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Spinal cord perfusion pressure correlates with anal sphincter function in a cohort of patients with acute, severe traumatic spinal cord injuries

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DETAILS PAGE

We confirm that:

1) The manuscript complies with all instructions to authors.
2) Authorship requirements have been met and the final manuscript was approved by all authors
3) This manuscript has not been published elsewhere and is not under consideration by another journal
4) We have adhered to ethical guidelines and indicate ethical approvals (IRB) and use of informed consent, as appropriate.

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We confirm use of the STROBE reporting checklist for cohort studies.

ABBREVIATIONS

ABP, arterial blood pressure

AP, anal pressure

ARM, anorectal manometry

ISCoPE, injured spinal cord pressure evaluation

ISP, intra cranial pressure

MAP, mean arterial pressure

NBD, neurogenic bowel dysfunction

NICU, neuro-intensive care unit

RAIR, recto-anal inhibitory reflex

SCIM, spinal cord independence measure

SCPP, spinal cord perfusion pressure

ABSTRACT

Background/Objective. Acute, severe traumatic spinal cord injury often causes faecal incontinence. Currently, there are no treatments to improve anal function after traumatic spinal cord injury. Our study aims to determine whether, after traumatic spinal cord injury, anal function can be improved by interventions in the neuro-intensive care unit to alter the spinal cord perfusion pressure at the injury site.

Methods. We recruited a cohort of patients with acute, severe traumatic spinal cord injuries, American Spinal Injury Association Impairment Scale grades A – C. They underwent surgical fixation within 72 hours of the injury and insertion of an intrathecal pressure probe at the injury site to monitor intraspinal pressure and compute spinal cord perfusion pressure as mean arterial pressure minus intraspinal pressure. Injury site monitoring was performed at the neuro-intensive care unit for up to a week after injury. During monitoring, anorectal manometry was also carried out over a range of spinal cord perfusion pressures.

Results. Data were collected from 14 consecutive traumatic spinal cord injury patients aged 22 – 67 years. Mean resting anal pressure was 44 cmH_2O, which is considerably lower than the average for healthy patients previously reported at 99 cmH_2O. Mean resting anal pressure vs. spinal cord perfusion pressure had an inverted U-shaped relation ($\hat{R}^2 = 0.82$) with highest resting anal pressures at spinal cord perfusion pressure of ~100 mmHg. The recto-anal inhibitory reflex (transient relaxation of the internal anal sphincter during rectal distension), which is important for maintaining faecal continence, was present in 90 % attempts at high (90 mmHg) spinal cord perfusion pressure vs. 70 % attempts at low (60 mmHg) spinal cord perfusion pressure ($P < 0.05$). During cough, the rise in anal pressure from baseline was 51 cmH_2O at high (86 mmHg) spinal cord perfusion pressure vs. 37 cmH_2O at low (62 mmHg) spinal cord perfusion pressure ($P < 0.0001$). During anal squeeze, higher spinal cord perfusion pressure was associated with longer endurance time and spinal cord perfusion

pressure 70 – 90 mmHg with stronger squeeze. There were no complications associated with anorectal manometry.

Conclusions. Our data indicate that spinal cord injury causes severe disruption of anal sphincter function. Several key components of anal continence (resting anal pressure, recto-anal inhibitory reflex, anal pressure during cough and during squeeze) markedly improve at higher spinal cord perfusion pressure. Maintaining too high spinal cord perfusion pressure may worsen anal continence.

INTRODUCTION

Traumatic spinal cord injury (TSCI) is a devastating event resulting in life-long disability including limb weakness, bladder and bowel incontinence, sexual dysfunction, autonomic dysfunction and, in cervical trauma, impaired breathing. TSCI affects ~180,000 people globally each year.[1] A major problem after TSCI is anal sphincter dysfunction, from impaired control of the voluntary sphincter, impaired control of pelvic floor muscles and impaired autonomic activity. Bowel function is ranked on par with limb weakness by TSCI patients when prioritising recovery goals.[2] Bowel dysfunction affects about 80 % of TSCI patients and is often ranked as more problematic than urinary incontinence or sexual dysfunction.[3] Faecal incontinence is common and often unpredictable. Constipation, diarrhoea, and nausea are also commonly encountered, and most patients take drugs and require manual stimulation to defaecate.[4] Such bowel regimens may consume considerable time.

Anal function may be assessed with anorectal manometry (ARM), which measures anal pressure (AP), rectal sensation and the neural reflexes needed for normal defaecation.[5] ARM allows the study of the sphincter when straining, at rest and when squeezing to identify incontinence issues linked to the inability of the muscles to contract correctly and difficulties in defecating due to the incomplete opening and release of the sphincter muscles.

To improve the management of TSCI patients in the neuro-intensive care unit (NICU), our group has developed monitoring from the injury site.[6-9] We monitor intra spinal pressure (ISP) and calculate spinal cord perfusion pressure (SCPP) as mean arterial pressure (MAP) minus ISP. Monitoring is safe[10] and analogous to intra cranial pressure and cerebral perfusion pressure monitoring for brain injury.[11] ISP and SCPP are clinically important parameters that correlate with injury site metabolism,[7,12] neurological status,[6,13] urinary function[14] and long-term outcome.[15]

Our objective was to test the effect of TSCI on anal function in the acute setting. We performed ARM whilst also monitoring ISP and SCPP in the first 10 days after TSCI. We hypothesised that AP, change in AP (ΔAP) during squeeze or cough, the recto-anal inhibitory reflex (RAIR) and rectal sensation are all influenced by ISP and SCPP. We also hypothesised that interventions to normalise these parameters improve anal sphincter function.

MATERIALS AND METHODS

Institutional Research Board Approvals. Approvals for the Injured Spinal Cord Pressure Evaluation (ISCoPE) study including the consent form and patient information sheet were obtained by the St Georges Joint Research Office and the National Research Ethic Service – Camberwell St Giles Committee (No 10/H0807/23). ISCoPE is registered at www.clinical trials.gov as NCT02721615.

Inclusion and Exclusion Criteria. We recruited consecutive TSCI patients who were enrolled into the ISCoPE trial in the period June 2018 – August 2019. Inclusion criteria for ISCoPE are severe TSCI defined as American Spinal Injury Association Impairment Scale (AIS) grades A – C, age 18 – 70 years and surgery performed within 72 hours of TSCI. Exclusion criteria are major co-morbidities, inability to obtain consent and penetrating TSCI.

Clinical examination and imaging. All patients were admitted to the neurosurgical unit at St. George's Hospital and underwent International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) AIS assessment by a neurosurgical resident trained in AIS which was repeated at discharge and in the follow up outpatient clinic. All patients had CT and MRI of the spine imaging before surgery and within four weeks of surgery.

Bowel function at follow-up. In clinic, functional and quality of life measures were completed using two standard scales, the Spinal Cord Independence Measure III (SCIM) bowel score[16] and Neurogenic Bowel Dysfunction Score (NBD).[17] The SCIM III bowel score is 0, Irregular timing or very low frequency (less than once in 3 days) of bowel movements; 5, Regular timing, but requires assistance (e.g. for applying suppositories) – rare accidents (less than twice a month); 8, Regular bowel movements, without assistance – rare accidents (less than twice a month); 10, Regular bowel movements, without assistance – no accidents. In the NBD score, bowel dysfunction is classed as 0 – 6, very minor; 7 – 9; minor; 10 – 13, moderate; 14+, severe.

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, using Oasys or Xia 3 (Stryker, Newbury, UK). Postoperatively, all patients were managed in the NICU.

Probe insertion. During posterior surgery, a pressure probe (Codman Microsensor Transducer®, Depuy Synthes, Leeds, UK) was inserted through the skin into the wound cavity. Using a microscope, the dura and arachnoid were opened one level below the injury. The pressure probe was inserted intradurally with the tip placed at the site of maximal cord swelling. The dural opening was sutured. This technique is described in detail elsewhere.[6,9,10,12,13,15,18,19] ISP measured this way differs from intrathecal pressure measured above or below the injury site because the injured cord is compressed against the dura thus compartmentalising the intrathecal space.[15,20-22]

ISP and arterial blood pressure (ABP) monitoring. The ISP probe was connected to a Codman ICP box linked via a ML221 amplifier to a PowerLab running LabChart v.8 (AD Instruments, Oxford, UK). Blood pressure was recorded from a radial artery catheter connected to the Philips Intellivue MX800 bedside monitoring system (Philips, Guildford, UK) and in turn connected to the PowerLab system. ISP and arterial blood pressure signals were sampled at 1 kHz and patients were monitored for up to 7 days. Fig 1 shows the set up. Data was analysed using Labchart v.8 (AD Instruments, Oxford, UK) and ICM+ (www.neurosurg.cam.ac.uk/icmplus). ISP and ABP was used to compute SCPP = MAP – ISP.

NICU Clinical management. Patients were transferred to the NICU following surgery and remained there for the duration of spinal cord monitoring and clinical need. Patients were nursed on pressure reliving mattresses, on their side where possible to take pressure off the laminectomy site. Antibiotic prophylaxis was given for 48 hours after surgery with vancomycin and gentamicin, which is the standard regimen in our hospital for instrumented spinal fusion. Subcutaneous prophylactic low molecular weight heparin was started at 24 hours post-surgery (held for 12 hours pre and post probe removal). Mechanical (compression stockings, intermittent calf compression) thromboprophylaxis was used throughout. Ventilatory support was managed by the NICU team. MAP targets were at the discretion of the NICU (unrelated to ISP or SCPP) except when the SCPP was altered to produce "low" and "high" SCPP for ARM measurements. Ionotropic support was with intravenous noradrenaline through a central venous line or, whilst central access was being established, with intravenous metaraminol via a peripheral venous line. All patients were reviewed and examined daily by the research team and any complications or concerns recorded and actioned immediately. As per NICU protocols, daily blood tests were performed including

full blood count, renal and liver profile, cardiac enzymes, C-reactive protein and clotting function.

Anorectal manometry. All patients underwent ARM in the NICU over a wide range of SCPPs in the week following surgery. The duration of ARM related to how long the patients were able to tolerate the intervention and whether the clinical condition of the patient was appropriate for this testing. AP was sampled at 1 kHz with a 4-channel water perfused manometer (Ardmore, Middleton-on-Sea, U.K.) levelled at the anal verge (Fig. 1). The pressure monitor was connected to the Philips Intellivue MX800 bedside monitoring system (Philips, Guildford, UK) in turn connected to the PowerLab system. AP was monitored at rest, during cough, during squeeze and during the recto-anal inhibitory reflex (RAIR) and balloon threshold testing. Resting and maximal AP was taken as the mean of the 4 channels, excluding rectal recordings. During cough, we noted the maximum AP and the increment from resting AP (ΔAP). During prolonged (10 s) anal squeeze, we noted the endurance time (duration AP >50 % of maximum) and the increase in AP from resting pressure (ΔAP). The RAIR was performed by rapid inflation to 50 mL and deflation of rectal balloon and was assessed for baseline AP, latency, maximal amplitude, amplitude reduction, recovery time and duration. Rectal sensation threshold tests were estimated by filling the rectal balloon at 50 mL increments (to maximum of 500 mL) and asking the patient to comment on first sensation, first desire to defaecate and urgency to defaecate. For cough, squeeze and RAIR, repeat assessments were performed in each patient at low and at high SCPP.

Variables assessed and bias. We investigated the relation between baseline AP *vs.* SCPP, the characteristics of the RAIR at low *vs.* high SCPP, the effect of cough on AP at low *vs.* high SCPP and the effect of voluntary anal squeeze on AP at low vs. high SCPP. For baseline

AP, we used data from the entire monitoring period at 1 Hz. The average hours of monitoring for each patient were 1.9 (range 0.4 – 12). To assess RAIR, cough and rectal sensation (elicited by inflating a rectal balloon), SCPP was manipulated using noradrenaline. Each patient had a period when the dose of noradrenaline was reduced and a period when the noradrenaline was increased. Each figure highlights the average SCPP for each patient at their "low" and "high" SCPP period of monitoring. We also asked the patients to strain at different SCPPs. To minimise bias, the ARM data were obtained without knowledge of the SCPP.

Statistics. Plots of ARM parameters (resting AP, prolonged squeeze endurance time, prolonged squeeze change in AP (ΔAP)) *vs.* SCPP were fitted with best-fit quadratic or linear regression curve, chosen to minimize the Akaike Information Criterion. Adjusted coefficients of determination ($\hat{R}^2$) were computed using https://mycurvefit.com with $\hat{R}^2$ >0.5 considered strong correlation. The relation between mean values of components of RAIR (baseline AP, recovery time, % amplitude reduction) and cough (maximum AP, mean change in AP) at high and low SCPP were compared using Student's t-test. The correlation between AIS grade or SCPP and anal sphincter outcome was assessed using Kendall's tau B. Analysis was done using XLStat Biomed v2018.1 for Mac (Addinsoft, Paris, France). Statistical significance was taken as $P < 0.05$.

RESULTS

Patient demographics. Data were obtained from 14 patients aged 22 – 67 years (mean 47.4), 13 male and 1 female. Most (64.3 %) patients had complete (AIS A) injuries and most (64.3 %) had cervical injuries. All patients had posterior surgical bony decompression including laminectomy and fusion with 21.4 % also requiring anterior stabilisation. Mean follow up

was 8.1 months (range 2 – 19). At follow-up, most (71.4 %) patients had improved by one of more AIS grades, 21.4 % remained the same and 7.2 % deteriorated by one AIS grade. These demographics are summarised in Table 1.

Anorectal manometry. We monitored resting AP for an average of 1.9 h per patient (range 0.4 – 12) over an average of 2.1 d per patient (range 1 – 4). Per patient, the average number of coughs was 11.8 (range 1 – 36), the average number of voluntary squeezes was 4.9 (range 0 – 13) and the average number of RAIRs was 10.9 (range 0 – 19). In some patients, the cough and squeeze tests could not be performed because of sedation used for respiratory support. At follow up the NBD score classified 21.4 % patients as having minor, 28.6 % moderate and 50.0 % severe bowel problems. For the SCIM III bowel scores, 7.1 % patients scored 0, 50.0 % scored 5, 28.6 % scored 8 and 14.3 % scored 10. Details are in Table 2.

Complications. There were no complications associated with ARM. 7 patients had asymptomatic pseudomeningoceles on post-operative MRI scan, 5 patients were treated for chest sepsis, 1 patient had a pulmonary embolism and 1 developed a sacral pressure sore.

Resting anal pressure. Altogether, we collected 26.3 h of resting AP data, i.e. 1.9 +/- 0.8 h/patient. Average resting AP was 44.3 cmH_20, considerably lower than the average for healthy male patients previously reported at 99 cmH_20.[23] For most patients, higher SCPP was associated with higher resting AP but, when the data were grouped, mean AP vs. SCPP showed an inverted U-shaped relation ($\hat{R}^2 = 0.82$) (Fig. 2). Maximal AP showed a similar correlation with SCPP as mean AP (Suppl. Fig 1). In the 7 patients who had intra-operative monitoring, there was no significant change in resting AP pre- vs. post- bony spinal cord decompression (Suppl. Fig. 2). There was no correlation between average or maximum

resting AP and ISP (Suppl. Figs. 3, 4). Also, there was no correlation between the dose of noradrenaline and the average AP, thus making it unlikely that the relation between SCPP and AP is due to a direct effect of noradrenaline on the anal sphincter (Suppl. Fig. 5). Since low resting AP is associated with incontinence, our findings suggest improved faecal continence at higher SCPP.

Recto-anal inhibitory reflex (RAIR). The RAIR was observed in all 13 patients in whom it was tested, but not in all traces from each patient (Fig. 3). The SCPP had a major impact on the RAIR: compared with low SCPP (60.3 mmHg), high SCPP (90.0 mmHg) was associated with higher baseline AP (55.6 vs. 35.2 cmH_2O) and the RAIR was more likely to be present at the higher SCPP (89.7 vs. 69.5 % of attempts). When considering only the AP traces with the RAIR present, there was no difference in the RAIR characteristics (excitation latency, duration of reflex, % amplitude reduction, recovery time) at low vs. high SCPP. The RAIR is an essential reflex for the voluntary control of defaecation by enabling the rectum to discriminate between gas, liquid and solid contents.[24,25] Therefore, the more frequent presence of the RAIR at higher SCPP suggests increased continence at higher SCPP.

Anal pressure during cough. Cough tests were performed in all patients. In 11 patients cough was assessed at high (81.6 mmHg) and low (61.4 mmHg) SCPP (Fig.4). At high SCPP, the maximum AP during cough was significantly higher than at low SCPP, by over 20.0 cmH_20 (P <0.0001) on average. The increase in AP compared with resting pressure prior to the cough was also higher at high vs. low SCPP, by about 14.7 cmH_20 (P <0.0001) on average. As episodes of incontinence are more likely during cough, our data indicate that anal sphincter continence is improved at higher SCPP.

Anal pressure during voluntary squeeze. Increase in AP during squeeze was significantly reduced in our patients compared with reported normal male controls (mean endurance time 5.3 vs. 16 seconds, mean ΔAP 3.7 vs. 195 cmH_20).[23] Fig. 5 shows that the sphincter response has longer endurance at higher SCPP. An inverted U-shaped curve fit the relationship between ΔAP and SCPP, with the sphincter showing a stronger response at SCPP ~80 mmHg and weaker response at hypo- and hyper-perfusion ($\hat{R}^2 = 0.87$).

Rectal sensation. Threshold tests for rectal sensation were performed in 10/14 patients at high and low SCPP. The other 4/14 patients were sedated for ventilatory reasons. 6/10 patients reported rectal sensation during the tests (4 AIS A, 2 AIS C). At follow-up, all 6 patients who had rectal sensation at presentation had the same or improved AIS grade. The 4 patients lacking rectal sensation at presentation, were all AIS A and remained so. The probability of experiencing rectal sensation, first urge to defaecate or maximum tolerance vs. volume of the balloon was the same at high and low SCPP (Suppl. Fig. 6).

Role of MAP. We tested whether MAP could be used as a surrogate for SCPP (Suppl. Fig. 7). In all patients, there was strong positive correlation between MAP and SCPP, but with wide inter-patient variability. As MAP increases, mean AP increases then plateaus, but as SCPP increases, mean AP rises then falls (Fig. 2). For prolonged squeeze (Fig. 5), there was strong linear correlation between endurance time and SCPP and strong correlation between ΔAP and SCPP, but no correlation between endurance time and MAP ($\hat{R}^2 = 0.07$) and only weak correlation between ΔAP and MAP ($\hat{R}^2 = 0.47$). We conclude that MAP is not a good surrogate for SCPP.

Long term outcomes. There was no correlation between AIS grade on admission and anal sphincter function at follow-up (NBD, SCIM III bowel score). There was significant correlation between the average SCPP on admission and anal sphincter function at follow-up assessed with the NBD score ($P < 0.05$), but not with the SCIM III bowel score. See Fig. 6.

DISCUSSION

We showed that TSCI, causes severe disruption of anal sphincter function. Our key finding is that intervention to increase the SCPP improves several components of anal continence including the resting AP, the RAIR, AP during cough and AP during squeeze. We also showed evidence that too high SCPP may worsen anal sphincter continence. Being able to improve anal sphincter function early by optimising the SCPP is a compelling prospect.

The reported variability in ARM findings and its dependence on physiological changes at the injury site, e.g. change in SCPP, suggest that interventions to optimise injury site physiology may improve anal function. This is further supported by the finding that the average SCPP on admission correlated with the NBD score at follow-up. Ultimately, a randomized, controlled trial is required to definitively determine whether intervening to optimize the SCPP improves outcome. Intervention to increase SCPP (=MAP – ISP) may be achieved by increasing the MAP using inotropes[6] or by reducing the ISP using expansion duroplasty.[26] The finding that over-increasing the SCPP may worsen anal sphincter function suggests that not only hypoperfusion, but also hyperperfusion, at the injury site is detrimental. The notion that, after TSCI, injury site hyperperfusion is detrimental is also supported by our earlier studies that used injury site autoregulation,[6] injury site blood flow,[27] limb power[13] and urinary bladder function[14] as outcomes.

ARM offers mechanistic insight into the impact of TSCI on anal sphincter function. We found that, in the acute phase, TSCI causes relaxation of the anal sphincter. The anal

sphincter no longer contracts effectively in response to cough or voluntary squeeze and the RAIR is not always present. As a result of these disruptions, faecal continence can no longer be maintained.

We did not detect an effect of increasing SCPP on the rectal volumes required to elicit sensations (first sensation, first urge to defaecate, maximum tolerance). The data show a trend, i.e. increasing SCPP reduces the rectal volume required to first produce desire to defaecate or to first produce maximum tolerance, though these findings did not reach significance. The small number of patients in our study may be the limiting factor here and it is possible that increasing the number of patients may yield significance.

Our study has limitations. First, some patients were sedated, because they required ventilation, which prohibited them from participating in the squeeze and in the sensory threshold tests. Second, some patients received noradrenaline infusions, raising the possibility of a direct effect of noradrenaline on the anal sphincter. Our finding that the dose of noradrenaline does not correlate with resting AP suggests that a direct effect of noradrenaline on the anal sphincter is unlikely. Third, because of the acute ICU setting, we were unable to perform additional studies, e.g. high resolution ARM or anal ultrasound. Fourth, the number of patients in our study is relatively small, due to the difficulty in performing multiple ARM assessments in acutely injured patients in NICU. Importantly, no patient experienced complications directly related to ARM testing. The outcomes of TSCI patients that undergo monitoring from the injury site are comparable to non-monitored patients as previously reported.[10]

The finding that optimizing SCPP is associated with improved anal sphincter function, is in line with our earlier findings that optimizing SCPP is associated with improved limb motor score,[6,13] sensory level[28] and urinary bladder function.[14] Our analysis suggests that using MAP as a surrogate for SCPP is inadequate because patients that have the same

MAP and different ISP, will have different SCPP. A randomized controlled trial has been set up, termed DISCUS (Duroplasty for Injured Spinal Cord with Uncontrolled Swelling) funded by the U.K. NIHR (NIHR130048) to determine if expansion duroplasty improves neurological outcomes, including anal sphincter function, compared with standard treatment for patients with acute, severe, cervical TSCI. DISCUS includes optional monitoring from the injury site.

CONCLUSIONS

Our ARM findings suggest that individualised therapy in acute TSCI, by monitoring and optimising spinal cord physiology and metabolism, may improve not only limb motor function, sensation and urinary function, but also anal sphincter function.

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FIGURE LEGENDS

Fig. 1. Setup for monitoring. A. (*top left*) Intradural pressure probe monitors ISP. (*bottom left*) Radial artery catheter monitors ABP, used to compute SCPP as MAP minus ISP. (*right*) Anal probe monitors from 4 pressure sensors to compute average AP and maximum AP. Rectal balloon used to assess sensation. **B.** Examples of signals monitored simultaneously including ISP, ABP, SCPP and average AP.

Fig. 2. Correlation between SCPP and anal pressure. A. AP *vs.* SCPP for each of the 14 patients. (*inset)* AP averaged for all patients *vs.* SCPP. Mean +/- standard error with best-fit quadratic, $\hat{R}^2$ = 0.82. **B.** Individual curves from A. spread out. Colours correspond to patients as shown.

Fig. 3. Effect of SCPP on the RAIR. A. Schematic showing characteristics of the RAIR signal. **B.** Typical RAIR signals from patients 82 (*left*) and 78 (*right*) at low (open circles) and high (solid circles) SCPPs. **C.** Mean high (HI) and mean low (LO) SCPPs corresponding to the RAIR measurements for each patient. Plots showing individual patient values (points) and means (lines) at HI vs. LO SCPP of **D.** Mean baseline AP, **E.** Recovery time, and **F.** % Amplitude reduction of the RAIRs. Colour-codes for patients 71 – 84. $P < 0.05^{*}$, $<0.0001^{\#}$.

Fig. 4. Effect of cough on anal pressure. A. Anal pressure changes during cough for patients 6 (*left*) and 11 (*right*) at low (open circles) and high (solid circles) SCPPs. Plots showing individual patient values (points) and means (lines) at HI vs. LO SCPP of **B.** Mean SCPP, **C.** Mean maximum AP, and **D.** Mean change in AP (ΔAP) during cough. Colour-codes for patients 71 – 84. $P <0.0001^{\#}$.

Fig. 5. Effect of prolonged squeeze on anal pressure. A. Anal pressure changes during squeeze for patients 5 (*left*) and 10 (*right*) at low (open circles) *vs.* high (solid circles) SCPPs. Plots of **B.** Endurance time, and **C.** Change in AP (ΔAP), during straining. N = 10, mean +/- standard error. Best-fit straight line (A, $\hat{R}^2$ = 0.93) and quadratic (B, $\hat{R}^2$ = 0.87).

Fig. 6. Bowel function at follow-up. A. NBD scores *vs.* (*left*) AIS grade and (*right*) *vs.* average SCPP on admission. **B.** SCIM III bowel scores *vs.* (*left*) AIS grade and (*right*) *vs.* average SCPP on admission. NS, not significant. $P < 0.05^{*}$.

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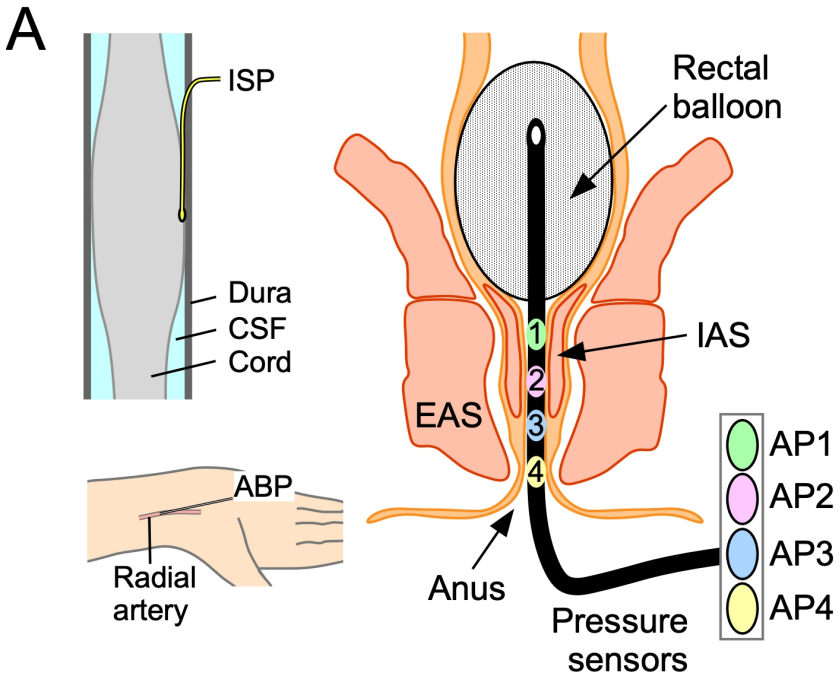
A
ISP
Dura
CSF
Cord
ABP
Radial
artery
Rectal
balloon
IAS
EAS
1
2
3
4
Anus
Pressure
sensors
AP1
AP2
AP3
AP4

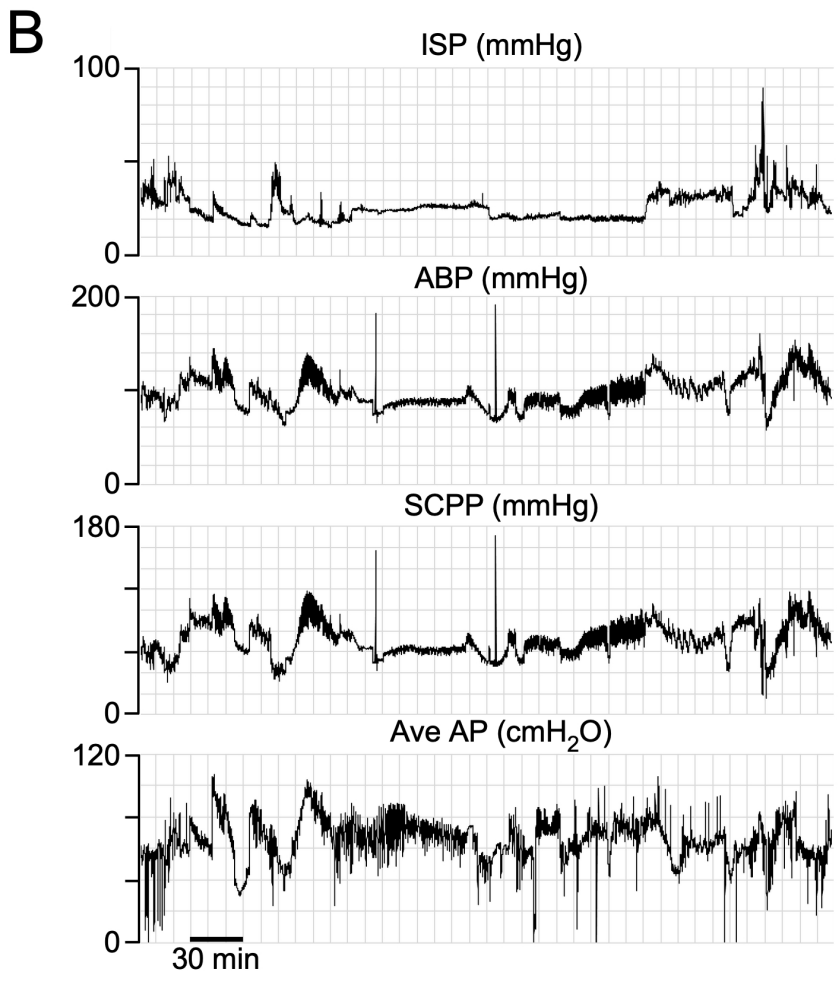
B
ISP (mmHg)
100
0
ABP (mmHg)
200
0
SCPP (mmHg)
180
0
Ave AP (cmH2O)
120
0
30 min

FIGURE 1

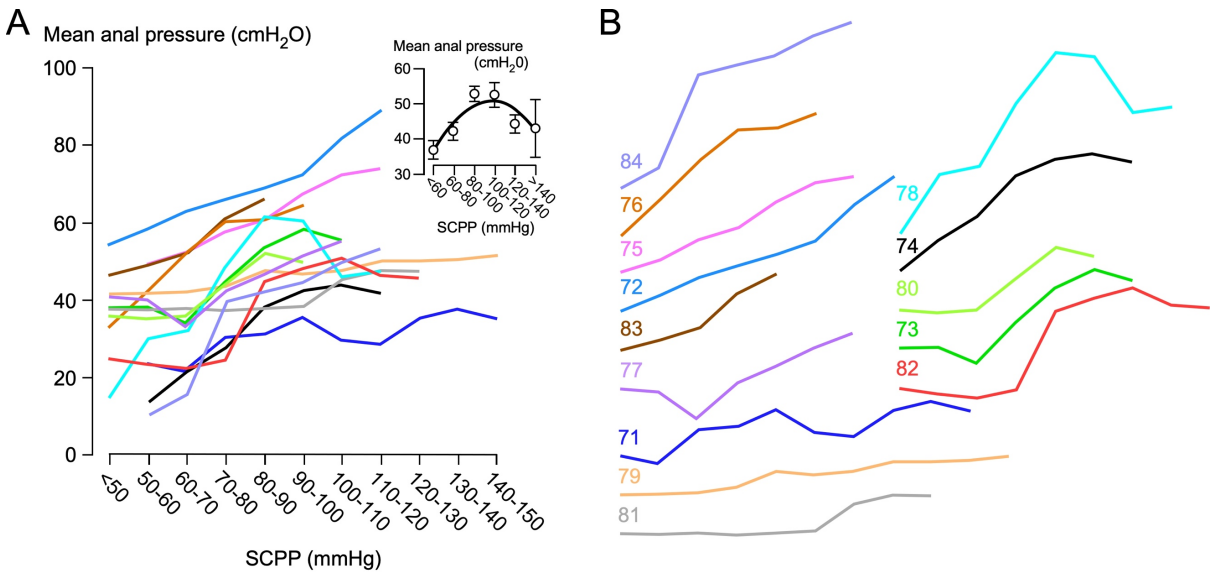
A
Mean anal pressure (cmH2O)
100
80
60
40
20
0
<50
50-60
60-70
70-80
80-90
90-100
100-110
110-120
120-130
130-140
140-150
SCPP (mmHg)
Mean anal pressure (cmH20)
60
50
40
30
<60
60-80
80-100
100-120
120-140
>140
SCPP (mmHg)
B
84
76
75
72
83
77
71
79
81
78
74
80
73
82

FIGURE 2

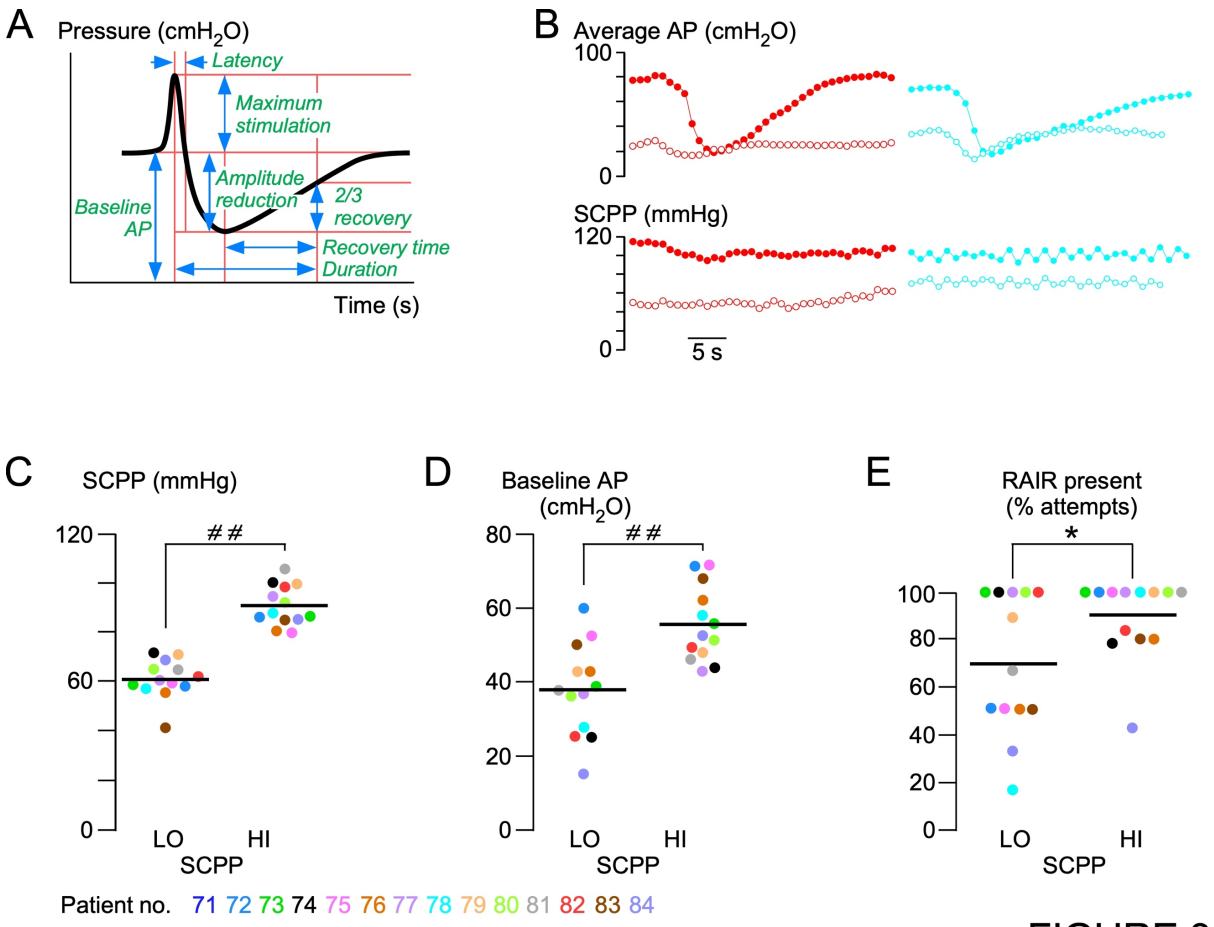
A
Pressure (cmH2O)
Latency
Maximum stimulation
Amplitude reduction
2/3 recovery
Baseline AP
Recovery time
Duration
Time (s)
B
Average AP (cmH2O)
100
0
SCPP (mmHg)
120
0
5 s
C
SCPP (mmHg)
120
60
0
##
LO
HI
SCPP
D
Baseline AP (cmH2O)
80
60
40
20
0
##
LO
HI
SCPP
E
RAIR present (% attempts)
*
100
80
60
40
20
0
LO
HI
SCPP
Patient no. 71 72 73 74 75 76 77 78 79 80 81 82 83 84

FIGURE 3

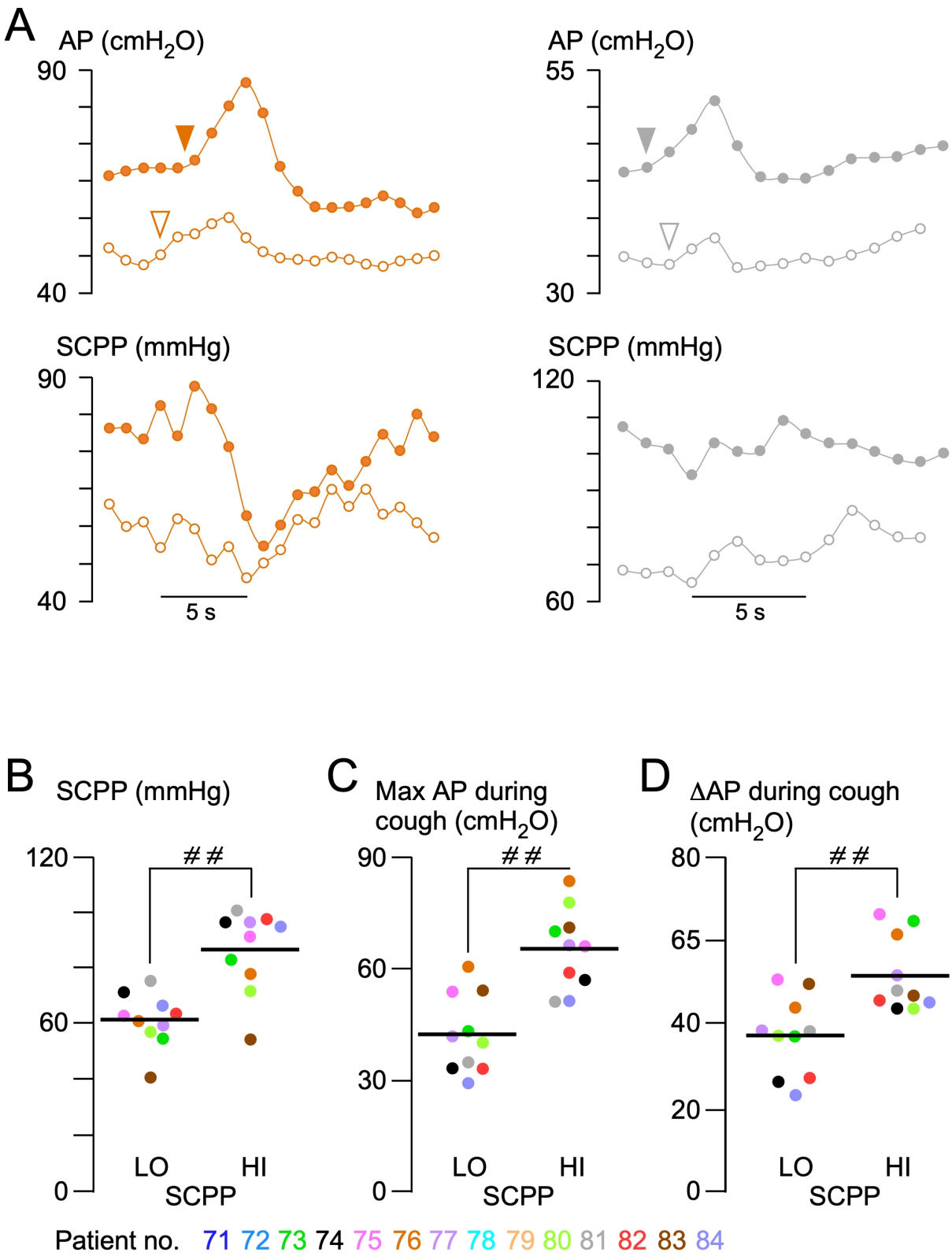
A
AP (cmH2O)
90
40
AP (cmH2O)
55
30
SCPP (mmHg)
90
40
5 s
SCPP (mmHg)
120
60
5 s
B
SCPP (mmHg)
120
60
0
##
LO
HI
SCPP
C
Max AP during cough (cmH2O)
90
60
30
0
##
LO
HI
SCPP
D
ΔAP during cough (cmH2O)
80
65
40
20
0
##
LO
HI
SCPP
Patient no. 71 72 73 74 75 76 77 78 79 80 81 82 83 84

FIGURE 4

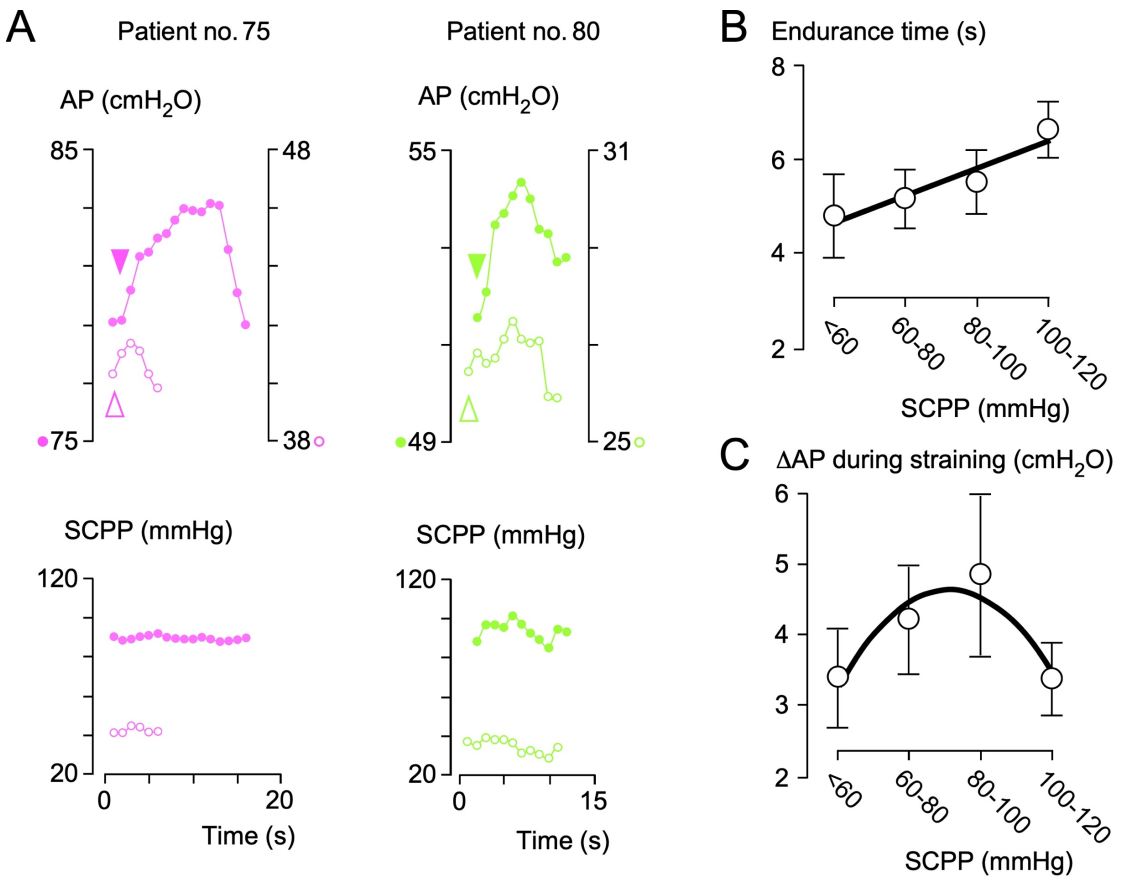
A
Patient no. 75
Patient no. 80
AP (cmH2O)
85
48
75
38
55
31
49
25
SCPP (mmHg)
120
20
0
20
15
Time (s)
B
Endurance time (s)
8
6
4
2
<60
60-80
80-100
100-120
SCPP (mmHg)
C
ΔAP during straining (cmH2O)
6
5
4
3
2
SCPP (mmHg)

FIGURE 5