

Measurement of intraspinal pressure after spinal cord injury: technical note from the Injured Spinal Cord Pressure Evaluation Study

¹Melissa C. Werndle, ¹Samira Saadoun, ¹Isaac Phang, ²Marek Czosnyka, ²Georgios Varsos,

²Zofia Czosnyka, ²Piotr Smielewski, ³Ali Jamous, ¹B. Anthony Bell, ⁴Argyro Zoumprouli,

¹Marios C. Papadopoulos

¹Academic Neurosurgery Unit, St. George's, University of London, London, UK;

²Department of Neurosurgery, University of Cambridge, Addenbrooke's Hospital,

Cambridge, UK; ³National Spinal Injuries Centre, Stoke Mandeville Hospital, Stoke

Mandeville, UK; ⁴Department of Anaesthesia, St. George's Hospital, London, UK.

Correspondence to: M.C. Papadopoulos, Academic Neurosurgery Unit, Room 1.122 Jenner Wing, St. George's, University of London, Cranmer Terrace, Tooting, London SW17 0RE, U.K. Tel. +44-20-87254179; Fax. +44-20-87255139; Email. mpapadop@sgul.ac.uk

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Summary

Intracranial pressure (ICP) is routinely measured in patients with severe traumatic brain injury (TBI). We describe a novel technique that allowed us to monitor intraspinal pressure (ISP) at the injury site in 14 patients who had severe acute traumatic spinal cord injury

(TSCI), analogous to monitoring ICP after brain injury. A Codman probe was inserted subdurally to measure pressure of the injured spinal cord compressed against the surrounding dura. Our key finding is that it is feasible and safe to monitor ISP for up to a week in patients after TSCI, starting within 72 hours of the injury. With practice, probe insertion and calibration takes less than ten minutes. The ISP signal characteristics after TSCI were similar to the ICP signal characteristics recorded after TBI. Importantly, there were no associated complications. Future studies are required to determine whether reducing ISP will improve neurological outcome after severe TSCI.

Key words

Monitoring, Perfusion pressure, Spinal cord injury

Introduction

After severe traumatic brain injury, the brain swells. This causes a rise in intracranial pressure (ICP) and a fall in cerebral perfusion pressure (CPP = mean arterial pressure (MAP) minus ICP), which may lead to secondary ischaemic brain damage [10]. The early management of severe traumatic brain injury (TBI) aims to reduce the elevated intracranial ICP and increase CPP to reduce secondary brain damage [10]. To achieve this, patients with TBI are urgently transferred to a neurointensive care unit for ICP monitoring. Low CPP (<60 mmHg) and high ICP (>20 mmHg) [3, 6] are associated with worse outcome after TBI.

In contrast to the management of severe TBI, the management of severe acute traumatic spinal cord injury (TSCI) in the neurointensive care is variable. Many anaesthetists do not measure arterial blood pressure invasively and the optimal levels of MAP and arterial pCO₂ (p_aCO₂) are arbitrary [12]. The American Association of Neurological Surgeons guidelines recommend MAP of 85 – 90 mmHg for 5 – 7 days after TSCI [5]. The UK

National Spinal Cord Injury Strategy Board guidelines recommended systolic ABP of 90 – 100 mmHg [8]. However, there is insufficient evidence to support these guidelines.

An key reason why TSCI management is so different from TBI management is because there is no method in clinical use for measuring intraspinal pressure (ISP) after TSCI. Measuring ISP after TSCI would be analogous to measuring ICP after TBI. The ability to measure ISP would be a major advance by allowing the various principles that are used clinically for managing severe TBI (reducing ICP, increasing CPP, optimizing cerebrovascular pressure reactivity) to be adapted for use in TSCI (reducing ISP, increasing SCPP, optimizing spinal cord vascular pressure reactivity). Here we describe our technique for measuring ISP at the injury site after TSCI.

Materials and Methods

Inclusion and exclusion criteria. The ISCoPE (Injured Spinal Cord Pressure Evaluation) study was set up in 2009. The initial findings from the ISCoPE study will be reported in Critical Care Medicine [11]. Approvals were obtained from the St. George’s Joint Research Office and the South London, Maudsley and the Institute of Psychiatry Local Research Ethics Committee (No. 10/H0807/23). We recruited 18 – 70 year old patients with severe TSCI (ASIA grades A – C). Exclusion criteria were inability to consent and other major injuries or significant co-morbidities. ISP monitoring was started within 72 hours of the TSCI and continued for up to a week.

Surgical technique. A Codman pressure probe was chosen because it is already licensed for use in patients and is widely used to measure ICP. The Codman wire is 1 m long (and therefore the patient does not lie on the connector), it has a small diameter of 0.7 mm (thus reducing the risk of spinal injury), it has low 10-day zero drift (<0.2 mmHg / day) and high

response frequency (100 Hz). The ISP probe was placed subdurally following laminectomy or a small laminotomy. The insertion technique is summarised diagrammatically in Fig. 1a. After reducing and fixing the spinal fracture, and inserting metalwork to stabilise the spine, a 14-gauge introducer was used to tunnel a Codman pressure probe through the skin into the wound. We used a 21-gauge needle bent at 90⁰ to perforate the dura one level below the injury. The Codman probe was calibrated and advanced through the dural hole until the probe tip was at the site of maximal spinal cord swelling according to the MR scan. The probe was secured to the skin with silk sutures. We found it important to insert a tightening stitch around the exit site to prevent CSF leak. The probe was connected to a Codman ICP box linked via a ML 221 amplifier to a PowerLab running LabChart v.7.3.3 (AD Instruments, Oxford, UK). Data was captured at 100 Hz. Satisfactory probe position was confirmed with CT before data collection.

Results

Patient recruitment. 14 consecutive patients with TSCI were recruited between October 2010 and September 2012. All except one patient who were approached consented for the study. 57 % TSCI patients were male. 71 % had cervical injuries and 29 % had thoracic injuries. 57 % were ASIA A, 14 % ASIA B and 29 % ASIA C. 21% of TSCI patients were recruited within 24 hours, 36% at 24 – 48 hours, and 43% at 48 – 72 hours.

Surgery and complications. 36 % of TSCI patients had anterior and posterior cervical fusion, 29 % posterior cervical fusion only, and 29 % thoracic pedicle screws. 64 % of TSCI patients had laminectomy. There was a learning curve for the ‘probe insertion and calibration time’, such that initially it took 31 – 43 minutes and by the end it only took 6 – 8 minutes (Fig. 1b). There were no complications related to ISP monitoring such as wound infection, meningitis,

cerebrospinal fluid (CSF) leak, pseudomeningocele, spinal cord or subdural haematoma (as assessed by MRI), or deterioration in ASIA score (before probe insertion *vs.* after probe removal). Fig. 2 shows preoperative and postoperative scans for a patient with cervical spinal cord injury. Followup of 10 patients at 5 – 13 months after the surgery showed that there were no wound-related complications.

ICP signal recording. The ISP signal was recorded for up to a week. Fig. 3a shows that the ISP waveform is similar to the ICP waveform with three peaks corresponding to the arterial pulsation, intracranial compliance and aortic valve closure [1-3]. Representative ISP recordings are shown in Fig. 3b. In some patients, ISP was high (>20mmHg) during the recording period, with ISP reaching very high values (>40mmHg). To put in context, if these were ICP recordings after TBI (rather than ISP recordings after TSCI) then ICP>20mmHg would typically be treated and ICP>40mmHg would be characteristic of a patient at risk of imminent death.

Discussion

We described a novel technique to measure subdural ISP. Our recordings indicate that after TSCI, ISP at the injury site is elevated in some patients. After TBI, high ICP is potentially lethal by causing brain ischaemia and herniation [3, 6, 10]. Future studies are required to determine whether high ISP is harmful in TSCI.

Our ISP monitoring method is technically simple and analogous to the one used to measure ICP. The ISP signal was stable for at least a week without probe-related complications. With experience, the procedure took <10 minutes. Previous attempts to measure ISP after TSCI had little success. Lumbar drains were inserted to measure CSF pressure below the injury [7], which (as shown here) differs from the ISP at the injury site.

Pressure in a spinal radicular artery has been recorded [4], but is technically difficult, risks vascular damage to the spinal cord and does not measure SCPP at the injury site.

The normal spinal cord is surrounded by CSF, which is contained within a non-distensible dural sac. After TSCI, the injured section of the spinal cord swells so that there is no CSF between the spinal cord and the dura. At the injury site, the lack of CSF around the spinal cord decreases the local reserve capacity (which can be quantified using the parameter sRAP, as discussed by Czosnyka et al. in this Supplement). Further spinal cord swelling causes a rapid local rise in ISP (as the spinal cord becomes compressed against the dura), which causes loss of autoregulation (which can be quantified using the parameter sRAP, as discussed by Czosnyka et al. in this Supplement). Together, these findings suggest that the basic concepts developed for managing severe TBI, such as tissue pressure, perfusion pressure, reserve capacity and autoregulation might also be applicable when managing severe TSCI.

In the future, we envisage that after TSCI patients will be admitted in neuro-intensive care units for ISP and arterial pressure monitoring to optimize ISP. For incomplete TSCI, the aim is to improve function below the level of injury and for complete TSCI, to limit cranial extension of the spinal cord damage. Perhaps the technique of ISP monitoring could also be applied to limit spinal cord damage in other conditions that cause high ISP, such as longitudinally extensive transverse myelitis [9].

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Conflict of Interest Statement

The authors report no conflicts of interest.

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Figure Legends

Fig. 1. Insertion of pressure probe. a. 1: A hole is made in the dura, using a 90° bent needle, one level below the injury site. 2: The needle is removed. CSF flows through the dural puncture. 3: Using forceps, the Codman probe is inserted in the subdural space and advanced to lie between swollen spinal cord and dura. 4: A tightening silk stitch is placed at the exit site and multiple stitches secure the probe cable to the skin. **b.** Learning curve showing time taken to insert the probe vs. patient number.

Fig. 2. Patient scans. Preoperative MRI, postoperative MRI and postoperative CT of a patient with cervical spinal cord injury. Arrow shows intradural pressure probe tip. The patient had C5 corpectomy, iliac bone graft, plate and screws.

Fig. 3. ISP recordings. a. ISP waveform showing peaks corresponding to (1) arterial pulsation, (2) intracranial compliance, and (3) aortic valve closure. **b.** Representative ISP from three patients who had acute TSCI.

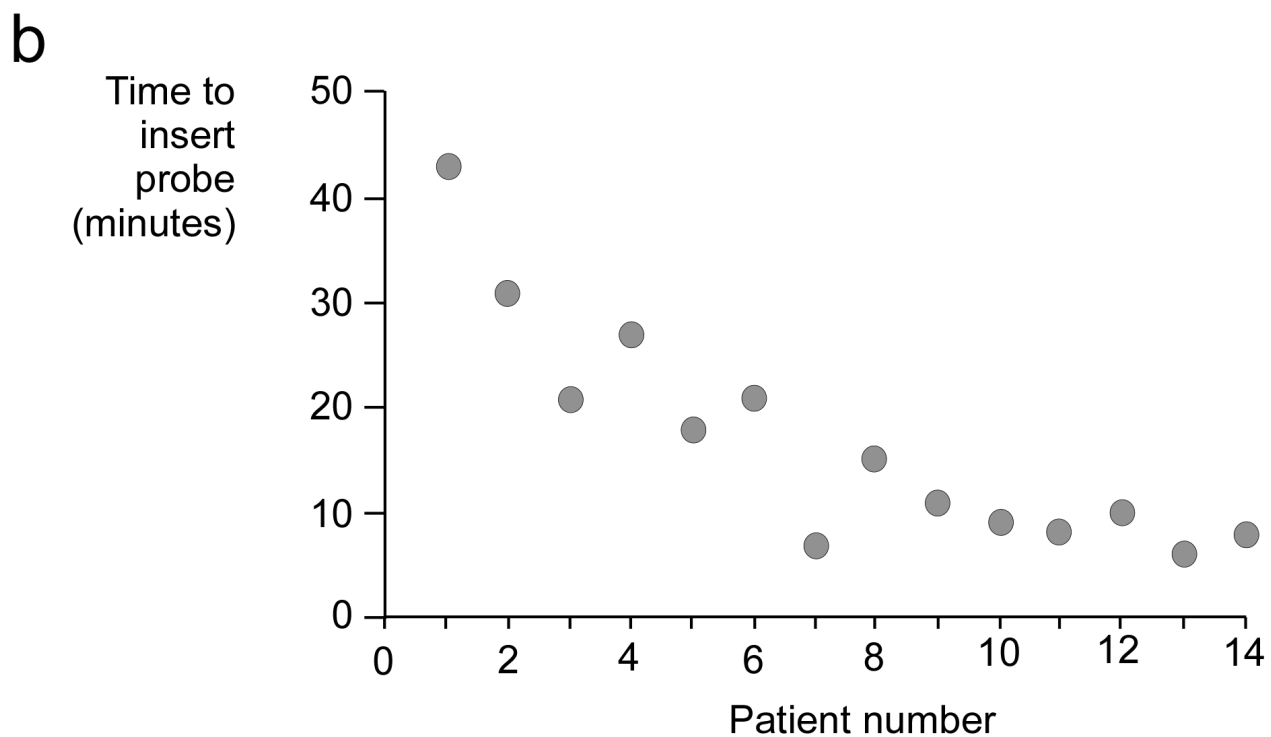
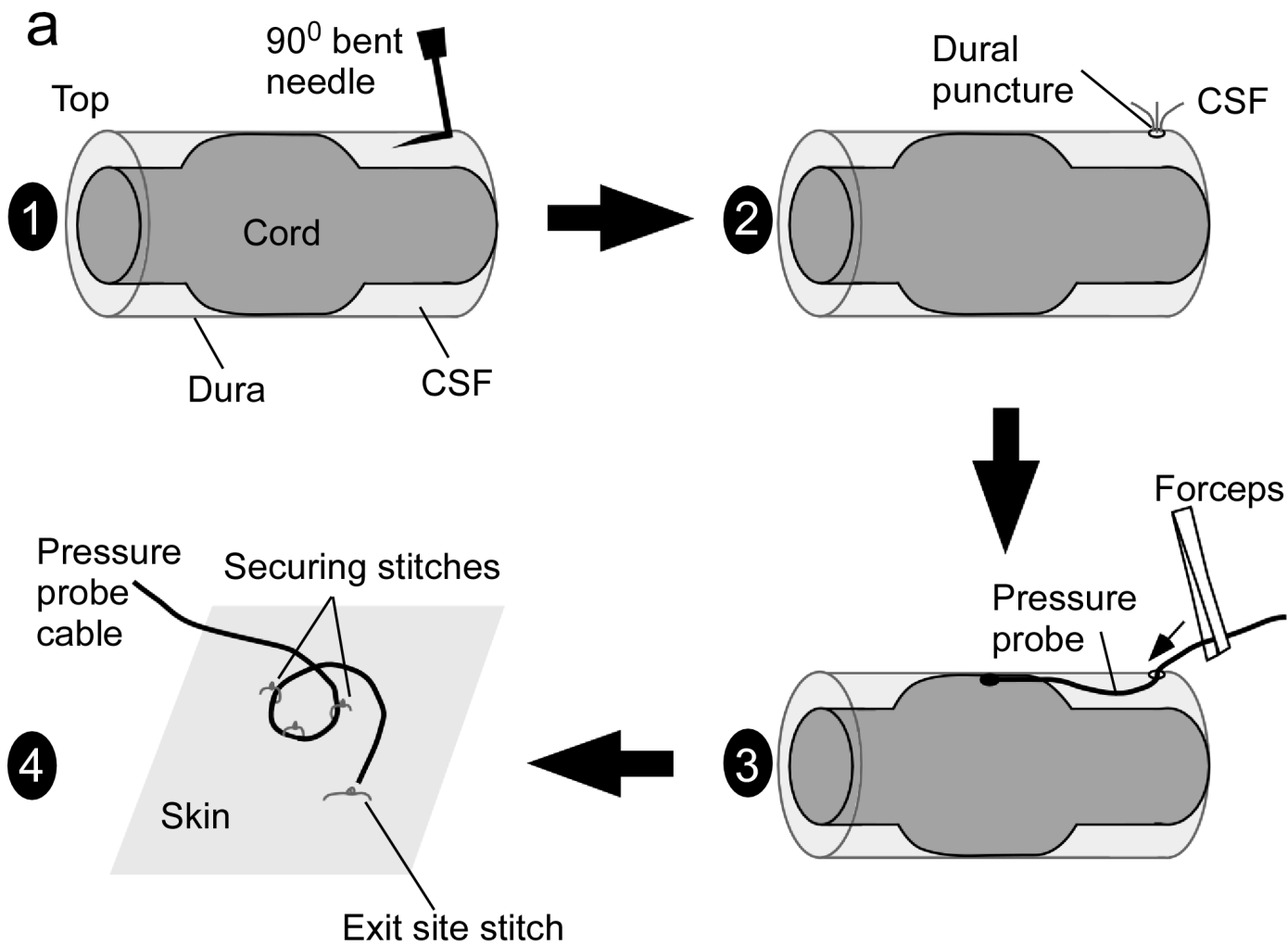


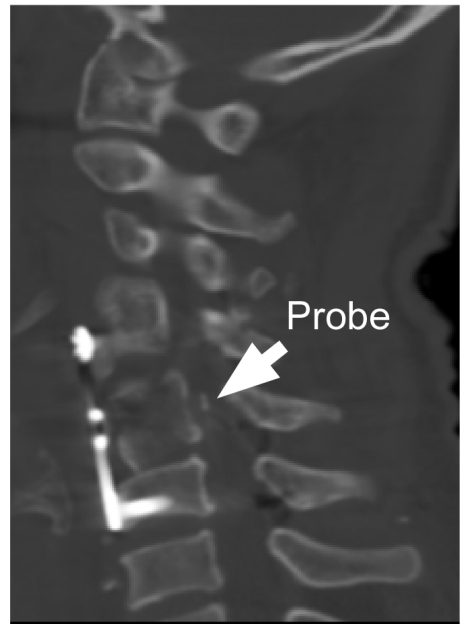
FIGURE 1



Preop MRI



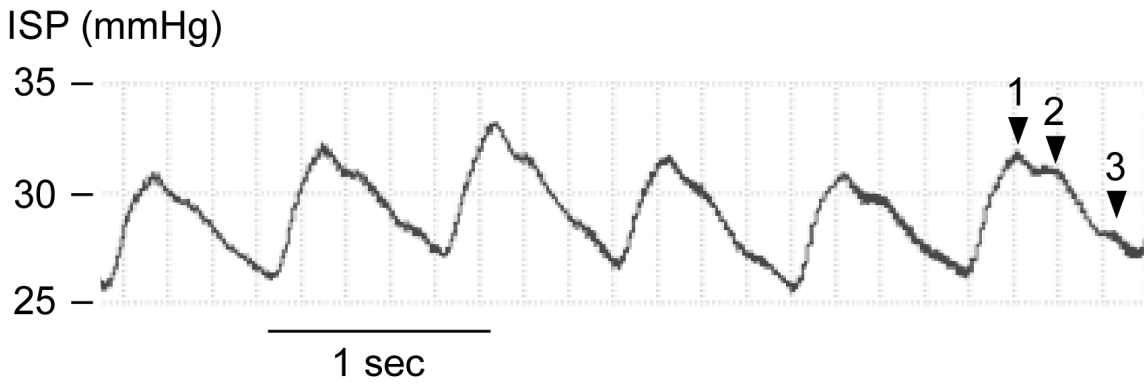
Postop MRI



Postop CT

FIGURE 2

a



b

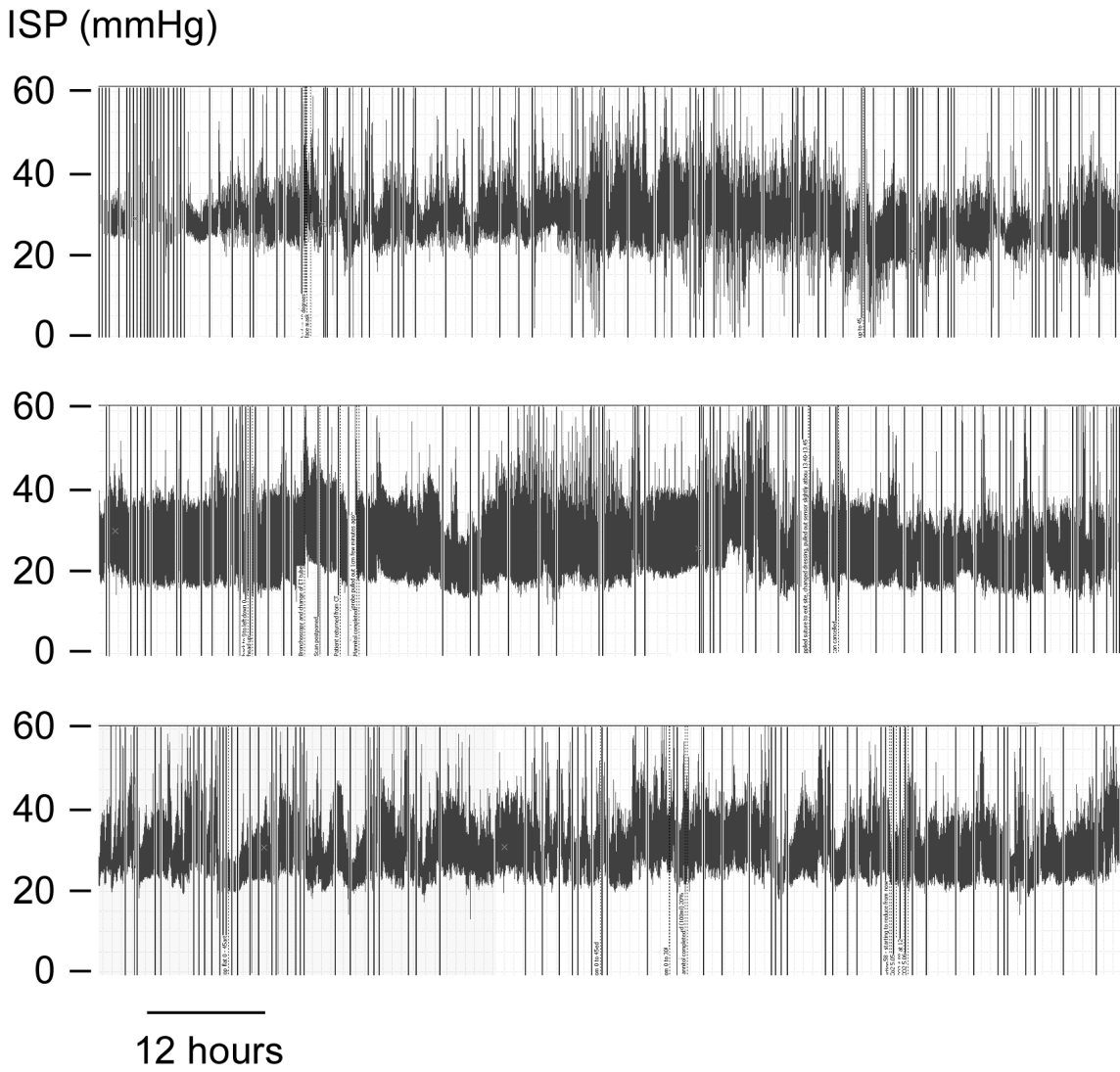


FIGURE 3