Spinal Cord (2001) 39, 193–203

www.nature.com/sc

Scientific Review

Bowel dysfunction following spinal cord injury

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Study Design: Review.
Objectives: To outline the present knowledge of bowel dysfunction following spinal injury, and look at future directions of management and research.
Setting: Spinal Unit and Colorectal Unit, Christchurch, New Zealand.
Methodology: Review.
Results: The underlying physiology of colorectal motility and defecation is reviewed, and consequences of spinal cord injury on defecation are reported. A discussion of present management techniques is undertaken and new directions in management and research are suggested.
Conclusion: There is need for more intervention in regard to bowel function that could improve quality of life, but there is also a need for more research in this area.
Sponsorship: Dr AC Lynch is a holder of a Royal Australian College of Surgeons Foundation Research Scholarship and the research has been supported by the Canterbury Medical Research Foundation, and the Burwood International Spinal Trust.
Spinal Cord (2001) **39,** 193–203

Keywords: spinal cord injury; bowel function

Introduction

Bowel dysfunction following spinal cord injury (SCI) is increasingly recognised as an area of major physical and psychological difficulty for SCI patients. Following spinal cord injury, changes in bowel motility, sphincter control, and gross motor dexterity interact to make bowel management a major life-style problem limiting quality of life.[1] Once the acute phase is over and patients have adapted to their loss of mobility, surveys have shown that approximately one third of subjects rank colorectal problems as worse than both bladder and sexual dysfunction.[2–6]

Colorectal problems can be a significant cause of morbidity immediately after SCI, and chronic gastrointestinal problems remain common, but may become more frequent with increasing time after injury.[7] Although many SCI patients achieve an adequate bowel frequency with drugs and manual stimulation, the risk and occurrence of faecal incontinence, difficulties with evacuation, and need for assistance remain significant problems. In a recent study, questionnaires were sent to 1200 SCI patients, and 1200 age and gender matched controls. The mean faecal incontinence score was higher for SCI patients than controls ($P<0.0001$), and for complete SCI compared with incomplete injury ($P=0.0023$).[8] Age or time since injury did not affect the faecal incontinence score. Incontinence affected quality of life for 62% of SCI patients, compared with 8% of controls. Faecal urgency and time spent at toilet were also significantly higher for the SCI group. Thirty-nine per cent of SCI patients use laxatives, compared to 4% of controls ($P<0.0001$). Haemorrhoidectomy was more common in the SCI population (9% *vs* 1.5% ($P<0.001$)), particularly among those requiring manual evacuations. Stone *et al*. found that improved management of bowel dysfunction led to improved well-being.[9]

In order to understand why SCI patients have such problems it is important to review the normal anatomy and physiology, the relationship between the enteric nervous system and autonomic nervous systems.

Normal colon anatomy and physiology

The human colon is a compliant sac approximately 1.5 m long, closed at one end by the ileocaecal valve and the anal sphincter at the other. It has two layers

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of smooth muscle. The inner layer is circular and thickens to form the internal anal sphincter (IAS) in the distal rectum. The outer layer is arranged in three distinct bands of longitudinal smooth muscle called the *taeniae coli*. Included in the pelvic floor are the levator ani, which form a funnel from the sides of the pelvis in which the pelvic organs sit. Just inside the anus is the external anal sphincter (EAS) complex, described by Shafik as three counterpoised U-shaped loops (Figure 1).[10] The upper loop, formed mainly by puborectalis, arises from the symphisis pubis, loops around the upper part of the rectal neck and opens ventrally. It is innervated by the inferior haemorrhoidal nerve. The intermediate loop, from the anorectal raphe and coccyx, opens dorsally and is innervated by the perineal branch of the fourth sacral nerve. The basal loop, from the skin anterior to the anus, opens ventrally and encloses the lower rectal neck. It is supplied by the inferior haemorrhoidal nerve.[10,11] As each loop has its own separate bilateral nerve supply, each can function as a sphincter independently.

Continence is maintained by the resting tone and reflex activity of the IAS, EAS and muscles of the pelvic floor. The resting anal canal pressure is maintained by tonic contraction of the IAS. Reflex contraction of the EAS complex on coughing or Valsalva prevents leakage by kinking the anal canal in opposing directions. Rectal distention produces stretching of the puborectalis and the urge to defecate. The distended rectum causes reflex relaxation of the IAS (rectoanal inhibitory reflex (RAIR)) resulting in faeces reaching the upper anal canal where receptors sample the rectal contents. Then voluntary EAS contraction maintains continence by mechanically sealing the rectal neck and mechanically preventing further relaxation of the IAS. Defecation is a coordinated event requiring simultaneous relaxation of the puborectalis to widen the anorectal angle, relaxation of the EAS, and rectal contraction.

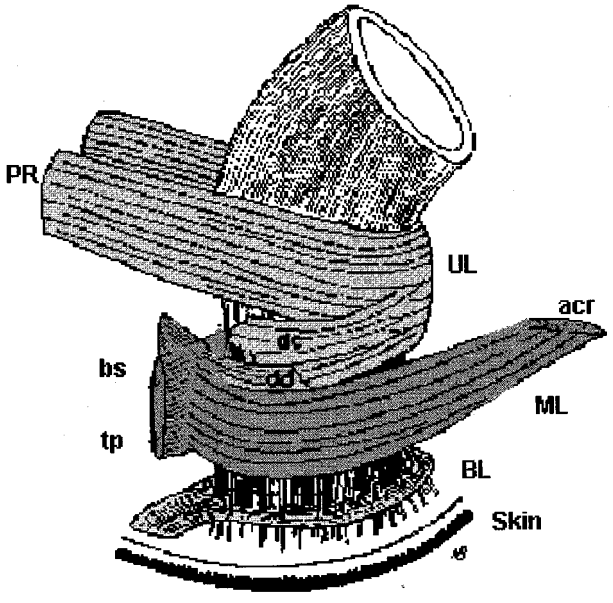

Figure 1 The external anal sphincter (EAS), summarising the basic arrangement of its fibres. Puborectalis (PR) forms the upper loop (UL) and has decussating fibres (dc) that blend with the longitudinal fibres of the rectum, or (dd) the perineal body. The middle loop (ML) is attached to the anococcygeal raphe (acr), bulbospongiosus (bs), and transversus perinei profundus (tp). The basal loop (BL) is perforated by longitudinal fibres of the rectum[11]

The enteric nervous system

Nervous control of the gastrointestinal tract is by the enteric nervous system. This complex network of intrinsic neurons is able to sense information, process it by means of interneurons and then through motor neurons effect secretion or muscular contraction. It retains contact with the central nervous system through afferent and efferent extrinsic neurons of the sympathetic and parasympathetic systems.[12]

There are two main ganglionated plexuses, the myenteric (Auerbach's) and submucosal (Meissner's). The myenteric plexus lies between the longitudinal and circular layers of muscle for the entire length of the gut. It provides motor innervation to the two muscle layers and secretomotor innervation to the mucosa. There are also projections from the myenteric plexus to the sympathetic ganglia. The submucous plexus is located in the submucosa between the circular muscle and the muscularis mucosa. It plays an important role in secretory control, especially in the small intestine. It also innervates the muscularis mucosa, intestinal endocrine cells and submucosal blood vessels. Smaller nerve fibres emerge from these plexuses to form nonganglionated plexuses in the circular and longitudinal muscle and muscularis mucosae.[13]

The neurons can contain not only acetylcholine and norepinephrine, but also Substance P (SP), vasoactive intestinal peptide (VIP), serotonin, somatostatin, and other neuropeptides, often coexisting in the same neurons.

The neurons of the enteric nervous system can be classified into intrinsic afferent, interneurons and motor neurons.

- Intrinsic afferent neurons form the sensory limb of intrinsic motor and secretomotor reflexes by projecting to interneurons in both nerve plexuses. They are all cholinergic and may contain other neurotransmitters such as SP.[12]
- Interneurons project either up or down the gut between the afferent and motor or secretomotor neurons. They form multisynaptic pathways to control the propagation of peristaltic waves. There are several subgroups based on neurotransmitter content, but their various physiological roles are unknown.

- Motor neurons are either excitatory or inhibitory. The excitatory neurons project either locally or orally to the circular muscle, their main neurotransmitters being acetylcholine or SP. The inhibitory motor neurons project to the circular muscle caudally and contain VIP and nitric oxide (NO).[12]

Neuropeptides

Peptides destined for secretion by neurons are synthesised in the cell body then transported intraaxonally to the nerve endings where they are stored in synaptic vesicles until release. There is no evidence for a re-uptake and re-use pathway for neuropeptides.

Substance P

Substance P (SP) was the first gut neuropeptide to be discovered in 1931. It occurs widely in brain, spinal cord, gut nerves, and mucosal endocrine cells. It is a dose-dependent excitatory neurotransmitter acting within the myenteric plexus on cholinergic neurons and directly on smooth muscle to cause contraction in both longitudinal and circular muscle layers. It is known to increase gut motility and may promote and maintain the peristaltic mode of intestinal motility. SP also increases bloodflow in gut, binds to specific receptors on pancreatic acinar cells associated with enzyme secretion, and inhibits acid secretion and intestinal absorption.[14]

Evidence for this role includes the observation that carcinoid tumors are often associated with increased gut motility and diarrhoea. These tumours can synthesise and release large amounts of SP, and serotonin, known to lower the threshold for the peristaltic reflex in isolated segments of intestine, into the portal and systemic circulations.

Vasoactive intestinal peptide

Vasoactive intestinal peptide (VIP) is present in a wide range of neurons in both gut and elsewhere. It is a strong stimulant of intestinal secretion, and causes gastrointestinal relaxation. Myenteric VIP neurons project in an oral to anal direction and are compatible with a role in descending inhibition.[14]

Central and enteric nervous system interaction

The coordination of transport, secretion and blood flow requires a high degree of extrinsic and intrinsic neuronal integration. The intrinsic neurons of the gut exhibit a sophisticated pattern of behaviour independent of, but modulated by the extrinsic parasympathetic and sympathetic nervous system.[14] The extrinsic system acts to modulate intrinsic reflexes and coordinate gut activity with that of the whole organism. When the gut is disconnected from the central nervous system, its function is preserved, but complex activities requiring both voluntary and reflex activity can be impaired.[13]

The control of colonic movement is largely autonomous. Intrinsic rhythmic slow waves originating in the submucous plexus occur sequentially along the colon. In the right colon slow waves of contraction may travel in both directions to produce mixing and kneading contractions of the circular muscle layer. The slow waves in the distal colon are directed towards the anus to produce a propulsive peristaltic force. Normal colonic transport is between 12 to 30 h from ileocaecal valve to rectum.[15]

Peristalsis results in the propulsion of intraluminal contents over long lengths of small and large intestine. The process requires coordinated contraction of the longitudinal muscle and inhibition of the circular muscle ahead of the bolus and simultaneous longitudinal muscle relaxation and circular muscle contraction immediately behind the bolus. It can occur in an isolated segment of gut *in vitro*, implying it is dependent on an intact integrative enteric nervous system.[16] Activation of the longitudinal muscle ahead of the bolus is produced by acetylcholine released from the myenteric plexus. Relaxation of the circular muscle is produced by increased discharge from the intrinsic inhibitory neurons. Contraction of the circular muscle behind the bolus may be a myogenic event following synaptic shutdown of continuously active inhibitory neurons, or it could be produced by cholinergic excitatory input to the muscle, or both.

The best evidence for intrinsic inhibitory mechanisms is in Hirschsprung's disease where there is spasm in an aganglionic section of colon. As no reflex relaxation is initiated, the aganglionic segment behaves like a sphincter, resulting in a functional obstruction to faceal transit. The failure of relaxation of the diseased segment is accounted for by its lack of inhibitory neurons containing VIP and NO.

Parasympathetic supply to the colon, rectum and anus

Parasympathetic outflow to the colon contains two types of preganglionic axons. One leading to intramural cholinergic excitatory neurons and one connecting with intramural noncholinergic nonadrenergic inhibitory neurons.

Stimulation of the pelvic nerves elicits a contraction in both colonic muscle layers, but if the cholinergic fibres are blocked with atropine, parasympathetic stimulation results in inhibition of contraction and relaxation due to muscle cell hyperpolarisation. In humans, this can be seen when sectioning of the pelvic nerves may result in impaired defecation.[17]

Distribution of vagal input is debated;[17] it may project as far as the rectum, although some authors feel only the ascending colon is innervated, and others, up to the transverse colon. It acts to increase contractile activity by modulating coordinated motor responses from the enteric nervous system.

Sacral input is transmitted by pelvic nerves to the large intestine. It issues from spinal roots S_2-S_4 via the pelvic plexus (Figure 2). It is mediated by nicotinic cholinergic receptors and functions to reinforce mass contraction of the terminal large intestine during defecation.[18]

The parasympathetic supply to the internal anal sphincter comes from the sacral spinal cord through the pelvic nerves. The parasympathetic effect is sphincteric relaxation through activation of nonadrenergic noncholinergic intramural neurons and a presynaptic action of cholinergic intramural neurons on sympathetic nerve endings.

Extrinsic parasympathetic reflexes

Mechanical stimulation of the colon or rectum elicits a coordinated reflex contraction of the rectum that may result in defecation. The sacral parasympathetic centre is of major importance in organising colonic motility, especially during defecation.[17] It is able to organise defecation even after upper spinal cord section.[19] However the sacral parasympathetic centre is also influenced by supraspinal nervous structures. In the dog, the firing of sacral parasympathetic neurons is modulated by the pontine reticular formation.[20]

Parasympathetic preganglionic neurons are also influenced by afferents from both the urinary bladder and colon. The firing of parasympathetic neurons to the colon is increased by activatin of colonic afferents and decreased by that of vesical afferents that reciprocally inhibit colonic parasympathetic neurons and excite vesical ones.[21]

Sympathetic supply to the colon, rectum and anus

Sympathetic supply consists of cholinergic preganglionic neurons and noradrenergic postganglionic neurons located in either the sympathetic chain or prevertebral ganglia (coeliac, superior mesenteric, or inferior mesenteric). Preganglionic axons from spinal roots T_9-T_{10} synapse in the coeliac and superior mesenteric ganglia. From there, postganglionic nerves innervate the small intestine and from the superior mesenteric ganglion to the ascending and transverse colon (Figure 2). Sympathetics are inhibitory and function to decrease bloow flow and slow motility by relaxing the colonic wall to increase compliance. Sympathetic

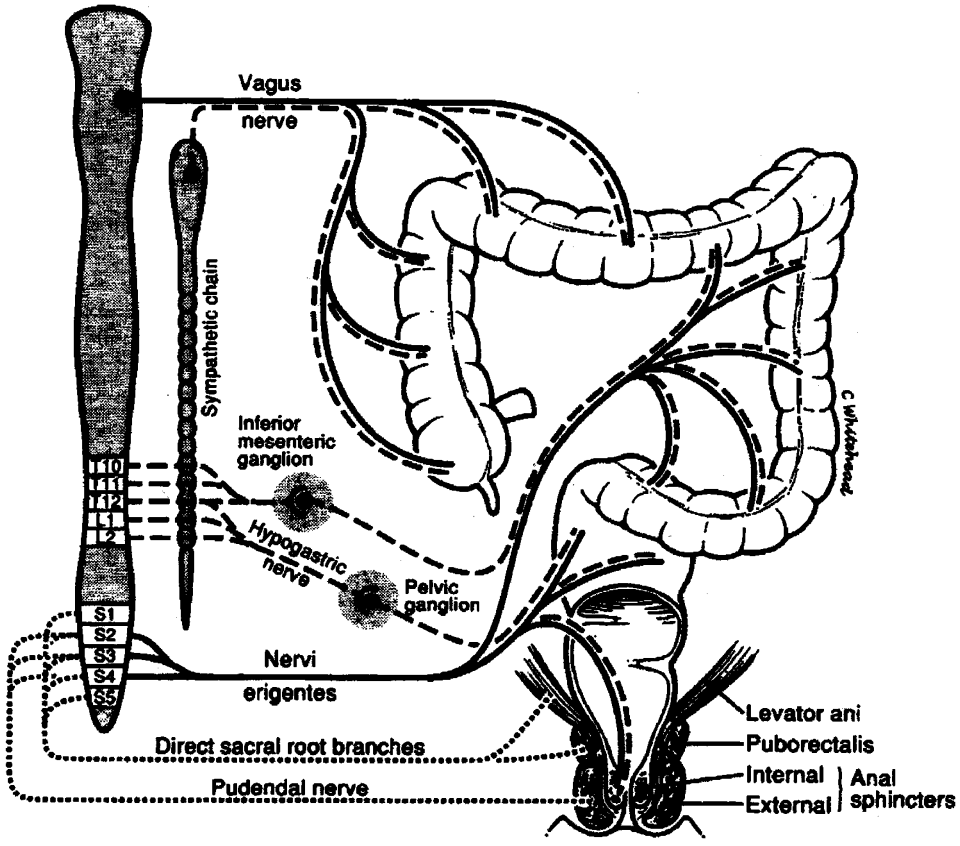

Figure 2 Autonomic and somatic innervation of the colon, anal sphincters and pelvic floor. Spinal cord segments and nerve branches are illustrated. Dashed lines represent sympathetic pathways with prevertebral ganglia. Solid lines depict parasympathetic pathways that synapse with ganglia in the enteric nervous system within the colonic wall. Dotted lines represent mixed nerves supplying somatic musculature of the external anal sphincter (EAS) and pelvic floor[15]

input acts on presynaptic terminals to prevent excitatory transmitter release within the enteric nervous system.[18]

There is evidence that the spinal cord is the source of tonic inhibition. In cats, after lumbar ventral root removal and spinal cord removal, there was a decrease in efferent lumbar colonic nerve firing and a parallel increase in colonic motility.[22] This suggests efferent inhibition from between T_{12}-L_5. This may not be the only source of efferent traffic in the colonic nerves as there are tonically discharging neurons in the cat inferior mesenteric ganglion.[23]

Somatovisceral reflexes have been described such as inhibition of intestinal motility in response to skin pinching. It appears to have a segmental organisation but its role in spinal patients is unclear although defecation appears enhanced by rubbing arms and abdominal massage. This may indicate the presence of somatic afferent pathways connected to vegetative central neurons involved in gut motility, perhaps via the sympathetic chain. However, patients who have had a complete bilateral sympathectomy do not generally present with change in bowel habit.[24]

Postganglionic sympathetic neurons in the prevertebral ganglia are the sympathetic final common path for the integration of information from the periphery and the CNS. Their function is complicated. Immunocytochemical data indicates that the nerve processes contain VIP, encephalin, SP, somatostatin, CCK and bombesin.[25,26] Each of the nerve trunks connected to ganglia contain specific peptidergic pathways.[25] These substances play a role in synaptic transmission within ganglia or as modulators of neurotransmitter release.[26]

SP at low concentration has been shown to increase the excitability of guinea pig prevertebral ganglionic neurons such that a subthreshold EPSP (excitatory postsynaptic potential) will generate a spike potential or give rise to tonic discharge of spike potentials in postganglionic neurones in the absence of excitatory input. Moreover, administering either SP, one of its analogs, or a SP-degrading endopeptidase inhibitor to rats will result in a significant increase in gastrointestinal transit.[27]

Sympathetic innervation of the internal anal sphincter

The IAS receives sympathetic innervation from the lumbar spinal cord through the lumbar splanchnic nerves and from the inferior mesenteric ganglion through the hypogastric nerves. The sympathetic effect is excitatory, and tonic discharge maintains IAS closure. With rectal distension or digital stimulation, the IAS relaxes probably due to the activation of intramural inhibitory nonadrenergic noncholinergic neurons. There may be some extrinsic participation since pelvic nerve stimulation induces sphincter relaxation.[28]

Vesicoanal reflex

Vesical filling increases electromyelographic activity in the IAS, and after voiding it decreases. The reflex is probably partly from the lumbar spinal cord and partly from the inferior mesenteric ganglion. Sphincter pressure is unchanged after high cord section, implying supraspinal centres have very little influence on IAS tone. A dual origin for excitation is proposed in the cat as IAS electrical activity remains after section of sympathetic excitatory fibres, but it is desynchronised. The implication is that motor activity is both myogenic and neurogenic, the latter reinforcing the former by synchronising electrical activity of smooth muscle cells.

Sympathetic innervation of the external anal sphincter

The EAS is a striated muscle innvervated from the sacral cord via the pudendal nerves (S2-S4). Its structure is shown in Figure 1. Some of the EAS motor neurones can be activated by stimulation of afferent axons in pudendal nerve branches from the external urethral sphincter. Clinical experience with patients following spinal cord injury does not seem to demonstrate a change in anal tone. This may be because supraspinal centres do not influence sphincter tone, the adaptation of remaining structures, or that a single level sympathectomy is insufficient to result in change.[29]

Chronic changes in colonic function following spinal cord injury

The extrinsic nervous system acts to modulate the intrinsic system. It is these complex integrated activities that require reflex and voluntary muscular control such as defecation that are affected most by spinal cord injury. Peripheral nerve lesions have been shown to result in transsynaptic degeneration and Schwann cell proliferation in the colonic wall, but local synaptic remodelling after spinal injury has not been demonstrated.[17,30]

The level of the spinal cord lesion determines the effect on colonic motility. A supraconal (Upper Motor Neuron or UMN) SCI results in loss of conscious sphincter control and an inability to significantly increase intraabdominal pressure. There is a loss of voluntary control of defecation and a degree of anorectal dyssynergy. This means that straining and rectal contraction result in increased tone in the EAS. Loss of rectal sensation and a spastic EAS require defecation to be anticipated. Reflex defecation can be initiated by mucosal stimulation, either digitally or with a suppository. Stimulation activates the rectoanal inhibitory reflex. It can be exploited to cause relaxation of the IAS, reflex relaxation of the EAS and pelvic nerve mediated peristalsis. If there is no reflex relaxation of the EAS complex, reflex evacuation

will not occur or be incomplete. Manual evacuation or enemata are often required in this situation.

Complete or partial injuries to the cauda equina result in a lower motor neuron (LMN) pattern of injury. The EAS and pelvic muscle are flaccid and there is no reflex response to increased intraabdominal pressure. The loss of parasympathetic control and reflex innervation of the IAS means a further reduction in resting anal tone and leads to faecal incontinence. A person with a LMN lesion following SCI will have absent EAS tone and decreased reflex peristalsis. Valsalva can result in faecal leakage and the rectum has to be kept empty to avoid faecal incontinence. Stool has to be removed digitally, assisted by Valsalva and abdominal massage.

Patterns of gut dysmotility have been described for different levels and degrees of SCI. Lesions above T1 result in delayed mouth-to-caecum time, but lesions below this exhibit normal transit times to the caecum.[31] Beyond the ileocaecal valve, transit times are markedly delayed. A LMN injury from a lesion affecting the conus, cauda equina or pelvic nerves results in interruption of the parasympathetic supply to the colon and reduced spinal cord-mediated reflex peristalsis. Stool propulsion is by segmental colonic peristalsis only. An UMN lesion results in variable changes in colonic transit. Marker transit studies and scintigraphy have demonstrated that patients with spinal cord lesions above the lumbar region have slowed transit throughout the whole colon.[32] One study involving 28 SCI patients also demonstrated distal small bowel dilatation in 10 patients, all of whom had abdominal symptoms and nine of whom had a spinal cord lesion above T5.[33] Radioisotope scintigraphy has shown the delay to involve the whole colon. The velocity of the median position of bowel contents throughout the colon was significantly slower in SCI patients (0.63 ± 0.33 cm/h in SCI, 2.58 ± 1.2 cm/h in controls, $P < 0.001$).[32]

The delay in transit may in part be due to loss of colonic compliance. The colon of patients with complete thoracic injury has been shown to have an abnormal response to increasing volume. Distension with water to generate a volume/pressure curve (colometrogram) produces a hyperreflexic response similar to that described in the bladder.[34] With a spinal cord lesion above L1, the left colon is less compliant. Above T5, the right colon is also affected. The lack of compliance leads to functional obstruction, increased transit times, and abdominal distension, bloating and discomfort. It suggests that the CNS is necessary to modulate colonic motility.[35] Colonic myoelectric activity has been recorded in a group of SCI patients with injuries at varying levels and controls. This demonstrated a significantly higher level of basal colonic activity in SCI patients (12.6 spikes per 10 min *vs* 3.3), and no demonstrable gastrocolic reflex.[36] This would support the assumption that the CNS exerts a tonic inhibitory influence on basal colonic activity and is consistent with the hypertonicity seen on colometrograms.[34]

The conclusion is that the increase in colonic activity and decrease in compliance may be due to loss of descending inhibitory input from the CNS. This is supported by animal studies in the cat where sympathetic nerve activity via α_2-adrenergic receptor activation resulted in profound inhibition of colonic motility,[37] and sectioning of preganglionic splanchnic sympathetic nerves produced an increase in colonic contraction.[38]

Effects of obstruction on motility and colonic neuropeptides

If the change in colonic motility was due to functional rectal obstruction secondary to anorectal dyssynergy alone, we should expect changes such as those produced by mechanical obstruction of the colon. Bowel muscle and ganglia hypertrophy, and if the obstruction is not removed, hypertrophy and contractile efforts continue until enzymatic mediator depletion occurs.[39] Non-constricting obstructive bands in rats produces histological changes such as ganglion cell elongation, probably due to bowel distension, but does not result in significant changes to the enteric nervous system.[40]

That said, obstruction has been shown to affect neurotransmitter levels.[41] One study looked at five patients with decompensated ileus due to tumour obstruction of the large bowel. Immediately after resection, samples were taken 10 cm proximally and distally to the tumour and SP and VIP levels were obtained by radioimmunoassay. A significantly decreased tissue level of both SP and VIP was found in the prestenotic sample. The mechanisms that lead to this decrease are unknown. Increased release of neurotransmitters due to endotoxins may be important. Decreased SP would impair motility by reducing bowel contractility. VIP may act by causing dysmotility due to loss of descending peristaltic inhibition. While the mechanical obstruction may be the major cause of obstructive ileus, the final rapid decompensation may be due to alteration of gut neuropeptides.[41]

Role of neuropeptides in bowel dysfunction

There is a distinction in the intramural distribution of regulatory neuropeptides within the bowel wall. SP is exclusively localised in nerves. Large quantities of SP and VIP are present in the lamina propria which is in close contact with the epithelium and muscularis mucosae.[42] Large numbers of VIP and SP-containing enteric nerves supply the ganglionated plexuses and are especially numerous in the circular muscle layer. They have a role in colon motility while those supplying the mucosa are involved with electrolyte and fluid transport. SP has been shown to accelerate the transit of a charcoal meal in rats.[27] It increases intraluminal pressure mainly by circular muscle contraction by direct action on the muscle as well as by simultaneous activation of excitatory cholinergic

pathways and inhibitory VIP-independent, NO-regulated pathways.[43]

SP is reduced in the colonic mucosa of patients with chronic constipation and increased in patients with ulcerative colitis. In both cases there was a significant correlation between mucosal SP levels and the disease states.[44] The elevated levels in ulcerative colitis may be a reactive effect secondary to other inflammatory factors. The fact that mucosal SP levels are associated with two disorders associated with colonic transit suggests a role in the pathogenesis of intestinal transit disorders.

A similar scenario exists with diabetic constipation where SP in the rectal mucosa of diabetics with constipation is significantly lower than in diabetics with normal bowel function.[45] Both groups of diabetic patients exhibited greater SP levels than controls. These abnormalities the mucosal content of SP may be the result of degenerative changes in the submucosal plexuses of diabetics.[46]

While mucosal SP may be decreased with chronic constipation, concentrations in the muscle layers may be increased. Sjolund *et al*.[47] examined tissue from the colon of 18 people with slow-transit constipation. Tissue concentrations of VIP and SP were measured by radioimmunoassay. Significantly increased concentrations of VIP and SP were found in the ascending colon, and in the descending colon, SP was increased in the myenteric plexus.

Dysfunctional colonic motility in children can result in severe constipation. A spectrum of dysganglionoses has been identified of which Hirschsprung's Disease (HD) is a subgroup. There are morphological differences between groups but the functional implication of this is unclear. One subgroup has been identified from immunofluorescence studies of neurotransmitters in full thickness colonic biopsies in children with colonic constipation.[48] These children have a markedly reduced number of SP-immunoreactive nerve fibres in the muscularis propria. This reduction in excitatory nerve fibres may be the basis of their functional impairment.[49] Intestinal Neuronal Dysplasia is a malformation of the enteric plexus that clinically resembles HD. Its pathogenesis is unknown, but patients appear to have defective innervation of the neuromuscular junction that results in chronic constipation.[50]

Enteric neurotransmitters such as SP and VIP appear to have a role in disease states affecting colonic transit. If this scenario is applied to colonic transit following SCI, impaired colonic transit may be reflected in abnormal concentrations of SP or VIP in the colonic muscle layers. This may result from chronic obstruction secondary to anorectal dyssynergy, or chronic dysmotility secondary to a change in CNS and sympathetic neuromodulation.

Our recent preliminary work on colonic neurotransmitters shows no difference in SP, VIP or NSE in SCI patients and controls.[51] In this study, specimens were obtained from four SCI and seven control patients. No histological differences were found between colonic specimens from SCI and control patients. The ratio of SP and VIP to NSE was qualitatively similar for both SCI patients and controls. There is still considerably more work to be undertaken in this area and it may provide an interesting and possible novel means on intervention.

Relationship of anorectal and bladder function

There are similarities between the bladder and rectum in terms of function and behaviour following spinal cord injury. The dysmotility seen in spinal patients may be due to decompensation of colorectal smooth muscle following chronic colorectal distension, similar to detrusor decompensation seen with chronic bladder outlet obstruction.[29] Colonic compliance is reduced following thoracic SCI, and the uninhibited contraction of the bladder seen following distension produces a cystometrogram analogous to the colonic compliance curves. This perhaps reflects a similar interruption of ascending somatosensory and descending visceral pathways.[35]

We have explored a possible relationship between patterns of anorectal physiology and patterns seen with cystometrograms. 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. 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. We concluded that SCI patients have abnormal anorectal function, and that anorectal manometry results were able to be classified into four patterns relating to rectal pressure and sphincter tone in response to rectal distension. The patterns of anorectal manometry seen were similar to those in cystometrograms, however there is no definite relationship to bowel dysfunction.

Mechanisms for bowel dysfunction following spinal cord injury

Colonic dysmotility has been noted, with delayed colonic transit times and a loss of colonic compliance. It is more marked in those with complete cervical injuries and can result in constipation, abdominal distention and discomfort. This may be due to a loss of descending inhibitory modulation from the sympathetic nervous system. The observation that transit delays are more profound in higher injuries supports this assumption.

The intrinsic enteric nervous system appears intact. No histological changes have been demonstrated to

occur in colonic specimens from long-term SCI patients.[51] Nerve fibres containing the intrinsic neurotransmitters SP and VIP appear to be present in qualitatively similar amounts in SCI patients and control specimens.[51] The colon may therefore continue to function independently of CNS modulation after SCI.

The difficulties with defecation following high SCI could be the result of discoordinate anal sphincter function.[52] The normal synergistic activity of colonic smooth muscle and pelvic striated muscle is lost. The conus-mediated increase in EAS tone with increasing intraabdominal pressure was seen for all groups of SCI patients. The reflex relaxation of the IAS and EAS can be exploited in order to defecate.[52] It can also lead to faecal incontinence as the anal sphincter may relax at relatively low rectal volume. Lower SCI can result in faecal incontinence secondary to reduced sphincter tone that is unresponsive to changes in intraabdominal pressure. A loss of rectal tone results in a capacious rectum and a reliance on the manual evacuation of stool.

The relationship between colonic dysmotility and anorectal dysfunction is unknown. Does the functional obstruction caused by dyssynergic EAS contraction during defecation lead to a change in colonic motility? In constipated non-SCI patients correction of pelvic floor abnormality often corrects abnormal transit time.[53] Alternatively, does the colon fail to transport faeces to the rectum at a rate and volume sufficient to generate sphincter relaxation and defecation? Patterns of bladder and bowel dysfunction following SCI, demonstrate a common loss of coordination in reflex and voluntary muscle functions.[52]

Current management strategies

The approach to bowel management in the spinal injured patient should address specific issues such as faecal incontinence, constipation, and functional mobility. This must be within the context of the patient as a whole person and consider their cultural, social, sexual and vocational roles. A bowel care regimen needs to be generated that fits the person's long-term routine. The aim should be effective colonic evacuation without faecal incontinence or other complications. Regularity of evacuation prevents excessive buildup of faeces and impaction. Appropriate equipment, such as commode chairs and wheelchair able toilets, needs to be supplied for an adequate long-term bowel programme.

Dietary manipulation is important. Adequate water intake promotes transit by keeping the stool soft. Fibre is promoted to give the stool bulk and plasticity.[15] This is thought to assist colonic transit in neurologically intact patients, probably by promoting propulsive activity secondary to increased colonic wall distention. However, an increase in stool bulk may mean more time spent with bowel care, and increasing fibre intake for SCI patients has not been shown to result in faster colonic transit time. The main reason to have a high fibre intake therefore is to absorb excess water and keep the stool soft. Forty-three per cent of SCI patients use fibre sometimes or regularly, compared with only 23.2% of controls. Fibre use didn't change with time after injury and users were on average younger than controls.

Stool softeners other than fibre, such as docusate sodium (Coloxyl, [Fawns & McAllan]) increase the amount of water in stool without increasing volume and have no effect on bowel motility. They can also affect the intestinal absorption of other drugs, resulting in higher plasma levels. The stool is more likely to be liquid, so continence will not be improved. They are most useful where faecal incontinence is not a risk and straining is to be avoided, such as for those patients with haemorrhoids or autonomic dysreflexia.

Stimulant laxatives act by increasing intestinal motility resulting in less time for water reabsorption. Senna (Senokot, [Reckitt & Colman]) has a direct stimulant effect on the myenteric plexus and also increases intraluminal fluid. Bisacodyl (Dulcolax, [Boehringer Ingelheim]) has a similar mode of action and is often used as a suppository to initiate bowel evacuation. Dose dependent side effects can occur. These include abdominal cramping, diarrhoea and electrolyte imbalance. Chronic use of stimulant laxatives, especially senna, can result in a progressive unresponsiveness. Osmotic laxatives such as lactulose (Duphalac, [Duphar]) draw fluid into the colon. They can result in more liquid stool and cause cramping. Laxative use was almost ten times more frequent among the SCI patients, becoming even more frequent with increasing time from injury. This may suggest a greater incidence of constipation.

Enemata are often employed when suppositories or digital stimulation fail. Long term use can result in enema dependence and side effects such as rectal trauma and autonomic dysreflexia can occur.

Prokinetic agents such as cisapride (Prepulsid, [Janssen-Cilag]) have been employed to reduce constipation in SCI patients. Transit times are improved, however, cardiac arrhythmias have been noted with long-term use.[54]

SCI patients with UMN lesions can exploit the rectocolic reflex to effect defecation. Digital stimulation can result in a reflex wave of conus-mediated rectal peristalsis. The intact rectoanal inhibitory reflex (RAIR) then causes IAS relaxation and defecation. Rectal sensation is reduced however, so defecation has to be anticipated on a regular basis. Based on the anorectal manometry data[52] it is possible to identify those SCI patients who will reflexly defecate at low rectal volume. These patients require a bowel management programme that keeps the rectum empty to reduce the incidence of incontinence. Those patients with UMN lesions and an obstructed defecatory pattern need a management plan that mimics anorectal trauma but still allows adequate rectal evacuation to avoid constipation.

Patients with LMN lesions have an areflexic bowel and reduced sphincter tone. The increase in sphincter pressure with Valsalva is reduced, leading to increased risk of incontinence, especially with liquid stool. The aim is therefore to keep stool consistency firm. Local anorectal reflexes are often insufficient to result in defecation, and a compliant rectum acts as a large reservoir, so stool is digitally removed.

A Brindley sacral anterior nerve root stimulator (S2–S4) can be used for electromicturition to achieve regular, complete bladder emptying. Often deafferentiation of the sacral posterior nerve roots is performed before the stimulator is implanted to produce detrusor areflexia and interim urinary continence. The deafferentation also results in loss of the sacral reflexes necessary for defecation. The stimulator can be used to initiate defecation, not during stimulation, due to simultaneous rectal and sphincter contraction, but when stimulation stops and the EAS relaxes instantaneously and the rectum relaxes slowly, resulting in spontaneous defecation. This has been shown to result in quicker, more controllable defecation than the reflex method.[55,56]

Colostomy may be an alternative for SCI patients with ongoing bowel management difficulties. It has been shown to result in improved quality of life, it is accessible for those with poor hand dexterity, and offers control of faecal incontinence. Risks and complications that are associated with any surgical procedure may not make it acceptable to all SCI patients. A recent study by us has revealed that patients with colostomies do not have their quality of life impaired.[57] A standardised previous validated question designed to assess quality of life in spinal injured patients was sent to 26 spinal cord injured patients with colostomies and 26 spinal cord injured patients without colostomy. The two groups were matched for level of injury, completeness of injury, length of time with injury, age (± 5 years) and gender. There was no significant difference ($P > 0.05$) in the two groups of patients in regard to their general wellness, emotional, social, or work functioning. Further prospective studies are needed and we have one underway at present.

The antegrade continence enema (ACE) procedure is now widely accepted as an option in the treatment of faecal incontinence in children who have not responded to medical management.[58,59] The operation involves attaching the appendix to the surface of the abdomen as a catheterisable channel to enable regular antegrade colonic washouts. There have been a number of modifications to the ACE procedure including non-reversal of the appendix,[60] various skin flap techniques,[61] a simplified laparoscopic technique (LACE),[62,63] and the use of the procedure in older patients.[64] The procedure appears to have a low incidence of complications and morbidity. It requires a motivated patient with good hand function and is generally considered best in patients with a lax anal sphincter. In a recent study we have undertaken children with spina bifida who had the LACE procedure, there was a consistently high level of functional continence achieved following surgery.[65] The use of the procedure in spina-bifida has shown that this procedure may have an as yet unidentified role in SCI patients.

Future objectives for the investigation and management of bowel dysfunction

The SCI patients with bowel management problems are easy to identify. Interview and clinical examination can generate an impression of their general bowel function and identify problems such as constipation, faecal impaction, anal fissures, and haemorrhoids. Simple tests of anorectal function are available. These could be performed on all SCI patients in the same manner that bladder dysfunction is investigated. Anorectal manometry will identify those with dyssynergic sphincter function. For those with abdominal bloating and constipation, abdominal X-ray and colonic motility studies give further useful information.

After 12 months from SCI, bowel function does not seem to change either with increasing time after injury, or increasing age. This allows for early identification of what may be ongoing problems.

After the acute phase of spinal cord injury, prompt identification of those with bowel management problems would be useful. Anorectal manometry could then be performed. Specific patterns identified may help determine future management.[52] Those with a compliant rectum and reflexly relaxing sphincter can employ digital stimulation to reflexly defecate. Patients with high rectal compliance (pattern 4) may require regular manual evacuation to ensure their compliant rectum is empty. Patients with increased rectal and sphincter tone (pattern 1) will probably have high injuries and need to be identified because this pattern may result in inadequate rectal evacuation. Anecdotally, these would be the patients where manual evacuation is difficult and often accompanied by autonomic dysreflexia.

Following SCI, the colonic nervous system may still be intact but functioning in an ummodulated way. The existence of SP analogues and inhibitors of SP degradation that are reactive *in vitro* and *in vivo* have already been used to demonstrate a reduction in colonic transit time in rats.[27] These compounds are useful tools to further investigate the actions of colonic neuropeptides. Increasing coordinated colonic peristalsis would be a useful therapeutic modality. This could be done either by direct stimulation of colonic smooth muscle or by stimulation of SP and VIP immunoreactive nerve endings.

Although improving colonic motility and appropriate bowel management may help, there will be some SCI patients with ongoing bowel problems. Colostomy formation has been used to provide the patient with relief from constipation and anorectal dysfunction and an independent means of managing their own bowel function. Further research needs to be done into the

differences in quality of life and bowel function following colostomy formation in SCI patients.

Another focus of future investigation is the intrinsic control of colonic motility. The findings of our work on colonic neurotransmitters imply that the intrinsic innervation of the colonic smooth muscle is intact (with respect to NSE, SP and VIP) following SCI. The way this muscle functions without spinal cord modulation may form the basis for some of these problems seen clinically, specifically, problems with colonic motility and rectal compliance. Further investigation may confirm that the intrinsic colonic nervous system is intact. Such research could involve immunohistochemical staining for colonic smooth muscle neurotransmitters to support our initial findings, or performing passive and active length-tension curves on isolated colonic smooth muscle to confirm its functional similarity to normal muscle.

The bowel problems resulting from SCI are due to the loss of the complex integrative pathways involving intrinsic and extrinsic innervation. Investigation targeted at improving bowel function for these people has been shown to improve quality of life. Pharmacological manipulation of colonic innervation may become a therapeutic option. However, improvement in motility requires a coordinated response that may not be possible following SCI. There is still scope to provide early assessment of SCI patients in order to institute appropriate bowel management, and more active surgical intervention (colostomy and ACE) that may improve quality of life.

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