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CLINICAL ARTICLE

Acute, severe traumatic spinal cord injury: improving urinary bladder function by optimizing spinal cord perfusion

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OBJECTIVE The authors sought to investigate the effect of acute, severe traumatic spinal cord injury on the urinary bladder and the hypothesis that increasing the spinal cord perfusion pressure improves bladder function.

METHODS In 13 adults with traumatic spinal cord injury (American Spinal Injury Association Impairment Scale grades A–C), a pressure probe and a microdialysis catheter were placed intradurally at the injury site. We varied the spinal cord perfusion pressure and performed filling cystometry. Patients were followed up for 12 months on average.

RESULTS The 13 patients had 63 fill cycles; 38 cycles had unfavorable urodynamics, i.e., dangerously low compliance (< 20 mL/cmH_2O), detrusor overactivity, or dangerously high end-fill pressure (> 40 cmH_2O). Unfavorable urodynamics correlated with periods of injury site hypoperfusion (spinal cord perfusion pressure < 60 mm Hg), hyperperfusion (spinal cord perfusion pressure > 100 mm Hg), tissue glucose < 3 mM, and tissue lactate to pyruvate ratio > 30. Increasing spinal cord perfusion pressure from 67.0 ± 2.3 mm Hg (average ± SE) to 92.1 ± 3.0 mm Hg significantly reduced, from 534 to 365 mL, the median bladder volume at which the desire to void was first experienced. All patients with dangerously low average initial bladder compliance (< 20 mL/cmH_2O) maintained low compliance at follow-up, whereas all patients with high average initial bladder compliance (> 100 mL/cmH_2O) maintained high compliance at follow-up.

CONCLUSIONS We conclude that unfavorable urodynamics develop within days of traumatic spinal cord injury, thus challenging the prevailing notion that the detrusor is initially acontractile. Urodynamic studies performed acutely identify patients with dangerously low bladder compliance likely to benefit from early intervention. At this early stage, bladder function is dynamic and is influenced by fluctuations in the physiology and metabolism at the injury site; therefore, optimizing spinal cord perfusion is likely to improve urological outcome in patients with acute severe traumatic spinal cord injury.

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KEYWORDS blood pressure; cystometry; detrusor; microdialysis; spinal cord injury; urodynamics

The management of acute severe traumatic spinal cord injury (TSCI) in the intensive care unit (ICU) varies widely; e.g., American guidelines recommend a mean arterial pressure (MAP) of 85–90 mm Hg for a week after injury, whereas UK centers have no MAP targets.[1,2] To rationalize ICU management, we developed the practice of multimodality monitoring of the spinal cord injury site,[3] using safe techniques,[4] to individualize patient management.[5,6] We monitor intraspinal pressure (ISP), spinal cord perfusion pressure (SCPP),[5–8] and cord metabolism.[9–11] ISP and SCPP are clinically important parameters that correlate with injury site metabolism,[9–11] neurological status,[6,12,13] and long-term outcome[14] and are causally linked to limb power.[12]

Chronic TSCI often leads to unfavorable urodynamics defined as dangerously low bladder compliance (< 20 mL/

ABBREVIATIONS AIS = American Spinal Injury Association (ASIA) Impairment Scale; DO = detrusor overactivity; ICU = intensive care unit; ISCoPE = Injured Spinal Cord Pressure Evaluation; ISP = intraspinal pressure; LPR = lactate to pyruvate ratio; MAP = mean arterial pressure; MD = microdialysis; P_{abd} = abdominal (rectal) pressure; P_{ana} = anal sphincter pressure; P_{det} = detrusor pressure (P_{ves} − P_{abd}); P_{ves} = intravesical pressure; SCIM III = Spinal Cord Independence Measure version III; SCPP = spinal cord perfusion pressure; TSCI = traumatic spinal cord injury; V_{max} = volume drained from the bladder at end-fill; $\Delta V/\Delta P_{det}$ = bladder compliance.

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cmH_2O), detrusor overactivity (DO), and detrusor sphincter dyssynergia. As a result, detrusor pressure rises (> 40 cmH_2O) causing vesicoureteral reflux, hydronephrosis, and ultimately, renal failure.[15] Before urological surveillance became established, about 50% of TSCI patients died from renal failure.[16] Even now, renal failure remains the fourth leading cause of death after TSCI.[17] Improving bladder function is one of the top priorities according to TSCI patients, carers, and health experts.[18,19]

Unlike chronic TSCI, the effect of acute TSCI on bladder function is poorly understood. Current thinking is that early after TSCI, the detrusor becomes acontractile (spinal shock) and, within weeks, a reflex neurogenic bladder gradually develops.[20] In the present study, we performed filling cystometry in the first few days after TSCI while also monitoring the spinal cord injury site while the patient was in the ICU. We asked whether, during this acute phase, the detrusor is indeed acontractile or whether unfavorable urodynamics develop earlier than expected. We also asked whether bladder function could be improved by optimizing spinal cord perfusion.

Methods

Approvals

Urodynamic investigations were approved as a substudy of the Injured Spinal Cord Pressure Evaluation (ISCoPE) study. Approvals were obtained from the St. George's Joint Research Office and the National Research Ethics Service—Camberwell St. Giles Committee (No. 10/H0807/23). ISCoPE is registered at www.clinicaltrials.gov as NCT02721615. Informed consent was obtained from all participants.

Study Inclusion and Exclusion

We prospectively recruited consecutive TSCI patients enrolled into ISCoPE in the period June 2018–August 2019. Inclusion criteria were the following: 1) severe TSCI (American Spinal Injury Association [ASIA] Impairment Scale [AIS] grades A–C), 2) age 18–70 years, and 3) surgery performed within 72 hours of TSCI. Exclusion criteria were the following: 1) major comorbidities, 2) inability to obtain consent, and 3) penetrating TSCI.

Clinical Assessments and Imaging

Patients were admitted to the neurosurgical unit at St. George's Hospital and underwent AIS grade assessment by a trained neurosurgeon, which was repeated at discharge and at follow-up. Whole-spine CT and MRI studies were obtained before surgery; a CT was done within 48 hours after surgery and an MRI after probe removal. Patients were followed up in outpatient clinic with assessment of AIS grade, Spinal Cord Independence Measure version III (SCIM III), and SF-Qualiveen, a short-from quality of life questionnaire for urinary symptoms.

Spinal Surgery

Surgical decompression, including laminectomies and spinal instrumentation, were performed based on patient requirements and surgeon preference. Lateral mass screws (cervical spine) or pedicle screws (thoracolumbar spine) were inserted above and below the fracture and were linked with rods secured with blockers (Oasys or Xia 3, Stryker).

Probe Insertion

During posterior surgery, an ISP probe (Codman microsensor transducer, DePuy Synthes) and a microdialysis (MD) catheter (CMA61, CMA Microdialysis AB) were inserted through the skin into the wound cavity. Using a microscope, the dura plus arachnoid were opened one level below the injury. The ISP probe and MD catheter were inserted intradurally with the tips placed at the site of maximal cord swelling (Fig. 1A). The dural opening was sutured. These techniques are described elsewhere.[4,6,8,10–12,14,21] ISP and MD measured under the conditions of this study differ from intrathecal pressure and MD measured above or below the injury site because the injured cord is compressed against the dura, thus compartmentalizing the intrathecal space.[14,22–24]

ISP Setup

Postoperatively, patients were managed in the neurosurgical ICU. The ISP probe was connected to a Codman ICP box linked via an ML221 amplifier to a PowerLab running LabChart version 8 (AD Instruments). Blood pressure was recorded from a radial artery catheter connected to the Philips Intellivue MX800 bedside monitoring system (Philips), in turn connected to PowerLab. ISP and arterial blood pressure signals were sampled at 1 kHz and patients were monitored for up to 1 week. Data were analyzed using LabChart version 8 (AD Instruments) and ICM+ neuromonitoring software. SCPP was computed as MAP – ISP (Fig. 1A).

MD Setup

MD was started postoperatively in the neuro-ICU as described.[9–11,25] CNS perfusion fluid was perfused at 0.3 μL/min using the CMA106 pump (CMA Microdialysis AB). MD vials were changed hourly and analyzed using ISCUS Flex (CMA Microdialysis AB) for glucose, lactate, and pyruvate, and the lactate to pyruvate ratio (LPR) was computed. The first two samples from each patient were discarded to allow priming of the catheter and stabilization of metabolite concentrations; 100× changes in metabolite concentration, compared with the preceding hour, were excluded from analysis.

Urodynamics

Filling cystometry was performed in the first week postoperatively according to international guidelines[26] (Fig. 1B). Patients were investigated, where possible, sitting at 45° without sedation. The bladder was filled through a transurethral double-lumen catheter (Bard Medical) with 0.9% saline at 25 mL/min. Intravesical pressure (P_{ves}) was monitored through the other lumen by a water-perfused pressure monitor zeroed at the pubic symphysis. Abdominal (P_{abd}) and anal sphincter (P_{ana}) pressures were monitored using a 4-channel water-perfused anorectal manometer (Ardmore Healthcare), respectively, leveled at the pubic symphysis and anal verge. Pressure probes

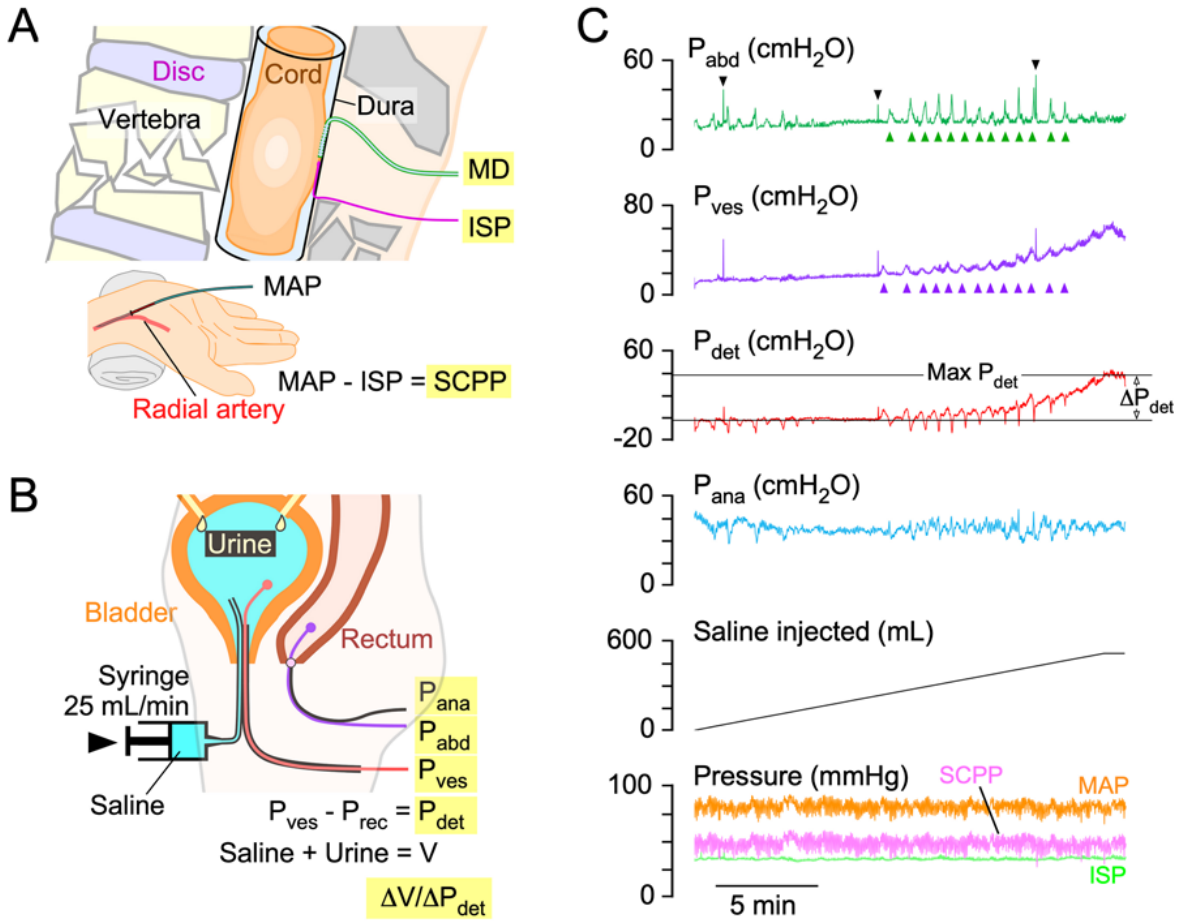

FIG. 1. Setup for injury site monitoring and cystometry. **A:** Injury site: ISP probe and MD catheter placed intradurally at the injury site (*top*); catheter in radial artery to monitor MAP (*bottom*); SCPP = MAP − ISP. **B:** Filling cystometry: normal saline is infused into the bladder at 25 mL/min up to 600 mL. Probes monitor intravesical (P_{ves}), abdominal (P_{abd}), and anal (P_{ana}) pressures. Detrusor pressure $P_{det} = P_{ves} - P_{abd}$. Bladder volume V = infused saline + urine produced. Bladder compliance is $\Delta V/\Delta P_{det}$. Key parameters are highlighted in *yellow*. **C:** Cystometry plot for patient 12 (T7, AIS grade A) at day 4 post-TSCI. $\Delta V/\Delta P_{det}$ = 12 mL/cmH_2O; at V_{max} (520 mL), P_{abd} = 19 cmH_2O, P_{ves} = 60 cmH_2O, and P_{det} = 39 cmH_2O. *Arrowheads*: *black* (cough), *purple* (bladder contractions), *green* (rectal contractions).

were connected to the Philips Intellivue MX800 monitor (Philips), in turn attached to PowerLab and sampled at 1 kHz. The bladder was filled to 600 mL or until detrusor pressure $P_{det} = P_{ves} - P_{abd}$ reached 40 cmH_2O. Patients were asked to report their first bladder sensation.

The volume drained from the bladder at end-fill (saline infused + urine produced) is V_{max}. Bladder compliance ($\Delta V/\Delta P_{det}$) is bladder volume change divided by change in detrusor pressure. P_{ana} and P_{abd} (rectal pressure) were respective surrogate markers of urethral and abdominal pressures. Detrusor overactivity (DO) is the transient increase in P_{det} during filling, quantified as the number, amplitude, and fill volume at the onset of phasic bladder contractions for each fill cycle.

Statistics

Urodynamic parameters versus injury site characteristics were fitted with linear, quadratic, or exponential regressions; compliance at presentation versus follow-up was fitted with a sigmoid curve. Adjusted coefficients of determination ($\hat{R}^2$) were computed (https://mycurvefit.com). We tested if drugs affect urodynamics (ANOVA) or bladder sensation using Fisher exact test on 2 × 3 contingency tables (http://vassarstats.net). Bladder volumes at first sensation were analyzed by Kaplan-Meier plots and log rank. Numbers of patients with/without sensation during bladder fill at presentation versus follow-up were compared by Fisher exact test on a 2 × 2 contingency table (http://vassarstats.net). Plots show average ± standard error. Significance was determined as $p < 0.05$.

Results

Patients

We recruited 13 patients aged 22.0–67.0 years (average 47.1 years), 12 (92.3%) males and 1 (7.7%) female (Supplement 1); 8/13 (61.5%) patients had cervical injuries, 4/13 (30.8%) thoracic, and 1/13 (7.7%) conus; 8/13 (61.5%) patients were assessed with AIS grade A injuries, 2/13 (15.4%) had AIS grade B, and 3/13 (23.1%) AIS grade C

impairment. All patients had posterior bony decompression with laminectomy and fusion; 2/13 (15.4%) also had anterior stabilization. Surgery was performed 14–70 hours after TSCI (average 41.9 hours).

Urodynamics

We performed 63 bladder fill cycles in the 13 patients at 2–7 days after surgery (Supplement 2). Unfavorable urodynamics occurred in 60.3% of cycles from 12 patients: bladder compliance < 20 mL/cmH_2O occurred in 19.0% of cycles, DO occurred in 55.6%, and end-fill storage pressure > 40 cmH_2O occurred in 15.9%. We infused 340–600 mL saline into the bladder, corresponding to V_{max} of 400–1300 mL. Patients received propofol during 57.1% of fill cycles, fentanyl during 63.4%, and norepinephrine during 95.2%. Fill cycles were performed over a wide range of SCPP (42.1–109.9 mm Hg), ISP (7.7–35.4 mm Hg), injury site tissue glucose (0.0–8.2 mM), and LPR (16.9–98.0). Figure 1C shows a typical set of urodynamics with DO and guarding reflex.

Complications

Adverse Events

One of 13 (7.7%) patients developed a sacral pressure ulcer, 1/13 (7.7%) had a penile meatal pressure ulcer from the indwelling catheter, 1/13 (7.7%) had microscopic hematuria, 5/13 (38.5%) had pneumonia, and 1/13 (7.7%) had pulmonary embolism. One of 13 (7.7%) patients had developed a syrinx shown on MRI scan at the 15-month follow-up.

Adverse Reactions

Seven of 13 (53.8%) patients had asymptomatic pseudomeningocele. There were no serious adverse events, no serious adverse reactions, and no suspected unexpected serious adverse reactions. No patients had cord damage or hematoma from the probes (MRI, neurological examination), CSF leak, wound infection, meningitis, urinary tract infection, or deterioration in renal function.

Variability of Urodynamic Measurements

Repeat fill cycles revealed variability in the unfavorable urodynamics. For example, in patient 3, increase in SCPP (from 59.7 to 80.7 mm Hg) reduced end-fill detrusor pressure (from 41.3 to 24.0 cmH_2O) and increased bladder compliance (from 12.0 to 28.6 mL/cmH_2O) (Fig. 2A). In patient 5, increase in SCPP (from 65.5 to 94.5 mm Hg) eliminated DO but had little effect on bladder compliance or end-fill detrusor pressure (Fig. 2B). Figure 2C summarizes the variabilities in bladder compliance, DO, and detrusor pressure at end-fill for each patient. We hypothesized that these fluctuations in urodynamics are caused by fluctuations in the physiology and metabolism of the injured cord. If our hypothesis holds, there should be strong correlations between urodynamic and injury site parameters.

Bladder Compliance Correlates With Injury Site Physiology and Metabolism

Figure 3 shows plots of the number of patients with dangerously low bladder compliance (< 20 mL/cmH_2O) versus average SCPP, ISP, tissue glucose, or tissue LPR (measured from the injured cord during cyctometry). The relation with SCPP is U-shaped with the optimum SCPP of approximately 80–90 mm Hg that eliminates dangerously low compliance. The percentage of patients with dangerously low bladder compliance was significantly higher during periods of low tissue glucose (< 3 mM) or ischemia (LPR > 30) at the injury site but did not significantly correlate with ISP. Thus, improved injury site perfusion and metabolism are associated with improved bladder compliance.

DO Correlates With Injury Site Physiology

Figure 4 shows how the severity of DO is related to the average SCPP, ISP, tissue glucose, and tissue LPR measured from the injured cord during each fill cycle. Phasic bladder contractions were more frequent, and their amplitudes were larger, during periods of spinal cord hypo- and hyperperfusion with the optimum SCPP, which minimizes DO, at approximately 75–85 mm Hg. Though the onset of DO occurred during hypo- and hyperperfusion at lower fill volumes than those for normal perfusion, the relations were not significant (not shown). The number of phasic bladder contractions did not significantly correlate with ISP. DO occurred more frequently during periods of low cord tissue glucose and high LPR, but the relations were not significant. There were also no significant correlations between the amplitudes of phasic bladder contractions or the fill volume at onset versus ISP, versus tissue glucose, or versus tissue LPR (not shown). These findings indicate that improved injury site perfusion is associated with less DO.

End-Fill Detrusor Pressure Correlates With Injury Site Physiology and Metabolism

Figure 5 shows plots of the percentage of patients with dangerously high end-fill detrusor pressure (> 40 cmH_2O) versus average SCPP, ISP, tissue glucose, and LPR measured from the injured cord during each fill cycle. The relation with SCPP is U-shaped with the optimum SCPP of approximately 80–90 mm Hg that eliminates dangerously high end-fill detrusor pressure. The percentage of patients with dangerously high end-fill detrusor pressure was significantly higher during periods of spinal cord swelling (ISP > 10 mm Hg), periods of low tissue glucose (< 4 mM), and periods of ischemia (LPR > 30) at the injury site. Thus, improved injury site physiological and metabolic parameters are associated with lower detrusor pressure at end-fill.

Bladder Volume at First Desire to Void Correlates With Spinal Cord Perfusion

Bladder sensation was assessable in 10 patients; the other 3 were sedated to aid ventilation. Of the 10 assessed patients, only 4 had bladder sensation; 3 of these patients had AIS grade C on admission and 1 had AIS grade A on admission who was AIS grade C at follow-up. These 4 patients had 14 fill cycles; for each patient, fill cycles were classed as high or low SCPP, with at least a 20–mm Hg difference between them, resulting in 8 high (82.6–100.5

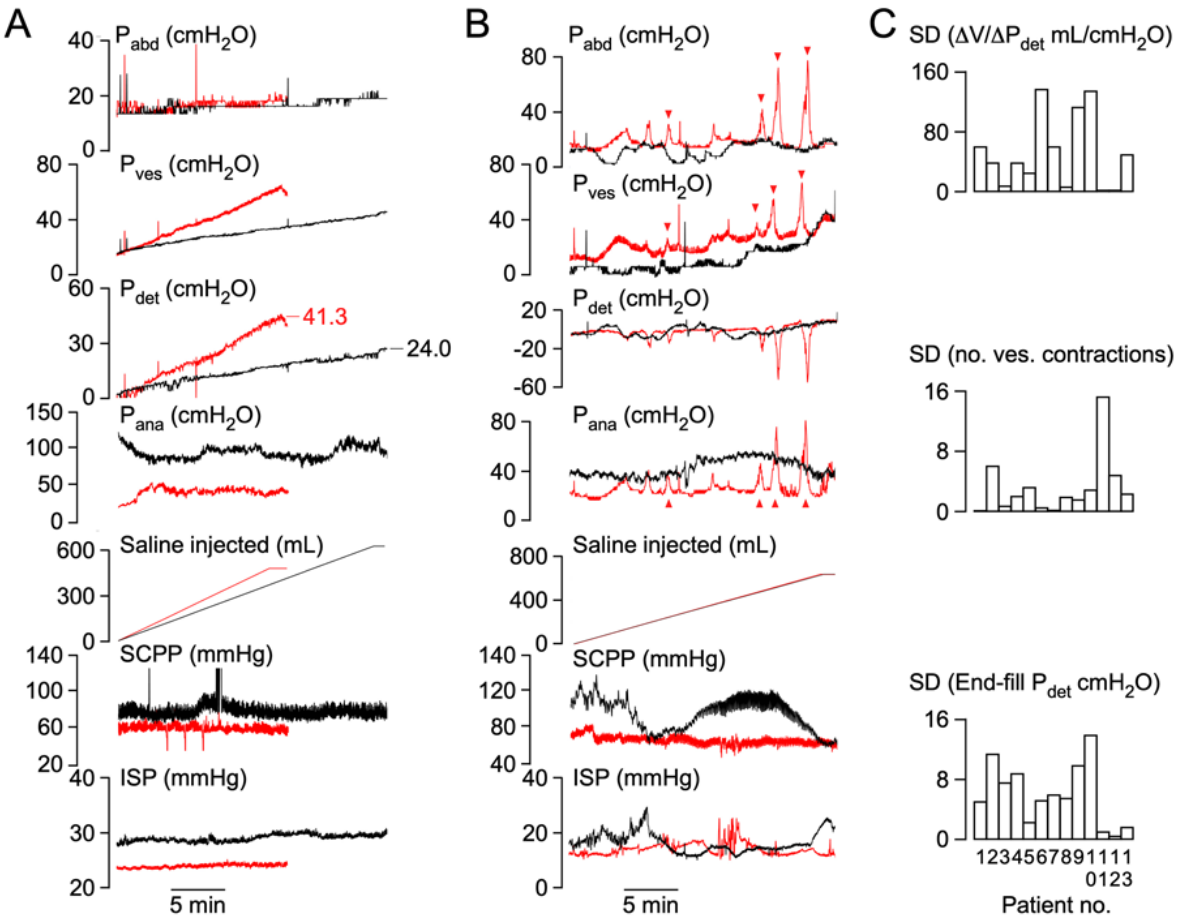

FIG. 2. Variability of urodynamic parameters in each patient. **A:** Cystometry plot for patient 3 (T8, AIS grade A). Day 4 post-TSCI (*black*): SCPP 80.7 mm Hg, $\Delta V/\Delta P_{det}$ 28.6 mL/cmH_2O, end-fill P_{det} 24.0 cmH_2O. Day 2 post-TSCI (*red*): SCPP 59.7 mm Hg, $\Delta V/\Delta P_{det}$ 12.0 mL/cmH_2O, end-fill P_{det} 41.3 cmH_2O. **B:** Cystometry plot for patient 5 (C4 AIS grade A). Day 4 post-TSCI (*black*): SCPP 94.5 mm Hg, $\Delta V/\Delta P_{det}$ 48.0 mL/cmH_2O, end-fill P_{det} 8.8 cmH_2O. Day 3 post-TSCI (*red*): SCPP 65.5 mm Hg, $\Delta V/\Delta P_{det}$ 42.0 mL/cmH_2O, end-fill P_{det} 9.1 cmH_2O, vesicular contractions associated with anal-rectal contractions (guarding reflex, arrows) at 245, 490, 524, 599 mL. **C:** SD of bladder compliance ($\Delta V/\Delta P_{det}$), number of vesicular contractions (DO), and detrusor pressure at end-fill (end-fill P_{det}) for patients 1–13.

mm Hg) and 6 low (56.2–74.1 mm Hg) SCPP fill cycles. The median bladder volume at which sensation was first experienced was significantly smaller for fill cycles at high versus low SCPP (365 vs 534 mL) (Fig. 6). Thus, improved spinal cord perfusion is associated with improved bladder sensation.

No Significant Effect of Sedation, Inotropes, Day After Injury, or Spinal Cord Level on Urodynamics

Bladder compliance, number of phasic bladder contractions per fill, end-fill detrusor pressure, and the probability of experiencing bladder sensation during a fill cycle were not significantly influenced by propofol, fentanyl, norepinephrine (Supplement 3), day after TSCI (Supplement 4), or cervical versus thoracic injury level (Supplement 5).

Long-Term Outcome

Average follow-up was 12 months (range 2–25 months) (Supplement 1). Bladder compliance assessed acutely correlated with compliance assessed at follow-up (Fig. 7). All patients with bladder sensation at presentation were sensate at follow-up, but with decrease in bladder volume causing first sensation to void from 442 ± 55 to 260 ± 31 mL (average ± SE). No one with absent sensation at presentation recovered the sensation at follow-up. DO at presentation did not correlate with DO at follow-up (not shown). At follow-up, in 7/13 patients (53.8%) AIS grade improved by 1 or more, 5/13 (38.5%) patients remained the same, and 1/13 (7.7%) deteriorated by 1 AIS grade, comparable to earlier findings.[4] At follow-up, SCIM III ranged from 0 to 15 and SF-Qualiveen from 1.13 to 4.00 (Supplement 6).

Discussion

We showed that > 90% of patients develop periods of unfavorable urodynamics within a week of TSCI, including dangerously low bladder compliance, dangerously high detrusor pressure at end-fill, and DO. Unfavorable urodynamics fluctuate; they become more pronounced when the injured cord is hypo- or hyperperfused, gluco-

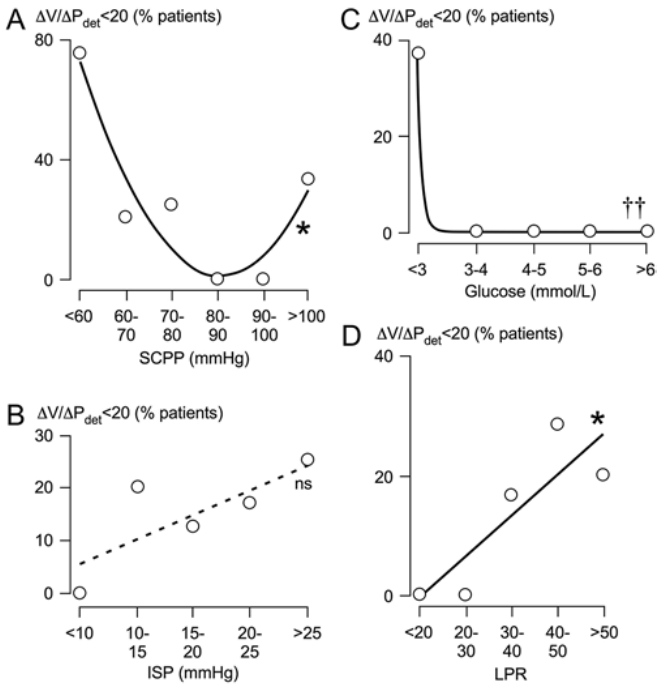

FIG. 3. Bladder compliance correlates with injury site physiology and metabolism. Percentage of patients with $\Delta V/\Delta P_{det}$ < 20 cmH_2O versus SCPP (best-fit quadratic, $\hat{R}^2 = 0.78$) (**A**), ISP (best-fit line, $\hat{R}^2 = 0.48$) (**B**), tissue glucose (best-fit exponential, $\hat{R}^2 = 1.00$) (**C**), and tissue LPR (best-fit line, $\hat{R}^2 = 0.86$) (**D**). *p < 0.05, ††0.00005. ns = not significant. Average ± SE.

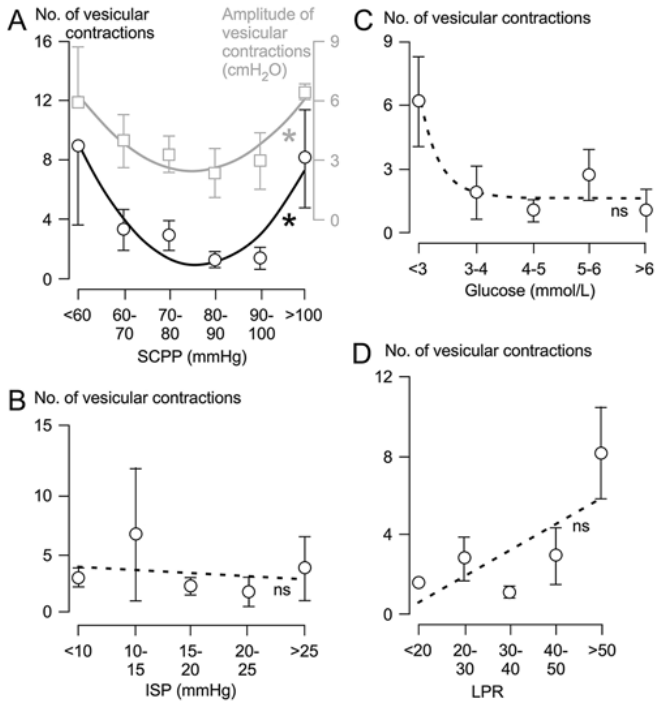

FIG. 4. DO correlates with injury site physiology. Number and amplitude of vesicular contractions versus SCPP (best-fit quadratics, $\hat{R}^2 = 0.79$ for number, $\hat{R}^2 = 0.84$ for amplitude) (**A**), ISP (best-fit straight line, $\hat{R}^2 = -0.26$) (**B**), tissue glucose (best-fit exponential decay, $\hat{R}^2 = 0.73$) (**C**), and tissue LPR (best-fit straight line, $\hat{R}^2 = 0.42$) (**D**). *p < 0.05. ns = not significant. Average ± SE.

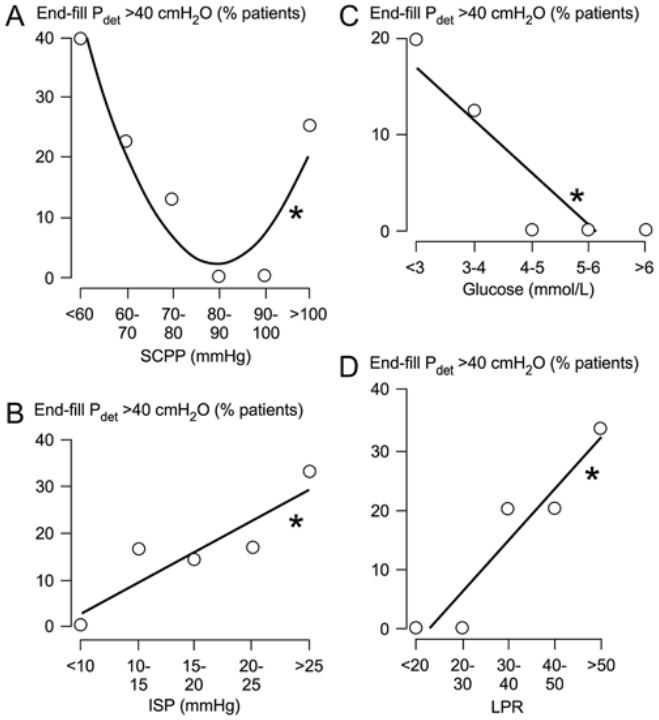

FIG. 5. End-fill detrusor pressure correlates with injury site physiology and metabolism. Percentage of patients with end-fill detrusor pressure > 40 cmH_2O versus SCPP (quadratic regression, $\hat{R}^2 = 0.83$) (**A**), ISP (best-fit straight line, $\hat{R}^2 = 0.72$) (**B**), tissue glucose (best-fit straight line, $\hat{R}^2 = 0.73$) (**C**), and tissue LPR (best-fit straight line, $\hat{R}^2 = 0.87$) (**D**). *p < 0.05.

penic, or ischemic (high tissue LPR). By intervening to eliminate hypoperfusion, we could improve bladder sensation with first desire to void occurring at smaller volume.

Our results challenge the prevailing dogma that the detrusor is acontractile early after TSCI with late development of overreactive bladder and agree with the few studies reporting unfavorable urodynamics in most patients within 40 days of TSCI;[27–29] in our study, unfavorable urodynamics were detectable as soon as 48 hours after TSCI. Initially, after severe TSCI, limbs are hypotonic/hyporeflexic, later progressing to spasticity/hyperreflexia.[20] It is assumed that the detrusor behaves similarly (initially acontractile, then overactive). Our data indicate discordance in the early responses to TSCI of limb muscles versus the detrusor.

Variability in the urodynamic parameters and their dependence on events at the injury site suggest that interventions to improve injury site physiology and metabolism will likely improve bladder function. For example, intervention to increase SCPP (MAP – ISP) may be achieved using inotropes to increase MAP[6] or expansion duroplasty to reduce ISP.[30] Increasing SCPP may increase cord tissue glucose and reduce cord LPR,[12] which may improve neuronal activity at the injury site, thus leading to improved bladder function. In our study, transiently increasing the SCPP reduced the threshold volume for bladder sensation. The finding that SCPP ≥ 100 mm Hg worsens bladder function supports the idea that not only hypoperfusion, but also hyperperfusion, is detrimental for the injured cord; we urge caution, however, because this statement relies on

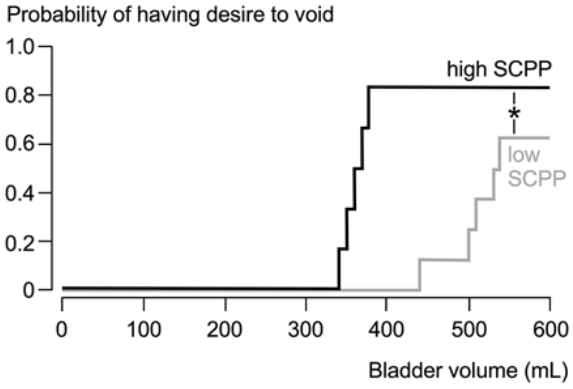

FIG. 6. Bladder volume at first desire to void correlates with spinal cord perfusion. Probability of experiencing desire to void versus bladder volume at high (92.1 ± 3.0 mm Hg) versus low (67.0 ± 2.3 mm Hg) SCPP. *p < 0.05.

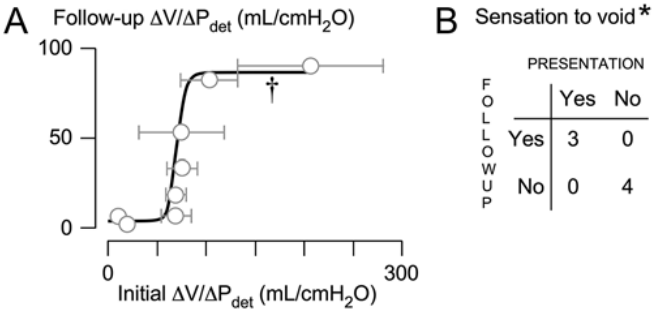

FIG. 7. Long-term outcome. **A:** Bladder compliance at initial presentation versus follow-up with best-fit sigmoid curve, $\hat{R}^2 = 0.90$. Average ± SE. **B:** Number of patients with and without sensation to void during bladder fill at presentation versus follow-up. *p < 0.05, †0.001.

only a single point on each graph (Figs. 3A, 4A, and 5A). Our earlier work, using limb power and sensory level as outcomes,[6,12–14] and the current study of bladder function suggest that SCPP is the key parameter to monitor and optimize after acute TSCI.

Some aspects of bladder function, assessed acutely, correlated with corresponding measurements at follow-up. The relation between average bladder compliance at presentation versus follow-up was sigmoidal; i.e., patients with low initial compliance (< 20 mL/cmH_2O) had low compliance at follow-up and patients with intermediate (50–100 mL/cmH_2O) compliance initially had low or normal compliance at follow-up, whereas patients with high initial compliance (> 100 mL/cmH_2O) also had high compliance at follow-up. These findings suggest that patients with intermediate bladder compliance may be the most sensitive to SCPP management. Improving cord perfusion did not influence the presence or absence of the sensation to void but reduced the bladder volume at which the first sensation occurred. Compliance is the most important urodynamic parameter affecting renal function. Thus, early identification of the TSCI patients with dangerously low bladder compliance would allow for more aggressive intervention before renal function has deteriorated. Based on our findings, we recommend that any early urodynamic testing is done once the SCPP has been optimized.

This study has limitations. First, though best urodynamic practice recommends monitoring awake/interactive patients, some patients were sedated because they required ventilation. Second, some patients received norepinephrine, raising the possibility of a direct inhibitory effect of norepinephrine on bladder contraction potentially masking DO and reducing compliance. Our analysis suggests that propofol, fentanyl, and norepinephrine did not significantly influence the urodynamics; however, there may be unknown confounding factors that we did not explore. Third, because of the acute ICU setting, we were unable to perform flow or videourodynamic monitoring. Fourth, the number of patients in our study is small, due to the difficulty in performing multiple urodynamic assessments in acutely injured patients in ICU and due to the relative infrequency of TSCI. Fifth, injuries varied in spinal level (cervical vs thoracic) and severity; our study was too small to assess differences between these groups. Sixth, while all urodynamic tests were performed within 8 days of injury, there was variability in the timing of the studies, which related to each patient's clinical progress and suitability for testing. Our analysis suggests that day from TSCI did not significantly influence the urodynamic parameters. Seventh, it is impossible to conclude that intervening to improve spinal cord perfusion improves long-term outcome; this would require a randomized controlled trial. Importantly, no patient experienced complications directly related to urodynamic testing.

Conclusions

Filling cystometry, performed within days of TSCI, identifies early patients with unfavorable urodynamics. Our findings suggest that individualized therapy in acute TSCI, by monitoring and optimizing cord physiology and metabolism, will likely improve not only limb motor function and sensation, but also urinary function.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Conception and design: Saadoun, Solomon, Papadopoulos. Acquisition of data: Hogg, Kearney, Gallagher, Zoumprouli, Papadopoulos. Analysis and interpretation of data: Saadoun, Hogg, Solomon, Zoumprouli, Papadopoulos. Drafting the article: Saadoun, Hogg, Solomon. Critically revising the article: Saadoun, Papadopoulos. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Saadoun. Statistical analysis: Saadoun. Administrative/technical/material support: Saadoun. Study supervision: Saadoun.

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