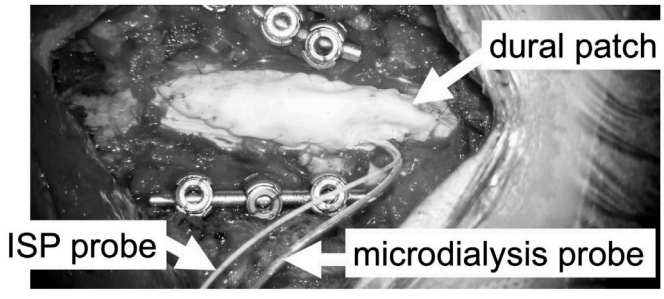

Fig. 4. Example of an expansion duroplasty with ISP and microdialysis probes *in situ*. Intraoperative photo taken under a surgical microscope.

A randomised controlled trial is underway, termed Duroplasty for Injured cervical Spinal Cord with Uncontrolled Swelling (DISCUS), clinicaltrials.gov NCT04936620 (Saadoun et al., 2023). Adult patients with acute severe cervical TSCIs are randomised 1:1 to have bony decompression including laminectomy *vs.* bony + dural decompression. The inclusion-exclusion criteria are listed in Table 1. The primary outcome measure is the change in the motor score of the American spinal injury association Impairment Scale (AIS) at 6 months post-randomisation *vs.* baseline. Monitoring ISP and/or microdialysis from the injury site, to test the hypothesis that duroplasty reduces ISP and improves metabolism (higher tissue glucose, lower tissue lactate-to-pyruvate ratio) at the injury site compared with bony decompression is optional.

5.3. *Effect of intervention to increase SCPP*

Some studies have used multi-modality monitoring from the injury site in which probes were inserted intradurally to monitor not only ISP and SCPP, but also microdialysis (Phang et al., 2016b) and $p_{sct}O_2$ (Visagan et al., 2022) of spinal cord tissue. This is analogous to multi-modality monitoring for TBI which involves monitoring ICP, microdialysis, and brain tissue oxygen (Denchev et al., 2023). Increasing SCPP improved the metabolic (Phang et al., 2016b) and oxygenation (Visagan et al., 2022) profiles of the injured spinal cord including higher tissue glucose, lower tissue lactate-to-pyruvate ratio as well as higher $p_{sct}O_2$.

There are several studies generally reporting positive experiences when intervening to increase SCPP in patients with acute, severe TSCIs. In these studies, SCPP is increased by increasing the MAP using inotropes. Osmotic diuretics, widely used to reduce ICP in TBI, do not effectively reduce ISP in TSCI (Werndle et al., 2016). In a cohort of AIS grade C patients, there was a linear correlation between limb motor score plotted against SCPP (Hogg et al., 2020b). Increasing SCPP has also been shown to lower the sensory level in some patients (Saadoun and Papadopoulos, 2016), and improve urodynamic parameters such as reducing the urinary bladder fill volume that produces the first desire to void (Hogg et al., 2021). In a recent study of patients with severe cervical TSCIs, there was an inverted U-shaped relationship between various measures of diaphragmatic function, primarily derived from ultrasound scanning, *vs.* SCPP (Visagan et al., 2023). There is also a strong correlation between the SCPP averaged over the first two weeks after surgery and long-term neurological outcome quantified as AIS grade conversion (Squair et al., 2017; Saadoun et al., 2017). Together, these findings suggest that SCPP is a fundamental physiological parameter after TSCI.

6. Future directions

Table 2 summaries key analogous concepts in TBI and TSCI. Monitoring ISP (or ITP) is not widely performed and remains experimental. This may be due to surgical pessimism regarding outcomes in TSCI,

Table 1
DISCUS trial inclusion and exclusion criteria.

INCLUSION	EXCLUSION
Age 16 years or older	Probable dural tear due to TSCI
Cervical (C2 – T1) TSCI	Life limiting or rehabilitation restricting co-morbidities
AIS Grade A-C	Thoracic or lumbar TSCI
Deemed to require and be suitable for surgery that includes laminectomy by local surgeons	Other CNS disease
Surgery within 72 h of TSCI	

AIS, American spinal injury association Impairment Scale; CNS, central nervous system; TSCI, traumatic spinal cord injury.

Table 2
Analogies between traumatic brain injury and traumatic spinal cord injury.

TRAUMATIC BRAIN INJURY	TRAUMATIC SPINAL CORD INJURY
ICP, Intra Cranial Pressure	ISP/ITP, Intraspinal pressure/Intrathecal pressure
CPP, Cerebral Perfusion Pressure	SCPP, Spinal Cord Perfusion Pressure
PRx, Pressure Reactivity Index	sPRx, spinal Pressure Reactivity Index
CPP_{opt}, Optimum Cerebral Perfusion Pressure	$SCPP_{opt}$, Optimum Spinal Cord Perfusion Pressure
RAP, Compensatory Reserve Index	sRAP, spinal Compensatory Reserve Index
P1 P2 P3, peaks on normal ICP waveform	P1 P2 P3, peaks on normal ISP waveform
Decompressive craniectomy	Laminectomy + duroplasty

concern of damaging the cord by inserting probes intradurally or draining CSF, concern of causing meningitis or metalwork infection, concern of difficult-to-control CSF leak, or shortage of ICU beds. Current clinical management of acute TSCI with emphasis on improving neurological function remains rudimentary, compared with TBI management where ICP monitoring is widespread. Recent consensus recommendations on the haemodynamic management of TSCI are to maintain an optimal MAP acknowledging uncertainty what is optimum MAP; SCPP remains experimental but with increasing efforts to elucidate this concept (Kwon et al., 2024). The concept of SCPP, while making sense, needs to be validated in large prospective studies; the evidence to date on the role of SCPP monitoring is low and does not at this stage support a shift in practice (Evaniew et al., 2024).

Future developments will likely arise from collaboration between the investigators who monitor ISP/ITP to develop concepts together, share monitoring data, exchange ideas, conduct multi-centre trials (because no single centre can provide enough TSCI patients), and publish consensus recommendations. The development of novel optical and ultrasound probes to monitor from the injury site that can be placed extradurally by percutaneous approach by ICU doctors without surgery may facilitate widespread use of such monitoring techniques in TSCI.

In severe TBI, DC has been shown to reduce mortality (Hutchinson et al., 2016). Many survivors have severe cognitive disabilities, which makes this surgical treatment option controversial. Because the brain has cognition, preservation of brain tissue in severe TBI may increase awareness of the disability enough to increase suffering. The spinal cord is a purely mechanical system lacking consciousness. Thus, any spinal cord tissue preservation in TSCI, by interventions to improve SCPP, should improve quality of life by increasing limb sensation, limb power, sphincter control, thermoregulation, and blood pressure control.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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