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Scientific Review

Anorectal physiology following spinal cord injury

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Purpose: Spinal cord injured (SCI) patients have delayed colonic motility and anorectal dysfunction resulting in functional obstruction and constipation. This may be caused by changes in descending modulation from the central or sympathetic nervous systems. Anorectal dyssynergy may demonstrate similarities to that seen in the bladder following SCI.
Methodology: Anorectal manometry was performed on 37 SCI volunteers. Patterns of rectal and sphincter function were identified. These patterns were then compared with questionnaire answers on bowel function and cystometrograms to identify a relationship between detrusor dyssynergy and anal sphincter tone.
Results: Rectal compliance and basal resting sphincter pressures were lower than normal values. Ramp rectal inflation demonstrated patterns of sphincter activity similar to that recorded in the patients' cystometrograms. There is no definite relationship of bowel function to the findings on manometry in SCI patients.
Conclusions: SCI patients have abnormal anorectal function. Anorectal manometry results were able to be classified into four patterns on the basis of rectal pressure and sphincter tone in response to rectal distention. The patterns of anorectal manometry seen were similar to those in cystometrograms, however there is no definite relationship to bowel dysfunction.
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Introduction

Bowel dysfunction is a major disability following spinal cord injury. Faecal continence requires the ability to maintain resting internal anal sphincter (IAS) tone and external anal sphincter (EAS) contraction in response to increased intra-abdominal pressure, rectal distention and rectal contraction. These are spinal reflexes that may be intact following SCI, but no longer modulated by cortical input. SCI patients may be able to elicit reflex defecation, but it is often inefficient and incomplete, resulting in a change in bowel function.

SCI patients often have no sensation of rectal fullness and are unable to consciously initiate reflex defecation. Therefore defecation is planned on a regular basis to avoid constipation or an increased chance of faecal incontinence. Above the conus the rectocolic reflex can be employed to assist defecation. Insertion of a gloved finger into the rectum with gentle sustained pressure towards the sacrum relaxes the spastic EAS and pelvic muscles. Rapid or excessive stretching can precipitate sphincter spasm. Rotation of the finger continues the stimulation until a reflex peristaltic wave is generated in the rectum, flatus is passed and the stool comes down. The rectoanal inhibitory reflex is initiated, the IAS relaxes, and the rectocolic reflex stimulates pelvic nerve mediated peristalsis. Similar effect may be inducted by the use of a microenema or suppository. Reflex mediated defecation does not occur with spinal cord lesions below the conus. In this situation, loss of sphincter tone means that the rectum must be kept empty to reduce the incidence of faecal incontinence. A finger can stimulate local peristalsis in the rectum, but usually the stool has to be hooked out with a gloved finger.[1]

In normal individuals there is synergistic activity between rectal smooth muscle and pelvic striated muscles. Defecation and bladder voiding are the result of simultaneous relaxation of striated muscle and contraction of smooth muscle. This may be disturbed following spinal injury and result in contraction of both types of muscle leading to incomplete evacuation or obstruction of urine flow. There is clinical evidence that SCI patients exhibit a relationship between urethrovesical and anorectal dysfunction. Anal sphincter activity recorded simultaneously during cystometrography has been used as an indirect measure of urethral sphincter function.[2]

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Altered anorectal function is the basis of many defecatory problems following SCI. As is the case with bladder dysfunction, adequate identification of the problem with appropriate clinical testing may improve long term management. Anorectal manometry has previously been used to investigate sphincter function in the SCI patient population. Clinical patterns of dysfunction correlate with manometric results and may also correlate with patterns of bladder dysfunction, as such the study was conducted to examine the following hypotheses (1) SCI results in changes to the manometric characteristics of anal sphincter function. (2) These differences relate to the level and degree of injury. (3) Patterns of anorectal manometry and cystometry data can be correlated and related to EAS and IAS tone, detrusor spasm and dyssynergy.

Method

Volunteers were recruited from SCI patients presenting for routine inpatient follow-up at the Spinal Injuries Unit, Burwood Hospital, Christchurch, New Zealand. Patients were informed of the study and asked if they wanted to participate. Details on level and degree of injury were obtained from the casenotes following patient consent. The study had ethical approval from the Canterbury Ethics Committee.

Cystometry

Cystometry was performed on most patients as part of their follow-up. Interpretation of these results was according to patterns described by Arnold *et al*.[3] The pattern of cystometrogram can be classified as outlined in Figure 1. The volume pressure curve is useful as it describes detrusor function and coordination with the urethral sphincter.[3] Pattern F is a normal detrusor reflex with bladder filling resulting in very little change in pressure and on voluntary initiation of flow, intravesical pressure rises smoothly until the bladder empties. Pattern A shows a sustained detrusor contraction, but the implication from the high intravesical pressure is that there is a constant urethral resistance. Pattern B represents a detrusor contraction with fluctuating spasm in the dyssynergic urethral sphincter again resulting in high intravesical pressures. Pattern C is a flat curve without provocation of detrusor contraction. This has been associated with detrusor atonia[4] from LMN lesions or detrusor damage from overdistention which may be related to bladder neck obstruction from either sphincteric dyssynergia or detrusor hypertrophy.[5] Patterns D and E are similar and show decreased compliance with bladder filling resulting in a steeply increasing intravesical pressure.

Anorectal manometry

Anorectal manometry was performed as detailed below. Results were correlated with level and degree of injury and cystometry results. Manometry results were also correlated with the patient's questionnaire answers (Tables 1 and 5). Accepted normal results currently used in the manometry laboratory were used as control values.[6]

A four port plastic manometric catheter (Arndorter Inc, Wisconsin, USA) was used for anorectal pressure studies. Four side holes were positioned at 1 cm

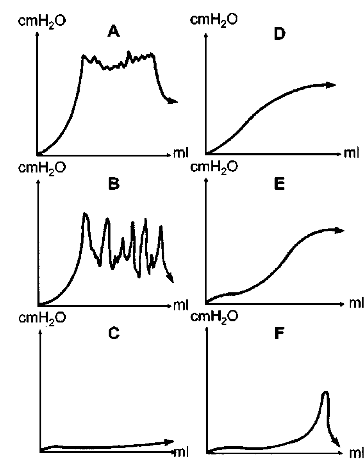

Figure 1 Classification of cystometrogram for SCI patients. Pattern A is a sustained detrusor contraction with constant urethral resistance resulting in high intravesical pressure. Pattern B represents detrusor contraction and fluctuating spasm in a dyssynergic urethral sphincter. Pattern C shows no detrusor contraction. Patterns D and E show decreased bladder compliance. Pattern F is the cystometry pattern generated by a normal bladder[3]

Table 1 Pattern of rectal ramp inflation curve according to level and degree of spinal cord injury (n = 37)

	Pattern 1	*Pattern 2*	*Pattern 3*	*Pattern 4*	*Total*
Complete cervical	1	2	1	7	11
Complete thoracic	1		2	5	8
Complete lumbosacral				4	4
Incomplete cervical		1		6	7
Incomplete thoracic			1	2	3
Incomplete lumbosacral			2	2	4
Total	2	3	6	26	37

intervals from the anal verge. Perfusion was with water using a hydraulic capillary perfusion system. Pressures were measured using pressure transducers situated in each perfusion line and connected via amplifiers to a four channel chart recorder. A distensible latex balloon was tied to the end of the catheter approximately 5 cm from the anal verge and used to inflate the rectum. The balloon pressure was monitored using a non-perfused air filled catheter with a separate side hole within the balloon. No bowel preparation was used. The study was performed on the afternoon following a planned bowel evacuation. With the patient in the left lateral position and the legs flexed to 90°C, the catheter balloon was then placed in the rectum and the force transducers equilibrated. The tube was then inserted further into the rectum and sphincter length measured. When the sphincter had equilibrated to a basal resting pressure, the following manoeuvres were performed: (1) voluntary contraction as if attempting to squeeze (2) increasing intraabdominal pressure by Valsalva manoeuvre (blowing up a party balloon).

The rectal balloon was serially inflated in 10 ml increments until sustained IAS relaxation was noted. Volume at first IAS relaxation and any sensations felt by the patient were recorded. After a further test, the balloon was inflated to maximum tolerable volume or until expelled, whichever came first.

Analysis
The following parameters were recorded or calculated for each subject: basal anorectal pressure (resting tone), change in rectal and anal pressures during voluntary squeeze, change in rectal and anal pressures with Valsalva, rectal sensitivity to balloon inflation, threshold for onset and sustaining of IAS relaxation during ramp inflation of rectum, sphincter pressure during ramp inflation, rectal pressures/rectal volume relationship, maximum distention volumes or volume at expulsion.

Results

Demographic data
Anorectal manometry was performed on 37 spinal cord injured patients (36 male, one female). The average age of the participants was 40 years (range 18–71 years). Mean time since injury was 8 years (range 3 months–25 years). Twenty-three had complete spinal cord injuries (11 cervical, eight thoracic, four lumbosacral). Fourteen had incomplete injuries (seven cervical, three thoracic, and four lumbosacral).

Anorectal manometry
Data for mean basal anorectal pressure are summarised in Figure 2. Sphincter pressures were higher than basal rectal pressure for all levels and degree of injury. IAS and EAS pressures were at the lower end of the normal range (40–70 mmHg). There was no significant difference in basal pressures for any level or degree of spinal cord injury.

Figure 3 shows the mean anorectal pressures during an attempted voluntary squeeze. The sphincter pressure was maintained above rectal pressure in all cases. Of note, the mean percentage increase (Figure 4) of pressure on voluntary squeeze was greater for the internal rather than the external sphincter. All injury groups could increase EAS pressure by at least 20%.

Mean anorectal pressure increased during Valsalva ($P<0.001$) (Figure 5). The increase in rectal pressure with Valsalva (Figure 6) was greater for incomplete injuries than complete. Increases in IAS and EAS tone were greater for all groups compared with attempted voluntary squeeze.

Rectal sensation
Thirteen of the 37 subjects noted some sensation on ramp inflation (Table 2). The mean rectal volume to generate sensation was 62 mls for complete injuries

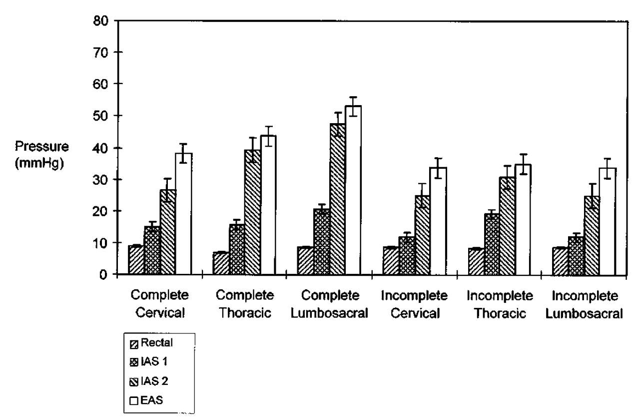

Figure 2 Mean basal anorectal manometric pressures (±SE) for spinal cord injured patients according to level and degree of injury ($n=37$)

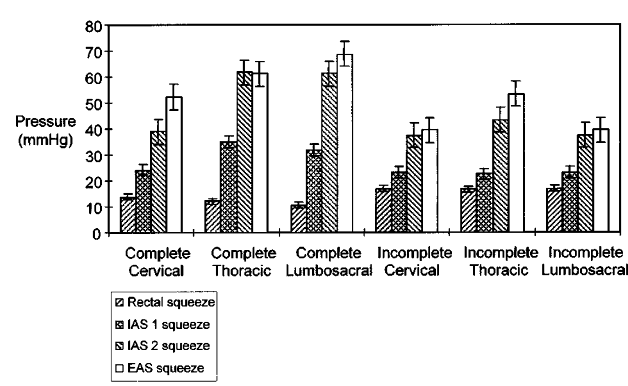

Figure 3 Mean manometric pressure (±SE) during voluntary squeeze for patients with SCI according to degree and level of injury ($n=37$)

(range 30–120 mls) and 32.5 mls for incomplete injuries. The volume for complete injuries was higher than the accepted normal range of 10 to 30 mls.[6] Of the 24 who reported no sensation during the procedure, 18 (75%) were complete injuries and 6 (25%) were incomplete injuries ($P=0.04$).

IAS relaxation

Ramp inflation curves were obtained for all patients and graded according to pressure changes in the rectum and sphincter. The mean threshold rectal volume for the onset and sustaining of IAS relaxation is presented in Table 3. It was not significantly different for any injury group. The rectal volume at which sustained IAS relaxation occurred was higher for those patients with incomplete lumbosacral injuries where the volume was almost double the mean for the group as a whole.

Table 2 Reporting of rectal sensation by SCI patients during ramp inflation of rectal balloon (n = 37)

	Sensation reported	*No sensation reported*	*Total*
Complete cervical	5	6	11
Complete thoracic	0	8	8
Complete lumbosacral	0	4	4
Incomplete cervical	4	3	7
Incomplete thoracic	2	1	3
Incomplete lumbosacral	2	2	4
Total	13	24	37

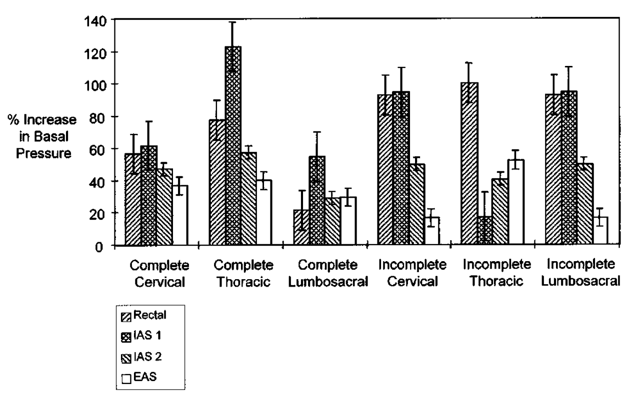

Figure 4 Mean percentage increase in anorectal pressures (±SE) during voluntary squeeze for patients with spinal cord injury according to degree and level of injury ($n=37$)

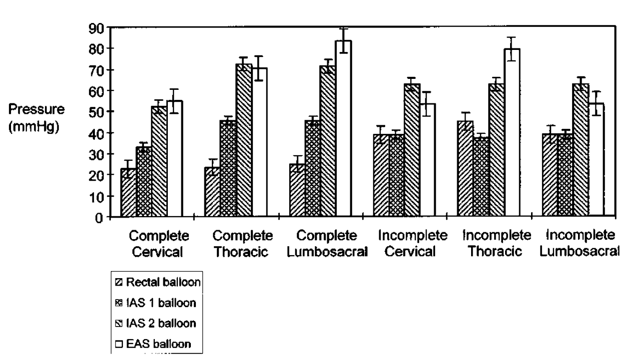

Figure 5 Mean manometric pressures (±SE) during Valsalva manoeuvre for patients with SCI according to degree and level of injury ($n=37$)

Ramp inflation

The mean maximum tolerated rectal volume is presented in Table 4. The rectal balloon was expelled in 24 subjects following complete sphincter relaxation. Two patients with incomplete cervical injuries had increased rectal tone and spasm with increased rectal volumes.

The ramp inflation curves generated show four basic types of pattern with increasing volume. The manometry results were grouped according to change in rectal pressure and sphincter response. The four patterns are shown in Figure 7. A sharp rise in rectal pressure with increasing volume implies low rectal compliance compared with the small increase in pressure seen normally. IAS relaxation and continued EAS contraction is a normal sphincter response with increasing rectal volume.

- Pattern 1 graphs show rectal and sphincter tone increase with increasing rectal distention. This

Table 3 Mean rectal distention required for the onset and sustaining of IAS relaxation by SCI patients during ramp inflation of rectal balloon (n = 37)

	Mean threshold volume (mls)	*Mean sustained relaxation volume* (mls)
Complete cervical	9	52
Complete thoracic	10	86
Complete lumbosacral	10	56
Incomplete cervical	12.5	73
Incomplete thoracic	13	56
Incomplete lumbosacral	12	126
Average volume	10	67

Table 4 Endpoint for ramp inflation of the rectum for SCI patients (n = 37)

	Mean maximum tolerated volume (mls)	*Range*	*Balloon expelled (/total)*
Complete cervical	120	30–240	8/11
Complete thoracic	96	30–120	4/8
Complete lumbosacral	165	120–240	4/4
Incomplete cervical	140	80–180	4/7
Incomplete thoracic	75	30–120	2/3
Incomplete lumbosacral	170	160–180	2/4
Total	170	30–240	24/37

occurred in two patients, one with a complete cervical injury and one with a complete thoracic injury.

- Pattern 2 graphs show decreased or stable rectal pressure and increased sphincter tone.
- Pattern 3 graphs demonstrate a normal defecatory pattern with increased rectal pressure and decreased sphincter pressure on rectal distention. However, volume at which defecation occurred was lower than would be expected in normal subjects.
- Pattern 4 graphs showed decreased or stable rectal pressure with decreased sphincter pressure on rectal distention.

The normal situation is similar to that seen in Pattern 2. A normally compliant rectum accommodates the increase in volume with little change in pressure. Both sphincters are maintaining a relatively normal tone and pressure, although the EAS is at the low end of the normal range at 40–50 mmhg. Pattern 1 demonstrates a spastic response of both sphincters and a non-compliant rectum. Pattern 3 describes those who reflexly defecated the balloon probe as rectal pressure rises higher than sphincter pressure at a mean rectal volume of 60 mls. Pattern 4 shows a normally compliant rectum and an IAS that relaxes, but no increase in EAS tone.

There was no significant change in anorectal manometry pattern observed for increasing age ($P=0.47$), or time since injury ($P=0.143$) for the study group.

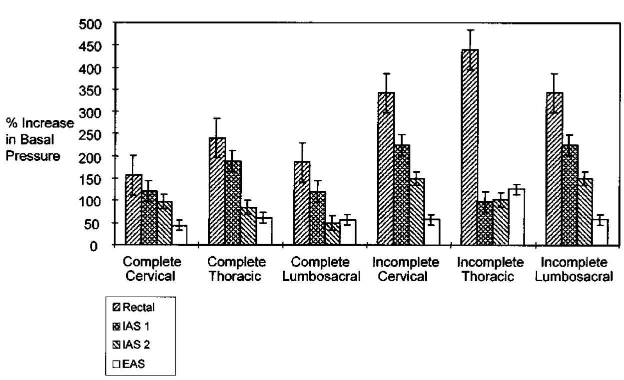

Figure 6 Mean percentage increase in anorectal pressures (±SE) during Valsalva for patients with spinal cord injury according to degree and level of injury ($n=37$)

Comparison with bowel function questionnaire

A previously described bowel function questionnaire was completed by 24 of the 37 SCI patients having anorectal manometry.[7] All patients were requested to complete the questionnaire but 13 declined. The Faecal Incontinence Score[8] was calculated for these patients (Table 5), and the mean score derived for each manometric pattern. For the group of 24 SCI patients as a whole, mean Faecal Incontinence Score was 4.75. It was lower for those SCI patients with Patterns 1 or 2, however the numbers are small ($P=0.213$). Incontinence did not affect the every day activities of anyone with manometry of Patterns 1, 2, or 3, and only occasionally for Pattern 4. Table 6 shows the distribution of cystometry patterns[3] for each anorectal manometry pattern. The cystometrogram was abnormal in all cases. Hyperreflexic cystometrograms (Patterns A and B) were present in 14 SCI patients. Ten patients had an areflexic cystometrogram (Pattern C), and 10 had cystometrograms showing decreased detrusor compliance (Pattern D or E).

Table 7 shows the anorectal manometry and cystometry patterns for all injury groups. SCI patients with complete lumbosacral injuries (four) had Pattern 4 manometry and either Pattern C or D/E cystometry, demonstrating no evidence of detrusor dysfunction. Incomplete injury resulted in evidence of dyssynergic detrusor function in three out of 13 patients, who all had incomplete cervical injuries. All of those with Pattern 1 or 2 manometry had complete cervical or thoracic injury.

Table 7 shows the distribution of ramp inflation patterns according to level and degree of injury. Pattern 4 was evenly spread over all types of injury, and included all patients with complete lumbosacral lesions. Both patients with Pattern 1 manometry had a complete cervical or thoracic injury.

Discussion

Basal sphincter tone was reduced for the SCI patient group compared to controls. The results are similar to a previous study where the maximal mean basal

Table 5 Mean Faecal Incontinence Score, bowel motion frequency, and frequency of manual evacuation and laxative use for SCI patients (n = 24)

Anorectal manometry pattern	*Number*	*Mean faecal incontinence score*	*Bowel motions/week*	*Manual evacuations*	*Laxative use*
Pattern 1	1	0	3	1	1
Pattern 2	1	2	5		0
Pattern 3	3	4	7	3	0
Pattern 4	19	5.3	7.2	13	9
Total	24	4.75	4	17	10

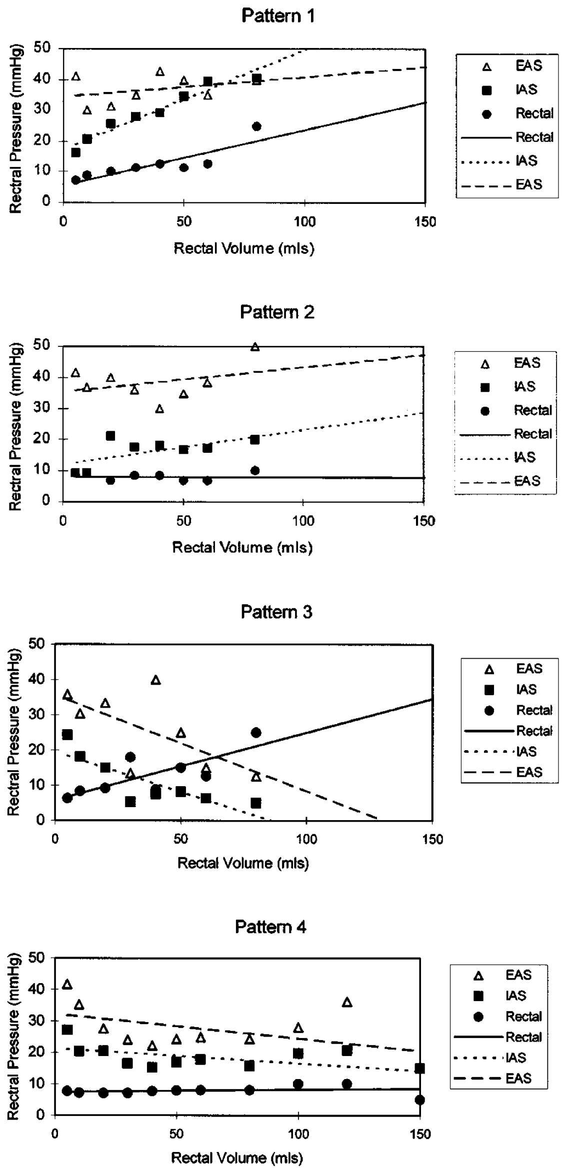

Figure 7 Anorectal manometry of SCI patients. Results for ramp rectal inflation grouped according to mean rectal and sphincter pressure trends ($n=37$). Pattern 1 – Increased rectal and sphincter tone. Pattern 2 – Normal rectal tone and increased sphincter tone with increasing rectal volume. Pattern 3 – Increased rectal and decreased sphincter tone (defecation). Pattern 4 – Normal rectal tone and decreased sphincter tone with increasing rectal volume

sphincter pressure (which probably reflects EAS pressure), was 63 mmHg. This was lower than the study's control group pressure of 116 mmHg ($P<0.01$).[9] Here, the results show a similar maximal mean basal sphincter pressure of 42 mmHg. The maintenance of continence by basal anal tone is mainly as IAS activity, demonstrated here by reduced but persistent anal tone following SCI. The maintenance of IAS tone may be due to a tonic excitatory

Table 6 Anorectal manometry patterns compared with cystometry patterns[3] for SCI patients (n = 34). The number with a urinary catheter is in parentheses

	Cystometry Pattern				
	A	*B*	*C*	*D/E*	*Total*
Pattern 1	1	1 *(1)*			2 (1)
Pattern 2		1 *(1)*		2 *(1)*	3 (2)
Pattern 3		2	4 *(2)*		6 (2)
Pattern 4	1 *(1)*	8 *(3)*	6	8 *(3)*	23 (7)
Total	2	12	10	10	24

sympathetic discharge. This was described by Frenckner and Ihre in 1976.[10] They found changes in anal tone in eight healthy subjects following spinal anaesthesia. High spinal anaesthesia resulted in a significantly lower resting anal pressure than either low spinal or pudendal block.

The EAS also continues to show tonic activity, but again generates a lower than normal pressure. Of note, those patients with lumbosacral injuries still appear to maintain a degree of anorectal tone, higher than rectal pressure, but lower than normal EAS tone.

All patient groups produced a small rise in sphincter pressure with attempted voluntary squeeze ($P>0.05$). The increase can be compared with a similar manoeuvre performed by control subjects that generated a fourfold increase in EAS pressure.[11]

SCI patients with incomplete injuries produced a greater increase in sphincter pressure with Valsalva than those with complete injuries. This may reflect the greater increase in intra-abdominal pressure, as measured by rectal pressure that patients with incomplete or low injuries were able to generate. Similar results are seen for complete injuries as a squeeze. This suggests that attempts at squeezing by these patients have resulted in a straining response rather than a true squeeze. Patients with lesions above T5 will be unable to use abdominal muscles and rely on intercostal and diaphragmatic muscle contraction to increase intra-abdominal pressure. Those with cervical injuries can only use the diaphragm. These observations fit the concept that EAS contraction is mediated by a spinal reflex, triggered by tension receptors in the pelvic floor that respond to an increase in intra-abdominal pressure. This is supported by another study that found the rise in sphincter pressure with Valsalva to be directly proportional to the rise in intra-abdominal pressure.[11]

To effect defecation, the EAS needs to relax in a coordinated fashion on straining. This cortically mediated pathway is no longer intact following complete supraconal spinal cord injury, and straining by increasing intraabdominal pressure does not improve evacuation, but rather, increases EAS tone.

Table 7 Anorectal manometry pattern and cystometrogram pattern compared to level and degree of injury for SCI patients (n = 34)

	Pattern 1		*Pattern 2*		*Pattern 3*		*Pattern 4*				
	A	*B*	*B*	*D/E*	*B*	*C*	*A*	*B*	*C*	*D/E*	*Total*
Complete cervical	1		1	2	1			2		2	9
Complete thoracic		1			1	1	1	3	1		8
Complete lumbosacral									2	2	4
Incomplete cervical								3	1	2	6
Incomplete thoracic						1				2	3
Incomplete lumbosacral						2			2		4
Total	1	1	1	2	2	4	1	8	6	8	34

Rectal sensation

The SCI patients who reported sensation on rectal distention described nonspecific abdominal sensation that did not prevent further filling of the balloon. One previous study examining similar sensation proposed that sympathetic nerves entering the thoracic spinal cord above the level of the injury conveyed this dull pelvic sensation.[11] In this study, such sensation was found to be present in five patients with complete cervical injuries, which does not support this hypothesis, making the origin of this sensation unclear. Normal subjects have been reported to experience a range of sensations starting at a rectal distention volume of about 10 mls, and ranging from sensations of 'wind' to pain.[11]

Rectoanal inhibitory reflex

The RAIR was present for all patients and occurred at a lower rectal volume than would be expected for controls.[11] Its presence would suggest that the RAIR is independent of spinal control. This may be a factor in precipitating episodes of faecal incontinence, especially among those with high injuries where threshold volume and volume to sustained relaxation are lowest (Table 2). The higher rectal volumes required to initiate and sustain the RAIR for those patients with low injuries may be related to their more compliant rectum needing a larger volume to produce the same degree of rectal stimulation.

Ramp inflation

We can see that defecation is assisted in SCI patients with ramp inflation Patterns 3 or 4. The normal rectal relaxation and sphincter contraction seen in Pattern 2 are exaggerated. A rapid rise in rectal pressure was accompanied by anal relaxation and balloon expulsion. This supports the hypothesis that IAS relaxation is an enteric reflex normally suppressed by descending inhibitory pathways. The loss of inhibitive sympathetic tone has also been proposed as a mechanism for the absent rectal relaxation and linear pressure/volume relationship during rectal distention seen in supraconal SCI.[11] The normal rectal response to ramp inflation produces a compliant, flat curve (Patterns 2 and 4), unlike to the linear response seen in Patterns 1 and 3.

Defecation requires the cortical inhibition of the EAS in response to rectal and intra-abdominal pressure increase. The Pattern 1 EAS response to ramp inflation seen with several patients with high complete injuries demonstrates how straining can often be ineffective in promoting defecation and confirms the importance of descending inhibitory pathways.

Of note, most patients with low lumbosacral injuries had Pattern 4 manometry, supporting the observation from previous studies that the rectum is areflexic with an attenuated sphincter response to rectal distention.[12] Age or time since injury had no significant effect on anorectal manometry pattern.

Bladder and anorectal correlations

There are analogies between colorectal and urinary dysfunction following SCI. Both are reservoir organs that require coordinated smooth and striated muscle interaction for normal functioning. With complete spinal injury, there is no sensation from the bladder or colon apart from what bypasses the lesion in the sympathetic nervous system. There is no voluntary control and loss of descending inhibition can result in facilitation of reflexes. This can result in spasticity as seen in skeletal muscle and the striated pelvic sphincters. The predominant clinical symptoms caused by an uninhibited neurogenic bladder are frequency of voiding, urgency, and urge incontinence. The main symptom of an uninhibited colon is constipation.[13]

LMN lesions can result in an atonic bladder (Pattern C) with high large volume and high compliance.[4] Some of these patients may go on to develop increased bladder tone and low compliance (Pattern D/E). The main reasons for patients with suprasacral lesions to develop an atonic bladder is an unrecognised sacral injury or an episode of bladder overdistention resulting in temporary loss of detrosor function. All those patients with a LMN lesion had either Pattern C or D/E cystometry, and Pattern 3 or 4 manometry.

In a survey of chronic gastointestinal problems in SCI patients, 72% of those complaining of difficulty in evacuating their bowel had required transurethral sphincterotomy for an inability to adequately void the bladder.[14] There is clinical evidence that spinal cord injured patients exhibit a relationship between urethrovesical and anorectal dysfunction. Simultaneous anal sphincter activity can be used as an indirect measure of urethral sphincter function during cystometrography. In one series,[2] most of those with Pattern A cystometrogram had anal sphincter activity that increased abruptly simultaneously with an uninhibited detrusor contraction. All of those with a Pattern B cystometrogram (demonstrating poor detrusor contraction) had injuries above T10 and increased anal sphincter activity with bladder filling, all had sphincter dyssynergy and difficulties with voiding. Those with a decrease in anal sphincter activity with simultaneous detrusor contraction usually had no troubles with voiding. A further study demonstrated changes in vesicourethral function following rectal distention.[15] In those with complete lesions and a hyperreflexic bladder, rectal distention correlated with reduction in bladder compliance, increased urethral sphincter dyssynergia, and earlier larger hyperreflexic contraction. In those with incomplete lesions rectal distention produced a reduction in bladder reflex activity.

Here the majority of those with incomplete injuries (10/13) did not have a hyperreflexic bladder or rectum. Those with complete suprasacral injuries were more likely to have detrusor hyperreflexia (Pattern A or B, 11/17), but not necessarily low rectal compliance.

All SCI patients studied had preserved resting sphincter tone characteristics. However, sphincter pressures were globally reduced and discoordinate response to squeeze and Valsalva was seen. Rectal distention generated patterns of sphincter responses. These show a range of response from reflex defecation to increased rectal compliance, but all intimate at the discoordinate nature of anorectal function following SCI. The identification of patterns of dysfunction may allow targeted intervention for those SCI patients with bowel problems.

The anorectal dysfunction seen in patients with high spinal cord injuries would seem to be related to: (1) Increased rectal contraction and anal relaxation in response to low distending volumes (2) Reduced rectal sensation (3) Loss of conscious EAS control. Faecal incontinence is exaggerated when these problems interact loss of sensation of rectal fullness together with an exaggerated tendency towards defecation result in unpredictable, episodic incontinence.[16]

SCI patients with low injuries produced: (1) Lower increase in sphincter pressure with Valsalva and squeezing, (2) Increased rectal compliance in response to rectal distention, (3) Reduced rectal sensation. This reduced sphincter response may contribute to the higher incidence of faecal leakage reported by these patients. Basal pressures, however were not as low as other similar studies.[16]

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