Spinal Cord Injury: Gastrointestinal Implications and Management

Frederick S. Frost

For persons with spinal cord injury, gastrointestinal disorders have a major impact on medical morbidity, maintenance of body image, need for caregiver support, and hospital discharge destination. Difficulties in achieving bowel continence have an impact on virtually every aspect of life; interfering with fulfillment of interpersonal, sexual, and employment roles. Strategies for management of SCI-related bowel disorders should take advantage of the considerable amount of normal, residual function that exists in the gastrointestinal tract, even after the most serious spinal cord injury. Key words: *gastrointestinal disorders, spinal cord injury*

GASTROINTESTINAL (GI) disorders rank seventh as a cause of death in persons with spinal cord injury (SCI[1]); dysfunction of this system has a profound impact on medical morbidity, maintenance of body image, need for caregiver support, and hospital discharge destination. More than one third of surveyed persons with paraplegia ranked the loss of bowel and bladder control as the most significant functional loss associated with their respective injury—more important than the loss of the use of their legs.[2] Difficulties in maintaining bowel continence can have an impact on virtually every aspect of life, interfering with fulfillment of interpersonal, sexual, and employment roles.

For persons with SCI, GI complaints are common, in both the acute and the long-term care setting. Studies of GI complications in patients with SCI have traditionally focused on the acute postinjury period. Gore et al[3] reviewed 567 admissions to an SCI center and identified ileus and gastric ulcer as the most common disorders of the digestive system during the first 4 weeks after injury. Over the next 3 months postinjury, morbidity was more likely to be attributable to lower tract dysfunction, with the most common problem being fecal impaction (n = 46).

More recent investigations attempted to assess the incidence and importance of GI problems over the long term, studying large outpatient clinic populations. Stone et al,[4] in a study of 90 SCI outpatients, found that 29% reported GI dysfunction that required medical intervention or altered their lifestyle. An increase in bowel-related problems was associated with complete neurologic lesions and a postinjury period of greater than 5 years. In a similar study, Albert et al[5] found that 6% of 1,100 SCI patients, during an average follow-up of 3.8 years, exhibited significant GI complications. Almost half of these complica-

Frederick S. Frost, MD, *is Assistant Professor of Medicine (Rehabilitation), Case Western Reserve University/MetroHealth Medical Center; and Project Director, Northeast Ohio Regional Spinal Cord Injury System, Cleveland, Ohio.*

Top Spinal Cord Inj Rehabil 1998;4(2):56–80

tions involved bleeding of the lower GI tract. For persons with chronic SCI, the deleterious effects of decades of suppository use, digital evacuation, and chronic constipation are just now being elucidated as more individuals present with declining satisfaction with their bowel function.

Nutrition

Although medical personnel agree about the importance of nutrition in the maintenance of good health, few appreciate the multiplicity of factors that serve as a deterrent to adequate dietary intake in the SCI population. It is particularly unfortunate to note that little attention is paid to these matters in the outpatient setting, particularly because poor nutrition is the most remediable of factors in the development of disease. In addition, nutrition often takes on a larger meaning than simple food intake; feeding may be seen as an important issue in independence or socialization or as simply a pleasurable sensory experience for an individual who is otherwise deprived of sensory input.

During the acute period, persons with SCI will invariably fall behind on protein intake in light of the huge protein losses associated with major trauma.[6] Despite protein supplementation, most SCI patients show evidence of poor assimilation of ingested protein over the acute injury period.[7] Such protein losses may be obligatory and not remediable.[8–10] When considering protein replacement based on urine urea nitrogen losses, it is impossible to provide the number of calories necessary to safely deliver such huge amounts of protein. Often this situation persists for several weeks after injury. At this point, the nutritional program is based on supplying calories sufficient to support the patient's basal metabolic rate (22.7 kcal/kg/d for tetraplegic patients and 27.9 kcal/kg/d for paraplegic patients).[11] In practice, careful resumption of oral or enteral feeding may be a major goal during the critical care period, as concomitant trauma to the GI tract or motility disorders associated with acute neurologic injury may preclude feeding. After the patient is discharged from intensive care, nutritional status may further decline for a variety of reasons.

Many patients discharged from intensive care may simply not feel well enough to eat. Most will find hospital diets (emphasizing carbohydrates and milk products) and meal times to be very different from their usual regimen. Although clinicians encourage the patient's family and friends to bring food from home, this practice is not without problems. Particularly in the pediatric and teenage population, increased vigilance is necessary to ensure that fiber intake is adequate and a balanced diet is maintained. Dental injury is common in association with SCI,[12] and weight loss may result in poorly fitted dentures. Arrangements for proper dental care constitute an important element in nutritional planning for this population.[13]

Often patients are constipated on admission to the rehabilitation setting, a remediable cause of nausea and poor appetite. Establishment of a regular bowel-emptying routine, correction of hydration deficits, and resumption of a higher level of activity will usually result in an increased appetite. For patients with tetraplegia at the C5 level and below, mealtime occupational therapy for training in feeding skills takes on medical and psychological importance.

The most important measures in nutritional assessment—the patient's weight and calorie count—are often not performed.

Whether because of issues of inconvenience or lack of proper equipment and personnel, the most important measures in nutritional assessment—the patient's weight and calorie count—are often not performed. Weekly weight measurements for newly injured persons with tetraplegia constitute an important standard of medical care. Calorie counts are especially valuable in this population, in that body weight measurements alone can be deceptive; severe malnutrition and tissue wasting can be masked by water retention. Sophisticated laboratory nutritional assessment of this patient population is rarely necessary or useful. Blood tests for serum creatinine, albumin, and hemoglobin, by themselves, are of questionable utility in this patient population,[14] and anthropometric measurements (skinfold thickness, muscle circumference) are confounded by neurogenic muscle atrophy.[15]

The maintenance of appropriate body weight is of obvious importance for the person with a chronic disability. Small increases in body weight can restrict physical mobility in persons with marginal physical function and can have devastating implications for those individuals prone to pressure sores.[16] In a study of persons with chronic SCI, Peiffer et al[6] established recommendations for calculation of ideal body weight, allowing for a decreased level of muscle mass. For persons with paraplegia, the recommended ideal weight was 10 to 15 lb (adjusting for body frame and build) below the standard calculated body weight for sex and height among nondisabled persons. For persons with tetraplegia, a decrement of 15 to 20 lb was allowed below standard calculated values for the nondisabled person. Persons falling 10% or more below these standards, with caloric and protein intake less than calculated maintenance levels, were considered at nutritional risk. In their series of patients, Peiffer et al found that 44% of persons in both tetraplegic and paraplegic groups were more than 10% below their ideal body weight, even after allowances had been made for their neurologic injury.

Swallowing

In the acute assessment of a person with cervical spine trauma, evaluation of the patency of the esophagus and the ability to swallow is of critical importance. As the posterior wall of the pharynx lies adjacent to the anterior aspect of the cervical vertebrae, severe cervical trauma may cause bony elements to impinge on or even perforate the pharynx or esophagus. Elderly patients with cervical osteoarthritis may develop esophageal dysphagia, even in the absence of trauma, as large anterior cervical osteophytes encroach on passage of a food bolus. Although the incidence of esophageal rupture in these patients is low, the index of suspicion on the part of the clinician must be high. The leakage of saliva and food into adjacent cervical tissue planes may result in the formation of a mediastinal abscess, a condition with disastrous consequences. Initial evaluation by endoscopy or water-soluble radiographic contrast study is recom-

mended if the clinical suspicion of perforation is present. Examination of a lacerated GI tract with barium sulfate is not recommended if mediastinal extension is a consideration.[17]

Although the neurologic mechanisms of swallowing should be unaffected even in high-level tetraplegic injuries, a variety of mechanical and medical causes of swallowing disorders may predispose the patient to aspiration of saliva or food material. Chiefly among these, the use of skeletal traction (usually holding the neck in forced extension) and the necessity for patient sedation may circumvent the basic components of airway protection. Because anterior cervical decompression and fusion procedures are gaining increased use in the acute setting, the swelling and local pain associated with these interventions, traction injury to the recurrent laryngeal nerve, and the soreness related to oropharyngeal airway use are potential causes of dysphagia.

Elderly patients and patients with premorbid psychiatric illness are at special risk for developing swallowing difficulties. A lack of somatic sensory input, increased pulmonary secretions, and forced supine positioning in the intensive care setting, combined with claustrophobia and disorientation, may result in panic episodes and the sensation of "drowning." For this reason, patients who have been advanced from tong traction to Halo vest immobilization must be observed carefully for the first few hours after application, and great caution must be exercised in making the decision to move the patient from the intensive care unit to a general nursing floor.

In the intensive care setting, interventions such as early tracheotomy and the use of a cuffed tracheostomy tube allow for better pulmonary hygiene and provide some airway protection against impaired swallowing. Inflation of the tracheostomy tube cuff, however, does not guarantee protection against aspiration of swallowed substances, as liquids may pass around the cuff. If aspiration prevention is the priority, larger, isobaric foam tracheostomy cuffs will create the best seal without increasing the risk of airway erosion over the short term.

Tracheostomy tubes (especially those attached to heavy ventilator tubing) may further affect swallowing in these patients. By applying traction to the neck, these devices may inhibit the normal upward translation of the pharynx necessary to complete the swallowing action. Stable ventilator tubing brackets are an important component of the bedside equipment setup, and an essential part of any wheelchair prescription for a ventilator-dependent tetraplegic patient.

Little systematic analysis of swallowing disorders in patients with high-level tetraplegia has been performed to date. In the evaluation of those with swallowing difficulty, there is preliminary evidence that the cricopharyngeal phase of swallowing is disrupted; this may be remediable by instructing the patient in the use of the Mendelsson maneuver.[18] The pathophysiologic explanation for this deficit is as yet undetermined.

Esophagus, Stomach, and Small Bowel

Although central nervous system input closely controls both ends of the digestive tract (the upper esophagus and external anal sphincter), the balance of the system can function fairly well in the absence of central nervous system input. Such autonomous activity is attributed to intrinsic reflexes medi-

ated by nerve ganglia found within the walls of the visceral structures. For this reason, in persons with SCI, the basic propulsive, secretory and absorptive functions of the digestive system are usually maintained. The fine-tuning of these actions, however, depends on the modulating influences of the autonomic nervous system and a variety of neurotransmitters and peptides.[19] An injury to the spinal cord can result in derangement of these secondary regulating factors, significantly affecting digestive function.

Gastroesophageal reflux disease

Stinneford et al,[20] in their 1993 study, found that symptoms of heartburn, esophageal pain, and intermittent dysphagia were much more common in persons with tetraplegia than in control subjects. Several factors may be present in the SCI patient that promote reflux disease (see box, "Factors Promoting Gastroesophageal Reflux in Persons with SCI"). Symptoms of reflux disease are usually nonspecific, and actual esophagitis may not necessarily be present. The authors found that 45% of symptomatic tetraplegia patients exhibited endoscopic evidence of esophagitis, whereas 91% had histologic evidence of esophagitis. In many instances, symptomatic therapy is instituted without endoscopic or histologic confirmation. Radiologic or endoscopic study is critical, however, when abdominal pain (rather than burning) or blood loss is present or when pulmonary aspiration is suspected. In addition, perfusion of acid into the esophagus through a catheter (Bernstein's test) can be carried out to determine whether such a challenge reproduces the patient's symptoms. The conventional assumption that sensory fibers supplying the esophagus are vagal in origin (and unaffected by SCI) has been arrived at through indirect evidence and animal studies. Thus, it is likely that acid challenge testing is reliable even in patients with high cervical cord lesions.

Factors Promoting Gastroesophageal Reflux in Persons with SCI

Medical

Recumbency and immobilization
Delayed gastric emptying
Increased gastrin and acid release
Irregular mealtime patterns

*Drugs**

Diazepam, theophylline, meperidine, anticholinergics, antispasmodics, tricyclic antidepressants, calcium channel blockers, nitrates

Miscellaneous

Cigarettes, fatty foods, chocolate, citrus juices, coffee

*These are a source of decreased lower pressure in the esophageal sphincter.

The treatment of reflux disease is directed at ameliorating the basic elements of this condition: poor esophageal acid clearance, impaired function of the lower esophageal sphincter, and increased acidity of stomach contents. Elevation of the head of the bed to 30 degrees is nearly always helpful, but use of skeletal traction and orthopedic stability may preclude this maneuver during the acute phase after SCI. As the patient's acute status stabilizes, dietary measures center on the restriction of fatty foods and reinstitution of a regular mealtime schedule, with moderation in size of meals. If such simple measures

are not of benefit, antacids of the alginic acid type can be given four times a day. Histamine-2 (H_2) antagonists may be used to reduce the acidity of regurgitated stomach contents. In the outpatient setting, cessation of cigarette smoking and restriction of caffeine and alcohol ingestion will result in resolution of symptoms without further therapy.

In those patients with documented esophagitis and refractory symptoms, bethanechol (25 mg four times daily) or metoclopramide (10 mg three times daily) may augment esophageal clearance while elevating lower esophageal sphincter pressures.[21,22] SCI patients, having been found to exhibit slowly propagating abnormal (double-peaked) peristaltic esophageal contractions,[18] may further benefit from newer pharmacologic agents that affect gut motility through noncholinergic mechanisms (eg, cisapride, domperidone). These agents have been studied in this population.[23–25] In refractory cases, surgery can be performed in hopes of restoring esophageal sphincter competence.

Impaired gastric emptying and ileus

The clinical observation that gastric distention and ileus commonly occur after acute SCI is not surprising, as impairment of GI motility may be associated with major trauma, immobilization, or recumbency. Although there is no evidence that central nervous system injury, by itself, causes impairment of transit through the stomach and small intestine, medical conditions associated with SCI no doubt have a significant impact on such processes. Intestinal obstruction is a frequent complicating factor after acute SCI and is a common cause of rehospitalization of patients in the chronic stage of disability.[3] The relative importance of the neurologic injury, with potential alteration in autonomic control of the GI tract, has been assessed by direct and indirect means.

Gut motility is no doubt influenced by general health status, diet, physical activity, electrolyte disturbances, and a variety of drugs commonly used in this population.

Persons with chronic spinal cord transection above the T1 level have demonstrated prolonged gastric emptying and abnormal propagation of propulsive motor complexes in the stomach and duodenum, when compared with control subjects with lower spinal cord lesions.[26,27] This observation is interesting from both a clinical and pathophysiologic standpoint, as it implicates the thoracic sympathetic outflow (T5–T10) as an important contributor to the digestive processes of the stomach and duodenum. Further confirmation of the relative importance of neurologic injury was provided by Peschiera and Beerman,[28] who found a low incidence of intestinal dysfunction in patients who had sustained spinal fractures without neurologic injury.

The role that neurologic injury plays in the development of motility disorders in persons with SCI may be overstated, as the underlying mechanisms behind the development of GI motility disorders are not well understood and are certainly multifactorial. Gut motility is no doubt influenced by general health status, diet, physical activity, electrolyte disturbances, and a variety of drugs commonly used in this population. The metabolic and endocrine changes associated with SCI[29] may also play a role.

Gastric emptying problems may present a wide range of signs and symptoms. Not infrequently, a stomach distended with swallowed air will be noted on a plain chest radiograph during the acute phase of SCI. This finding is always important; such distention can restrict pulmonary ventilation and promote the development of atelectasis. In addition, these abnormalities in gastric emptying have been shown to decrease the bioavailability of orally administered drugs.[30] Most importantly, the presence of gastric distention may herald a more serious GI disturbance and the development of ileus.

Intestinal obstructions of a nonmechanical nature are often termed *adynamic* (or *paralytic*) *ileus* or *pseudo-obstruction.* The impact of spinal shock (absence of somatic reflexes) on the GI tract is unclear; signs and symptoms of adynamic ileus are commonly seen, regardless of the level or completeness of neurologic injury. Adynamic ileus can occur solely as a response to a major trauma, medication/anesthesia, recent surgery, or infection. Although symptoms may mirror those seen in mechanical intestinal obstruction, they are typically less striking; adynamic ileus tends to follow an indolent course, developing gradually over 3 to 4 days. In the acutely injured patient, auscultation for bowel sounds must be carried out carefully and frequently; bowel sounds may appear and disappear, frustrating the clinician attempting to start or advance feeding. On plain films of the abdomen, dilation is commonly present throughout the stomach, small intestine, and colon. Every effort should be made, despite orthopedic restrictions, to obtain upright or lateral decubitus films to determine whether air–fluid levels are present.

Abnormalities in serum electrolytes and fluid status may occur either as a cause or an effect of adynamic ileus. Once ileus occurs, several liters of plasma volume may be sequestered into the edematous intestinal wall, making restoration of fluid balance difficult. Most often this picture is associated with hyponatremia and hypochloremia.

The clinician who is treating adynamic ileus in a person with acute SCI must be attentive and patient. In most cases the cause of the condition is multifactorial; with resolution brought about by careful restoration of electrolyte balance and general supportive measures. Intestinal decompression through nasogastric suction is indicated and should be maintained until symptoms improve and plain abdominal radiographs begin to show resolution of the abnormal gas patterns. The reinstitution of oral or enteral feeding is likely to be successful if

- consistent (not intermittent), normal-pitched bowel sounds are apparent on examination
- nasogastric output diminishes
- stool or gas is passed spontaneously from the rectum and
- abdominal radiographs show normalization of bowel gas patterns.

Surface electrical stimulation has been shown to increase upper and lower GI motility and may aid in resolution of ileus during the acute period.[31] Whenever possible, feeding tubes should be placed distal to the pylorus, thereby bypassing the impairment in antral/duodenal sweep noted early after SCI.[24]

Upper GI tract symptoms of nausea, epigastric distention, and anorexia often persist into the rehabilitation phase after injury and are too nonspecific to be diagnostic in most cases. The presence of vomiting, especially

when it occurs an hour or more after meals or medication, is particularly worrisome and usually warrants further investigation and intervention. Before embarking on a therapeutic plan, it is important to determine that gastric obstruction is not present. Gastric outlet obstruction occurs most commonly as a result of peptic ulcer disease and may occasionally occur as the initial presentation of an ulcer. Less commonly, mechanical blockade may be caused by local tumor, pancreatitis, or inflammatory bowel disease.

A plain radiograph of the abdomen is the first step in evaluation of these patients, primarily in determining if free air is present in the peritoneal cavity, a sign of gastric perforation. Fecal impaction can be quickly ruled out in this fashion. A nasogastric tube is necessary if vomiting is uncontrollable and will often provide objective evidence of obstruction. A saline load test can be performed by instilling 750 mL of isotonic saline into the stomach. The removal of more than 400 mL of fluid 30 minutes thereafter supports the diagnosis of obstruction. An upper GI series or radionuclide scintigraphy[32] will further outline the problem.

A large percentage of patients with noncancerous obstruction will respond to conservative medical management, with emphasis on gastric decompression (by nasogastric suction), correction of fluid and electrolyte disorders, and nutritional support. Persistent output of large gastric volumes after 72 hours of nasogastric suction is a poor prognostic sign, indicating that surgical correction may be necessary.

If a physical obstruction is not present, a delay in gastric emptying, manifested by early satiety, nausea, and belching, commonly resolves with conservative care. These patients may benefit from approaches utilized in persons with emptying disorders secondary to other conditions (eg, diabetic gastroparesis, vagotomy, viral illnesses), although no careful evaluation of efficacy of these methods has been carried out in patients with SCI. From a pharmacologic standpoint, metoclopramide (10 mg 30 minutes before meals) is the best studied agent available[33] but is of little benefit when used over the long term. If these symptoms persist after initial hospital discharge, dietary management constitutes the most effective therapy. A change to small portions of soft cooked foods, chewed well, will almost always result in improvement of symptoms. For tetraplegic patients with severe and chronic gastric distention, placement of a gastrostomy tube for gaseous decompression has been shown to be effective and well tolerated.[34]

Just as in the case of impaired gastric emptying, intestinal obstruction, seen in the rehabilitation phase of SCI, may be secondary to mechanical blockage or to disorders of bowel motility. Because persons with SCI may have impairment of pain appreciation, obstruction may initially be manifested only by nausea, obstipation, and abdominal distention. If the blockage is distal in the colon, vomiting may not be seen until very late in the clinical course. On examination, bowel sounds that are frequent and high pitched (suggestive of an early mechanical obstruction) or completely absent indicate a need to take action. Liberal use of diagnostic testing is necessary, as plain abdominal radiographs and serum electrolyte determinations are critical in establishing a diagnosis.

Mechanical obstruction of the small bowel has been reported in the postacute stages of

SCI.[35] A clear demarcation between proximal dilated bowel and distal collapsed bowel may be seen on plain radiographs. The treatment for such lesions is almost invariably surgical, and the cause for the obstruction (eg, tumors, adhesions, hernias) is rarely determined prior to surgical exploration.

As an exception, the superior mesenteric artery syndrome, has been reported to occur with frequency in persons with tetraplegia.[36] This condition has a characteristic prodrome, as such patients exhibit epigastric pain (especially in the supine position) and frequently vomit large volumes. A characteristic picture is seen on contrast fluoroscopy: a cutoff between the third and fourth portions of the duodenum is demonstrated in the supine posture that disappears with the subject in the upright posture. This condition is usually seen in patients who have experienced significant weight loss and is probably caused by a loss of the mesenteric fat that serves to shield the duodenum as it passes behind the superior mesenteric artery and in front of the lumbar spine and aorta. This type of mechanical obstruction is also exceptional in that it may respond to medical management (ie, upright body positioning and weight gain).

Long after rehabilitation discharge, chronic neglect of proper bowel emptying and poor general health may predispose the patient to the development of intermittent episodes of ileus, more appropriately termed *secondary intestinal pseudo-obstruction*. Although primary pseudo-obstruction, a devastating and rare global disorder of bowel motility, is probably an inherited disorder, secondary pseudo-obstruction relates to *recurrent* episodes of ileus that occur as a response to a chronic condition (eg, collagen vascular disease, drug effects, infections, neurologic motility disorders). Most commonly, this is seen in persons with chronic SCI who may be hospitalized (eg, for urosepsis, pneumonia, or failure to thrive), exhibiting symptoms of abdominal distention and signs of ileus on plain abdominal radiograph. Treatment of the underlying medical condition is the key, although management may be made more difficult because of the coincident presence of large amounts of stool in the colon.

In most cases this clinical picture involves an acute decline in health status combined with the effects of hospitalization of a patient with poor baseline fitness. Admission to the hospital, by itself, will take the patient away from his or her standard activity, dietary, and bowel-emptying routine. Health care providers often do not anticipate the need for artificial bowel-emptying programs during hospitalization. With the appearance of GI symptoms, abdominal radiographs are obtained, and the discovery of large amounts of stool in the colon, a normal finding in patients with chronic SCI, often leads to the incorrect conclusion that fecal impaction is the primary problem. This, in turn, will lead to therapeutic maneuvers that may make the situation worse rather than better. Strong oral cathartics may be given to relieve the "impaction," an action that serves only to fill a poorly motile GI tract with more gas—thereby worsening symptoms and producing further dilation of the small bowel notable on radiographs. In some instances GI perforation can occur.[37] Fortunately, in most cases treatment of the primary medical illness is successful and general supportive measures result in a return of intestinal motility, and the pseudo-obstruction resolves despite the "treatment." Of course, there will

be instances where fecal impaction is indeed the causative factor of intestinal obstruction. Management of this condition is described below.

Abdominal pain and the acute abdomen

The evaluation of abdominal pain in persons with SCI is fraught with difficulties. Many patients will exhibit impairment of visceral sensation, thereby eliminating many of the clues to diagnosis often gained by history and physical examination. In the presence of an obstruction, perforated ulcer, or ruptured viscous, abdominal discomfort may not be present until the inflammatory process has reached the peritoneum of the upper abdominal quadrants, where afferent enervation arises from the upper cervical segments. In acute SCI, laparoscopy is often necessary to rule out the possibility of concomitant injury to the viscera.

In the outpatient clinic setting, acute abdominal complaints are too often incorrectly ascribed, early in their course, to pyelonephritis or constipation. In the emergency department, mild leukocytosis and pyuria are nonspecific findings in many SCI patients. The presence of chronic constipation puts these persons at even greater risk for acute abdominal disorders, as it results in a higher incidence of diverticuli and rectal fistulas, increasing the likelihood of bowel perforation.[38] Immobilization and atherosclerosis in older patients with SCI may predispose these patients to the development of mesenteric vascular thrombosis or infarct. Gallbladder disease is especially common in these patients, probably as a result of dysmotility.[39] The predilection toward the development of biliary tract disease, combined with a high incidence of previous abdominal trauma and alcoholism in many patients, presents a clinical picture that is favorable for the development of acute pancreatitis. As a diagnosis of exclusion, neurogenic abdominal pain must also be considered. A burning or lancinating pain, localized to the border between sensate and insensate skin (often described as a "tight belt"), may represent a neurologic, dysesthetic pain syndrome. Persons with lumbar lesions frequently describe severe burning pain in the rectum or perineum. Often no associated GI pathology can be found.

The clinician must be particularly vigilant when confronted with vague abdominal symptomatology in this setting, and early surgical consultation is recommended. Subtle clues, such as an increase in abdominal muscle spasticity, may be present early in the course of an abdominal perforation. As noted above, plain abdominal radiographs are essential to determine whether free air is present in the abdominal cavity. On examination, close attention is given to the presence and nature of bowel sounds. A serum amylase level and daily leukocyte counts should be obtained. Noninvasive duplex ultrasound scanning can be used to determine whether vascular thrombosis or infarct is present. Contrast-enhanced computed tomography of the abdomen and pelvis should be performed when an SCI patient presents

Contrast-enhanced computed tomography of the abdomen and pelvis should be performed when an SCI patient presents with nonspecific systemic illness, leukocytosis, and an absence of localizing signs.

with nonspecific systemic illness, leukocytosis, and an absence of localizing signs.

Ulcer disease and bleeding

The reported incidence of GI bleeding and ulceration in patients with SCI varies from 0.5% to 22%.[40] Kiwerski[41] found that 52 of 2,000 (2.6%) SCI patients had documentation of significant postinjury upper GI bleeding and noted that bleeding most often occurred in patients with a complete injury of the cervical spinal cord during the acute postinjury period. Most patients had no previous history of peptic ulcer disease. In the chronic stage of SCI, more than half of patients with nonspecific abdominal symptoms will be shown to have abnormalities on endoscopy.[42]

In the acute injury setting, multiple factors may predispose the patient to gastritis and ulceration, including stress; malnutrition; hypovolemic shock; and the use of steroids, antithrombotic agents, and nonsteroidal anti-inflammatory drugs (NSAIDs). The maintenance of vagal tone in the absence of effective thoracic sympathetic input to the stomach may result in gastric hypersecretion and hyperacidity in these patients.[43] In comparison with other types of severe injuries, central nervous system trauma has been shown to bring about higher levels of serum gastrin.[44] Corticosteroid therapy, commonly used in patients with central nervous system trauma, may contribute to the high incidence of ulcers.[45] Steroid treatment may also be associated with nonhemorrhagic perforation of the lower GI tract, especially if colonic diverticuli are present.[46] It remains to be seen whether recent trends involving the short-term use of large doses of glucocorticoids after acute trauma will result in a higher incidence of GI bleeding.[47]

Prophylaxis for stress gastritis and ulcer disease during the acute injury period centers around the alteration of acid secretion. Both antacids and H_2 receptor blockers (cimetidine and ranitidine) have been shown to be superior to placebo treatment for the prevention of mucosa erosion in patients who are critically ill. Newer H_2 blockers (nizatidine, famotidine), as well as omeprazole, a powerful suppressant of gastric acid secretion, are still under evaluation for use in the acute setting. Sucralfate, through enhancing mucosal defenses, has also been shown to be effective in preventing acute GI bleeding in critically ill patients.[48] Misoprostol, an analogue of prostaglandin E_1, inhibits gastric acid secretion and may prevent gastric ulceration caused by the effect of NSAIDs on the prostaglandin pathways. This drug, although expensive, may have greater use in the rehabilitation and chronic care setting, where NSAIDs are important in the treatment of pain and inflammation.

The suspicion of active upper GI bleeding invariably calls for endoscopic evaluation. Barium studies must be avoided, as the presence of contrast media may impair endoscopic therapeutic maneuvers should the patient have an acute decline in hemodynamic status. Treatment in this setting may involve coagulation or embolization via endoscope or infusion of intraarterial vasopressin via arteriography. If the bleeding vessel lies posteriorly, either in the duodenal bulb or high in the posterior wall of the stomach, endoscopic coagulation is often unsuccessful, owing to the large caliber of vessels in this region. Bleeding vessels larger than 2 mm in diameter are likely to need open surgical ligation.

The Colon and Anorectum

The term *neurogenic bowel* is frequently used to describe a variety of bowel complications after SCI. This is unfortunate, in that the term overemphasizes the importance of extrinsic enervation and central control of the lower GI tract and downplays the significant amount of reflex activity and local control that remains even in patients with profound neurologic deficits. Knowledge of these local control mechanisms and how they can be manipulated provides a key to successful bowel management.

Neurologic and hormonal control of the colon

Unlike the urinary bladder, the colon serves as more than a simple storage and expulsion vessel. Throughout the length of the large bowel, the mucosa plays the key role in the dynamic processing of waste material before elimination. Control of this activity is carried out through the input of the autonomic nervous system, with a significant contribution from GI peptide hormones.

Several peptides have been identified that affect colonic motility. Substance P, gastrin, enkephalin, and cholecystokinin have been implicated as colonic stimulants. The specific location of their action is still unclear, as these substances have been identified in the circulation, within the wall of the colon, and within the axons of the myenteric nerve plexuses.[17] Secretin and glucagon have long been known to inhibit colonic function. Vasoactive intestinal peptide (VIP), a strong inhibitor of smooth muscle contractility, plays an important role in the forward propulsive activities of the gut.[49] The discovery of VIP is especially interesting, as this substance has been identified as a neurotransmitter in the central, somatic, and autonomic nervous systems, perhaps serving as a neurochemical link that explains the obvious influence of the higher functions of the central nervous system (stress, anxiety) over the autonomic control of the bowel.

The extrinsic neural control of the colon has been studied extensively in animals, but significant anatomic differences found between species cast doubt on the relevance of these studies to human neuroanatomy. Recently, human studies involving direct electrical nerve stimulation in tetraplegic patients have helped map human neural control of the bowel directly[50,51]

Confusion arises over the terminology used to describe neurologic control over the propulsion of waste material through the bowel. The maintenance of normal bowel activity involves a complex coordination of relaxation of the bowel wall, as well as contraction. A hypertonic bowel may impede waste flow if not accompanied by patterned relaxation. As a result, use of the terms *facilitation* and *inhibition* is confusing, as facilitation of smooth muscle tone may actually inhibit the flow of digested material. For this discussion, such terms will be applied solely to their effect on propulsion of intraluminal contents.

The parasympathetic output to the colon is almost exclusive stimulatory to the movement of feces. The vagus nerve supplies the GI tract to the level of the transverse colon, with the lower bowel under the parasympathetic control of the sacral pelvic nerves, particularly S2 and S3.[49] Sympathetic nervous system influences are inhibitory to colonic propulsion. Sympathetic adrenergic enervation is carried from the thoracolumbar

segments of the spinal cord, through the superior mesenteric ganglion and splanchnic nerves to the proximal colon. Distal colonic segments receive efferent sympathetic fibers from the lumbar colonic nerves through the inferior mesenteric ganglion. Sensory impulses arise primarily from mechanoreceptors in the wall of the colon, travel through peripheral parasympathetic nuclei, and send collateral fibers along preganglionic pathways to corresponding sacral dorsal root ganglia. The role of these sensory fibers is gaining increasing attention as local reflex loops are being recognized that affect colonic motility in animals.[52]

Near the mucocutaneous junction in the rectum, GI smooth muscle merges with the striated fibers of the external anal sphincter, which is under somatic, voluntary control. Although the rectum derives its sensory supply from parasympathetic fibers, the anus sends cutaneous sensory impulses through the inferior hemorrhoidal nerves. Motor supply to the perianal striated musculature comes by way of the internal pudendal nerve and the perineal branch of the fourth sacral nerve.[53]

Numerous areas of the brain, brain stem, and spinal cord have been shown to exert a direct influence over bowel function. Recent advances in brain stem imaging have allowed investigators to correlate specific central nervous system lesions with abnormalities in colonic motility studies in humans.[54] It is likely that the higher levels of the central nervous system serve to inhibit spinal cord control centers that promote propulsion (Fig 1). This is clearly shown in patients with high spinal cord transection, who exhibit a generalized increase in colonic tone and diminished functional propulsion.[55]

It is clear that the spinal cord plays an important role in processing information at segmental levels and that specific areas of the cord are important in the control of bowel and bladder function (see Fig 1). Individuals with spinal cord lesions of the high lumbar cord exhibit a marked increase in colonic motility, whereas patients with surgical or chemical lesions of the lumbar sympathetic chair show no significant changes in bowel function.[53] These studies implicate the lumbar spinal cord as an inhibitory center for propulsion in the distal colon. In contrast, sacral lower motor neuron transection has been carried out in humans, resulting in total elimination of colonic motility.[56] These observations, along with several animal studies, point to a facilitory function present in the sacral nerves, as might be expected by the predominance of parasympathetic fibers in this region.

Although no longer connected with the brain, the spinal cord below the level of a traumatic lesion is usually alive and active, with considerable residual segmental reflex activity. Thus, the patient with a cervical or thoracic spinal cord lesion is usually left with functioning lumbar and sacral control centers, lacking only weak descending inhibitory influences from the brain. Although these individuals exhibit a generalized increase in bowel tone and diminished motility, their lumbar and sacral control centers are still operative, and their bowel function is easier to manage compared with individuals with lumbar or sacral lesions.

The basic propulsive properties of colonic smooth muscle are mediated by the myenteric nerve plexus. These fibers promote local relaxation, as evidenced by the increase in muscularis activity after administration of neural antagonists and by the hypertonic bowel noted in humans with congenital absence of the myenteric plexus (Hirschsprung's disease).[57]

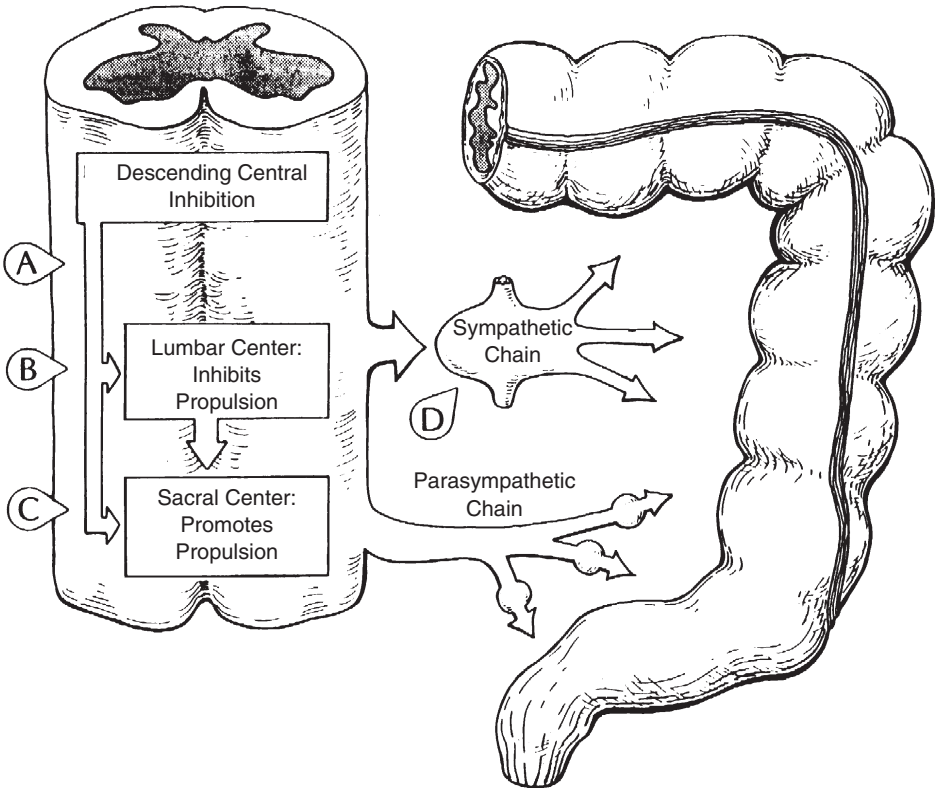

Fig 1. Schematic representation of extrinsic neurologic control of the descending colon and rectum. Fecal propulsion, involving regulation of relaxation and contraction of the bowel wall muscle, results from the interaction of extrinsic control with the enteric nervous system. A central nervous system lesion (UMN lesion) above the lumbar segments of the spinal cord (A) leaves the lumbar and sacral reflex centers intact and functional, although withdrawal of descending inhibition from higher centers may result in the predominance of the motility-slowing influences from the lumbar center. A lesion of the lumbar cord (B) decreases sympathetic inhibition and removes the inhibitory influences of the lumbar center on the promotility sacral center, resulting in increased bowel propulsion. Lesions involving the sacral cord (C) or peripheral preganglionic parasympathetic fibers (LMN lesions) result in intractable loss of lower colonic tone and motility. Chemical or surgical sympathetic ablation (D) may have little effect on propulsion, as the lumbar spinal cord center remains intact, capable of modulating and inhibiting sacral promotion of bowel activity. Reprinted with permission from Frost FS. Gastrointestinal dysfunction spinal cord injury. In Yarkony G. (Ed.) *Spinal Cord Injury: Medical Management and Rehabilitation.* Gaithersburg, Md: Aspen Publishers, 1994. ©1994 by Aspen Publishers.

The neurologic activity of the colon is difficult to study. Disorders of colonic function are usually evaluated by pressure recordings or *colonometrograms*. Such motility investigations are limited by the large number of variables affecting bowel motility that are nearly impossible to control in the experimental setting (eg, diet, medications, level of activity). Thus, it is difficult to determine whether a change in motility has taken place as a result of a single experimental intervention. In light of this, strategies aimed at improvement of bowel function are best evaluated by assessing patient satisfaction. Studied in the electrophysiology laboratory, normal neural control at the local level of the bowel wall can be recorded by surface or needle electrode as spike potentials and as slow waves, which are responsible for the mixing activity of the bowel. Abnormalities in the patterns of slow waves and spike potentials have been shown in SCI.[58] Persons with cervical spinal cord lesions exhibit bowel "spasticity and dysrhythmia," with irregular frequency, amplitude, and velocity electrical potentials, whereas those with lumbosacral and conus lesions have silent electrorectograms. This lends physiologic evidence to the concept of upper motor neuron (UMN) and lower motor neuron (LMN) bowel disorders.

Linking local control mechanisms to central control elements in the spinal cord, GI reflex loops have been demonstrated and are of clinical importance in individuals with SCI. Most notably, the rectoanal reflex, elicited by anorectal stimulation or distention, produces an automatic contraction of the rectal smooth muscle and relaxation of the proximal anal canal. This reflex, as noted below, is often present after SCI and can be utilized to promote defecation. Vesicoanal and local anal sphincter reflexes are also described.[59]

The crucial element in the management of defecation disorders is the maintenance of a consistent diet and bowel care pattern.

Disorders of bowel emptying

Defecation problems may be caused by a change in bowel motility, an alteration in the consistency of waste material, or loss of the normal mechanics that promote rectal emptying. In individuals with SCI, all three factors play a role; all of these factors are also amenable to manipulation by artificial means.

The crucial element in the management of defecation disorders is the maintenance of a consistent diet and bowel care pattern; this can be carried out by the patient, nursing staff, or by family and caregivers in the home. Early in the rehabilitation phase of injury, the nursing staff must keep accurate records of bowel-emptying interventions and rough estimates of the amount of stool expelled. Ongoing teaching with patient and family is necessary to ensure that adequate problem-solving skills are developed prior to discharge.

The most common errors in the initial inpatient management of emptying disorders lie in inaccurate or incomplete record keeping and in a lack of patience on the part of caregivers. Incomplete daily medical records and confusion may lead to an underestimation of the amount of stool eliminated,

resulting in overuse of chemical emptying agents. In this situation, the patient is never given the chance to build up a continuous column of stool that can be effectively moved through the colon. Each patient presents to the rehabilitation setting with a unique preinjury "bowel ethic," and insistence on resuming old habits may be strong. Pressure to resume a pattern of *daily* bowel emptying leads to overuse of cathartics, thus interfering with the establishment of a new baseline of defecation frequency. Many patients must learn that their injury, with coincident diminished physical activity and loss of sphincter control, necessitates the establishment of a totally new pattern of bowel emptying. Often the goals of daily bowel emptying and fecal continence are mutually exclusive.

The bowel program

The goal of the bowel program for a person with an SCI is simple. Because the external sphincter is usually not under voluntary control, the goal is to consistently move solid waste material into the rectum and keep it there until the time and place is convenient for emptying. Achieving this goal is not so simple. The first avenue of treatment involves an appreciation of the mechanics of defecation and the points at which intervention, substitution, and augmentation are possible.

In persons who are neurologically intact, mass movement of feces into the rectum may take place only once a day, usually in the morning, and often in association with a gastrocolic reflex. Sensing that the rectum is full, the nondisabled individual can get to the toilet and assume a body position appropriate for emptying. A low toilet is optimal, as flexion of the knees and forward bending of the trunk increases abdominal pressure and puts the rectum at the optimal angle for emptying. Next, the glottis is closed and intraabdominal pressure is raised, pushing the waste material downwards toward the anorectal verge. With stretching of this area, the anorectal reflex is activated, relaxing the proximal anal region. Voluntary relaxation of the external sphincter completes the emptying process.

At each point of the defecation process, the person with SCI is disadvantaged. Recumbency and impaired motility may prevent mass movement of waste through the bowel. The gastrocolic reflex may be absent after spinal cord transection.[60] The sensation of a full rectum may be absent. Mobility deficits may make toileting impossible. In many instances raised toilet seats are used to make transfers easier, placing the patient in an unfavorable posture for defecation. The lack of chest and abdominal wall muscle tone or the presence of a tracheostomy will result in the inability to raise intraabdominal pressures. In cauda equina or conus (LMN), the patient may be devoid of reflex defecation mechanisms.

Simple maneuvers may be of benefit. An abdominal binder will raise Valsalva pressures to the rectum. The patient can be situated in an upright position and flexed at the hip, or the bowel program can be carried out in the right lateral side-lying position, to take advantage of gravity and the natural rectal curvature. Most importantly, progressive physical mobilization will promote general health and normal mass movement of fecal material.

Most acutely injured patients will be quite constipated at the time of discharge from the intensive care unit. Once this is treated (see

Steps to a Successful Bowel-Emptying Program

1. Evaluate and treat bowel impaction.
2. Encourage physical mobilization and facilitate the physical mechanics of defecation.
3. Eat a healthy diet.
4. Keep accurate records of bowel output until regularity is achieved.
5. Allow formation of a normal column of stool in the colon by avoiding strong oral cathartic medication.
6. Regulate stool consistency by adjusting dietary fiber.
7. Rectal bowel-emptying methods (suppositories, digital stimulation) are best for maintaining continence.
8. Do not insist on daily bowel emptying if suppositories or enemas are used.
9. Don't overemphasize preinjury bowel habits and patterns.

below), the efficient bowel program (see box, "Steps to a Successful Bowel-Emptying Program") calls for oral management of stool consistency through diet and rectal management of defecation through a combination of evacuants and digital (finger) stimulation and fecal removal. Ideally, oral cathartics will play a minimal role. In practice, many of the oral medications used in the hospital for bowel management are unnecessary after the patient returns home to previous dietary habits. Attention must be given to all medications that the patient receives. Those that slow bowel function (eg, clonidine, ferrous sulfate, codeine preparations, anticholinergics, tricyclic antidepressants) will interfere with the establishment of a regular emptying pattern.

The role of dietary fiber in these instances is clear. Because the presence of liquid stool in the rectum is synonymous with incontinence, maintenance of proper stool consistency is dependent on proper regulation of fiber intake. Patients must achieve "therapeutic constipation" to maintain continence. Education in the fiber content of foods is essential, as a change of only 25% in the amount of fiber in the diet can result in a significant change in stool consistency.[61] Experimentation with fiber intake must be supervised, however, especially in the case of bran fiber. Patients with erratic fluid intake or dehydration may develop intestinal obstruction, as bran fiber increases stool bulk beyond the capacity of the body to saturate the waste material. If the patient is unable or unwilling to adhere to a healthy diet, the use of fiber supplementation in the form of commercial fiber preparations will be necessary.

Stool softeners, chiefly docusate preparations (Colace), are commonly used in the rehabilitation setting, although their effectiveness is questionable.[62] They may act to soften by adding gas to feces, not by adding water (even the hardest stool is fully saturated with water). There is also the potential for hepatotoxicity, as these drugs enhance the absorption of substances that are metabolized by the liver.

In most instances patients with spinal cord dysfunction should be encouraged to avoid the use of strong oral cathartic medications in their regular bowel routine. The time to onset of action is unpredictable. As sphincter control is not present, bowel accidents will be the rule, not the exception. In addition, many of the commonly used cathartics have been shown to present significant risks when used

over the long term. The regular use of cathartics in persons with refractory motility disorders and recurrent impaction will be discussed below.

The final component of the bowel program, emptying of the rectum, is accomplished by artificial means. Occasionally persons with incomplete injuries and good rectal sensation are able to empty the rectum by abdominal massage and Valsalva maneuver. Many persons with UMN lesions will require artificial stimulation of rectal reflexes by chemical suppositories or enemas or by finger stimulation of the anorectal region. If chemical stimulants are used, the routine is best performed every other day or every third day. Daily use of suppositories or enemas usually results in overstimulation of the rectal mucosa and increased production of mucous and gas. The increase in the net amount of fluid in the rectum will promote incontinence in those patients who have no control over the anal sphincter.

Patient counseling is critical at this time. Most will be disillusioned and disappointed over the need for assistance with bowel emptying—either physical assistance by a caregiver or chemical assistance by suppositories and enemas. Confidence may be broken by a single episode of incontinence. Reassurance must be given; these "tried-and-true" methods of bowel emptying have been taught successfully for decades and have allowed thousands of persons with SCI to maintain continence and minimize the time needed for bowel management.

The basic elements of the bowel routine for a person with a UMN lesion are quite simple. Digital stimulation can be performed with the patient on the commode or recumbent in the right side-lying position. Inserting the finger with stimulation of the anus will result in an increase in the electrophysiologic activity of the rectum[57] and will stimulate distal propulsion of fecal material. Sometimes finger stimulation alone is sufficient to promote emptying. Usually, however, finger stimulation is augmented by chemical stimulation, either by suppository or enema. These agents will soften and lubricate the stool for passage, but their ability to actually "flush" the bowel is minimal. In fact, they act primarily by providing an artificial irritant to the anorectal mucosa, which serves to promote reflex propulsion in this region. Bisacodyl, glycerine, and carbon dioxide preparations are gentle and commonly used in this setting. Newer, small-volume, liquid bisacodyl preparations and propylene glycol–based suppositories speed bowel emptying, as these delivery systems allow the medication to diffuse across the bowel mucosa more quickly.[63,64] None of these preparations work well when used in isolation, however. Finger stimulation of the anorectal reflexes will bring stool to the rectum and must still be carried out every 10 minutes after suppository/enema delivery until the rectal vault is empty. For patients with impaired hand function, special equipment for anal suppository self-insertion is available, but these devices must be used with care to guard against sphincter injury.

In some patients, local defecation reflexes may be aberrant or absent. A failure of sphincter relaxation in response to increased rectal pressures has been demonstrated in a subgroup of tetraplegic patients.[65] This may be analogous to the uncoordinated activity of bladder and urinary sphincter known as *detrusor–sphincter dyssynergia*. Other patients with low spinal cord or sacral nerve (LMN)

lesions may have no response to stimulation or chemicals. Similarly, persons with long-standing SCI and chronic distention of the rectum may not possess any discernible rectal reflex activity. In such instances, finger removal of stool is the treatment of choice. Because chemicals are not used, this technique can be performed daily, or even more often, as persons with this type of lesion frequently can carry out this activity without physical assistance. In contrast to patients with UMN lesions who may have residual muscle tone of the internal anal sphincter, these patients have flaccid sphincters. Continence is maintained solely by managing stool consistency with dietary manipulation, and by avoiding foods that cause liquid waste to reach the rectum.

Chronic manipulation of the anus and rectum for artificial bowel emptying no doubt promotes the formation of hemorrhoids; 58% of patients who are more than 5 years post SCI complain of symptomatic hemorrhoids.[63] Usually bleeding is the problem, but rectal mucosal prolapse may also result in chronic external secretion of mucous and result in skin breakdown in the perianal region. Most small hemorrhoids will respond to hydrocortisone suppositories inserted after the completion of the bowel program. Sclerotherapy, elastic band ligation, and photocoagulation may be indicated when internal hemorrhoids prolapse or bleed persistently. Rectal bleeding should not be attributed to hemorrhoids until an anorectal examination is done. The relative risk of GI neoplasms (ie, compared with that in the nondisabled population) is unknown. Unfortunately, routine colon cancer screening by colonoscopy or barium enema is rarely carried out. The reason for this is simple: The bowel preparation necessary for an adequate screening examination presents a great inconvenience to these patients. Often a bowel preparation of this magnitude will require hospital admission or several days off work for the patient, caregiver, or both. The patient's regular bowel routine may be disrupted for weeks after such an examination

Fecal impaction

A fecal obstruction of the colon can quickly lead to life-threatening complications. Spontaneous colonic perforation is always a threat; the patient is presented with even more risk during disimpaction, when a damaged colonic mucosa is subjected to chemical and physical injury. In addition, chronic fecal distention of the rectum can result in stercoral ulcers of the rectal mucosa ("pressure sores" of the bowel). Overfilling of the colon with stool is known to have a deleterious effect on proximal function of the GI tract and is no doubt the most frequent cause of nausea in this population.[66] In addition, the promotion of autonomic dysreflexia secondary to rectal distention is potentially life threatening.

Most SCI patients have some degree of fecal impaction on admission to the rehabilitation hospital from the acute care setting. The diagnosis is not difficult. Those with mild degrees of impaction will complain of loss of appetite and nausea. Often these patients are mistakenly given antinausea preparations, most of which have anticholinergic and constipating properties, and the problem is further exacerbated. On examination, abdominal distention and fecal masses may be noted. Factitious diarrhea may be present, as brown liquid may be expressed around a

blockage of inspissated stool. Plain films of the abdomen may show a remarkable amount of stool in the colon, and air patterns in the intestines may be abnormal.

In patients with new SCIs, the impaction is usually of short duration and will usually be remediable by the simple and noninvasive bowel-emptying techniques described above. If the patient is not experiencing vomiting or abdominal pain and bowel sounds are preserved, the impaction can be approached gradually, from the rectum only. Many SCI centers automatically perform rectal evacuations with suppositories for the first three to four consecutive days after rehabilitation admission.

Persons with chronic spinal cord conditions and fecal impaction may not respond to a such an approach. Because parasympathetic input (from the vagus nerve) is normal up to the transverse colon in these patients, chronic motility deficits may be most striking in the left colon.[67] Thus, fecal impactions may develop in the proximal colon as a result of poor distal propulsion. In many cases dehydration is present, and stool will be extremely hard. The distal colon may be chronically damaged by long-standing stretching and chemical damage and may not respond to simple pharmacologic stimulation. Not uncommonly, intestinal pseudo-obstruction will be present, with vomiting, abnormal bowel sounds, and dilation of small bowel loops on radiograph.

Efforts to remove a chronic fecal impaction solely by the use of oral cathartics are often unsuccessful and may worsen clinical symptoms.

Efforts to remove a chronic fecal impaction solely by the use of oral cathartics are often unsuccessful and may worsen clinical symptoms. If vomiting and ileus are present, decompression of the upper GI tract by nasogastric suction is usually necessary. The use of strong oral cathartic agents in this setting often adds to abdominal pain and gaseous distention and may contribute to serious complications. Suppositories and low-volume enemas are usually of little use; these chemicals act distal to the blockage, resulting only in mucous secretion. Sodium phosphate, mineral oil, or tap water enemas can be tried, with volumes limited to 1 pint per application. Soapsuds enemas should be avoided, if possible, as they may be injurious to the rectal mucosa and precipitate serum electrolyte disturbances. Digital removal of stool from the rectum is the cornerstone of therapy; if stool can be reached with the examiner's finger, fragmentation and extraction of stool should be carried out using lidocaine jelly as a lubricant. In persons prone to autonomic dysreflexia, spinal anesthesia may be necessary.

When the colonic impaction is proximal, a colonoscopic-directed lavage may be necessary, using a water-soluble contrast media (Gastrografin or Hypaque) in a 20% to 50% solution. These substances are high in osmolality and stimulate emptying by drawing water into the gut lumen and stimulating peristalsis.[68] It must be remembered that malignancies or intestinal adhesions may be responsible for the development of these obstructions. In light of this, it is not unreasonable to perform endoscopic or radiologic evaluation of the colon to determine whether tumors or other local pathologic abnormality

is present. Patients with recurrent, severe fecal impactions may be candidates for surgical decompression and, ultimately, colostomy. As secondary benefits, bowel care may be more convenient with a colostomy, and healing of pressure sores in the perianal region can be accelerated.[69]

Ultimately, the best approach to the problem of recurrent fecal impactions is prevention. Clinicians are now faced with a growing number of persons who have survived decades post injury, many of whom are noting increasing difficulty in maintaining a successful bowel-emptying schedule after years of artificial bowel stimulation. Some of these individuals will need to employ regular use of oral laxatives. Debilitated, bedridden patients will invariably need oral medication to prevent impaction, unless hyperosmotic tube feedings are used. In addition to the likelihood of producing fecal incontinence, as noted above, the use of oral laxatives carries a variety of additional risks.

Most oral laxatives have little primary effect on bowel motility; they exert their action by changing the net water and ion transport across the bowel mucosa. The chronic use of irritant laxatives, including bisacodyl, phenothalein, senna, and cascara, will cause damage to the mucosa and ultimately may produce abnormal intestinal motor function.[70] The so-called "bulk former" laxatives, containing polysaccharide or cellulose, are among the safest preparations to use on a chronic basis. Many of these are derived from psyllium seed, and often they are combined with the irritant compounds listed above. Despite the fact that these agents are considered to be inert fiber supplements, in actuality they augment the secretory function of the gut and decrease its absorptive function. Adequate hydration is crucial to the success of this type of therapy, and use of these agents must be monitored in diabetic patients, as most contain a high sugar content.

If this therapy is not successful in preventing recurrent impaction, then regular use of a hyperosmotic laxative such as milk of magnesia or lactulose is justified. Therapy is instituted with the knowledge that both may worsen cramping and produce electrolyte abnormalities (especially in patients with renal disease) when used on a chronic basis. Lactulose is metabolized in the colon to low-molecular-weight compounds that lower the pH of the colon and stimulate peristalsis. Lactulose will improve peristalsis, but it is an expensive drug, and removal of waste from the rectum may be more difficult, as the stool will be poorly formed. Cisapride has gained considerable attention as a pro-motility agent for chronic use. Preliminary studies in patients with SCI have suggested that it may be of benefit in the long-term care setting.[71,72] Stimulation of the anterior sacral roots via implantable electrodes is also under investigation as a means of promoting bowel emptying.[73,74]

Diarrhea

For the person with a physical impairment, an increase in stool volume, liquidity, and stool frequency is more than an uncomfortable nuisance. Diarrhea can predispose the patient to the development of pressure sores and may become a dangerous medical condition for those who are unable to physically manage their replacement oral fluid intake. An exhaustive discussion of the potential causes of diarrhea is beyond the scope of this article. There

are, however, a number of special considerations relevant to patients with SCI.

Initial consideration must be given to fecal impaction as the probable cause of diarrhea. Fecal stasis in the proximal colon may allow only liquid waste to pass, and treatment of this presumed "diarrhea" with constipating agents may be disastrous. As impaction may not be evident on examination, a single plain radiograph of the abdomen to assess for fecal blockage, done early on, is usually part of an efficient initial evaluation of this problem. Hyperosmotic tube feedings and increased ingestion of milk products are other common causes of loose stool in the hospital setting.

A great percentage of patients seen in the rehabilitation setting will have been treated with antibiotics during their acute hospitalizations, and development of pseudomembranous colitis is not uncommon. Most often, *Clostridium difficile* is the responsible pathogen, especially in persons who have been treated with ampicillin, clindamycin, and cephalosporins. Institutionalized patients with secretory diarrhea, without visible blood, should be evaluated for the presence of *C difficile* toxin immediately and placed on enteric precautions until results are available. Metronidazole 500 mg four times daily usually is sufficient in the treatment of those with confirmed colonization. Of course, stool examination for other pathogens is indicated in selected clinical situations, especially in pediatric rehabilitation units, where rotavirus outbreaks can have devastating effects.

In the outpatient setting, the high incidence of polypharmacy in this population should alert the physician to the use and abuse of a variety of drugs with the potential for producing diarrhea. Surreptitious laxative abuse or overuse of chewing gum, candy, or ethanol on the part of the patient should be considered, but more often the condition is idiopathic. Drugs such as magnesium antacids, diuretics, and a host of antibiotics are common offenders.

In the majority of instances, as in the nondisabled population, episodes of diarrhea are self-limited. After eradicating correctable causes, treatment should focus on body fluid replacement and protection of the perianal skin; an emollient ointment (Desitin, A&D Ointment) without antifungal compounds should be used. Once diarrhea has resolved, acidophilus preparations and yogurt may assist in reestablishing the normal bacterial flora of the intestinal tract. Acidophilus products, widely used in Europe, contain dried but viable lactobacilli cultures appropriate for oral administration. These bacteria produce lactic acid and creative an environment that favors the establishment of a normal acid bacterial flora in the colon. A 2- or 3-day course of treatment is helpful. The initial increase in flatus associated with this therapy will resolve spontaneously.

REFERENCES

1. Apple D, Hudson L. *Spinal Cord Injury, The Model: Proceedings of the National Consensus Conference on Catastrophic Illness and Injury, December 1989.* Atlanta, Ga: The Georgia Regional Spinal Cord Injury Care System, Shepherd Center for Treatment of Spinal Injuries, Inc; 1990.
2. Hanson R, Franklin M. Sexual loss in relation

to other functional losses for spinal cord injured males. *Arch Phys Med Rehabil.* 1976; 57:291–293.
3. Gore R, Mintzer R, Calenoff L. Gastrointestinal complications of spinal cord injury. *Spine* 1981;6(6):538–544.
4. Stone JM, Wolfe V, Nino-Murcia M, Perkash I. Bowel care problems and anorectal function after SCI. *ASIA Abstracts Dig.* 1989;April:74.
5. Albert T, Levine M, Cotler J, Balderson R. Gastrointestinal complications in spinal cord injury. *ASIA Abstracts Dig.* 1990;**:116.
6. Peiffer S, Blust P, Leyson F. Nutritional assessment of the spinal cord injured patient. *J Am Diet Assoc.* 1981;78:501–505.
7. Cooper I, Rynearson E, MacCarty C, Power M. Metabolic consequences of spinal cord injury. *Neurology.* 1950;10:858.
8. Laven GT, Huang CT, DeVivo MJ, et al. Nutritional status during the acute stage of spinal cord injury. *Arch Phys Med Rehabil.* 1989;70:277.
9. Rodriguez DJ, Clevenger FW. Obligatory negative nitrogen balance following spinal cord injury. *J Parenter Enteral Nutr.* 1991; 15:319.
10. Kolpek J. Comparison of UUN and measured energy expenditure in SCI and nonsteroid-treated severe head trauma patients. *J Parenter Enteral Nutr.* 1989;13:277.
11. Cox SA, Weiss SM, Posyniak EA , et al. Energy expenditure after spinal cord injury: Evaluation of stable rehabilitating patients. *J Trauma.* 1985;25: 419–423.
12. Haug RH, Wible RT, Likavec MJ, et al. Cervical spine fractures and maxillofacial trauma. *J Oral Maxillofac Surg.* 1991;49:725–729.
13. Thornton JB, Sneed RC. Dental management of patients with spinal cord injury. *Compend Contin Educ Dent.* 1995;XIII(2):13–15.
14. Ring J, Seifert J, Lob G, Stephan W, Probst J, Brendel W. Elimination rate of human serum albumin in paraplegic patients. *Paraplegia.* 1974;12:139
15. Chin DE, Kearns P. Nutrition in the spinal injured patient. *Nutr Clin Pract.* 1991;6:213–222.
16. Greenway R, Houser H, Lindan O. Long term changes in gross body composition of paraplegic and quadriplegia patients. *Paraplegia.* 1969;7:301.
17. Gelfand D. Complications of gastrointestinal radiologic procedures. *Gastrointest Radiol.* 1980;5:293–315.
18. Logeman J. Swallowing disorders following spinal cord injury. Paper presented at the American Congress of Rehabilitation Medicine Annual Meeting; October 1990, Phoenix, AZ.
19. Wattchow DA, Furness J, Costa M. Distribution and coexistence of peptides in nerve fibers of the external muscle of the human gastrointestinal tract. *Gastroenterology.* 1988;95: 32–41.
20. Stinneford JG, Keshavarzian A, Nemchausky BA, et al. Esophagitis and esophageal motor abnormalities in patients with spinal cord injuries. *Paraplegia.* 1993;31(6):384–392.
21. Humphries T, Castell D. Effect of oral bethanechol on parameters of esophageal peristalsis. *Dig Dis Sci.* 1981;26:129.
22. McCallum R, Ippoliti A, Cooney C. A controlled trial of metoclopramide in symptomatic gastroesophageal reflux. *N Engl J Med.* 1977;296:354.
23. Smout A, Bogaard J, Grade A. Effects of cispapride, a new gastrointestinal prokinetic substance, on interdigestive and postprandial motor activity of the distal esophagus in man. *Gut.* 1985;26:246–259.
24. Binnie N, Creasy G, Edmond P, Smith A. The action of cisapride on the chronic constipation of paraplegia. *Paraplegia.* 1988;26:151–158.
25. Longo WE, Woolsey RM, Vernara AM, et al. Cisapride for constipation in spinal cord injured patients. *J Spinal Cord Med.* 1995; 18(4):240–244.
26. Fealey R, Szurszewski J, Merritt J, DiMagno E. Effect of traumatic spinal cord transection on human upper gastrointestinal motility and gastric emptying. *Gastroenterology.* 1984;87:69–75.
27. Shuster M, ed. Motor disorders of the stomach. *Med Clin North Am.* 1981;65(6):1,269–1,289.
28. Peschiera J, Beerman S. Intestinal dysfunction associated with acute thoracolumbar fractures. *Orthop Rev.* 1990;XIX(3):284–288.
29. Claus-Walker J, Halstead L. Metabolic and endocrine changes in spinal cord injury: I. The nervous system before and after transection of the spinal cord. *Arch Phys Med Rehabil.* 1981;62:595–601.
30. Segal J, Brunnemann S, Gordon S, Eltorai I. Decreased theophylline bioavailability and impaired gastric emptying in spinal cord injury. *Curr Ther Res.* 1985;38(6):831–846.
31. Richardson R, Cerullo L. Transabdominal

neurostimulation in the treatment of neurogenic ileus. *Appl Neurophysiol.* 1979;42: 375–382.
32. Nino Murcia M, Friedland GW. Functional abnormalities of the gastrointestinal tract in patients with spinal cord injuries: Evaluation with imaging procedures. *Am J Roentgenol.* 1992;158(2):279–281.
33. Segal JL, Milne N. Metoclopramide induced normalization of impaired gastric emptying in spinal cord injury. *Am J Gastroenterol.* 1987;82:1,143–1,148.
34. Cosman BC, Sullivan GL, Johnson DM. Gastrostomy for gaseous decompression in a quadriplegic man. *Am Paraplegia Soc Abstracts.* 1990;September:7.
35. Greenfield J. Abdominal operations on patients with chronic paraplegia. *Arch Surg.* 1948;59:1,077–1,087.
36. Roth E, Fenton L, Gaebler D, Frost F, Yarkony G. Superior mesenteric artery syndrome in acute traumatic quadriplegia: Case reports and literature review. *Arch Phys Med Rehabil.* 1991;72(6):417–420.
37. Moses FM. Colonic perforation due to oral mannitol. *JAMA.* 1988;260(5):640.
38. Charney KJ, Juler G, Comarr A. General surgery problems in patients with spinal cord injuries. *Arch Surg.* 1975;110:1,083–1,088.
39. Chassin SL, Apstein MD, Williams W. Gallbladder dysmotility in spinal cord injury. *ASIA Abstracts Dig.* 1988;May:5
40. Kewalramani L. Neurogenic gastroduodenal ulceration and bleeding with spinal cord injuries. *J Trauma.* 1979;19(1):259–265.
41. Kiwerski, J. Bleeding from the alimentary canal during the management of spinal cord injury patients. *Paraplegia.* 1986;24:92–96.
42. Tanaka M, Motoaki U, Motoo K. Gastroduodenal disease in chronic spinal cord injuries. *Arch Surg.* 1979;114:185–187.
43. Pollack L, Finkelman I. The digestive apparatus in injuries to the spinal cord and cauda equina. *Surg Clin North Am.* 1954;34:259–268
44. Bowen J, Fleming W, Thompson J. Increased gastrin release following penetrating central nervous system injury. *Surgery.* 1974; 75(5):720–724.
45. Messer J, Reitman D, Sacks H, Smith H, Chalmers T. Association of corticosteroid therapy and peptic ulcer disease. *N Engl J Med.* 1983;309:21–24.
46. Fadul C, Lemann W, Thaler H. Perforation of the gastrointestinal tract in patients receiving steroids for neurologic disease. *Neurology.* 1988;38:348–352.
47. Bracken MB, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. *N Engl J Med.* 1990;322(20):1,405–1,411.
48. Cannon LA, Heiselman D, Gardner W. Sucralfate verses antacid in preventing acute gastrointestinal bleeding in the ventilator patient. *Crit Care Med.* 1986;14:358.
49. Koch TR, Carney A, Go L. Idiopathic chronic constipation associated with decreased colonic vasoactive intestinal peptide. *Gastroenterology.* 1988;94:300–310.
50. Brindley G, Polkey C, Rushton D. Sacral anterior root stimulation in paraplegia. *Paraplegia.* 1982;20:365–381.
51. Varma JS. Autonomic influences on colorectal motility and pelvic surgery. *World J Surg.* 1992;16(5):811–819.
52. Longhurst JC. Gastrointestinal reflexes. *Gastroenterology.* 1988;95:524–533.
53. Goligher JC, Leakcock AG, Brossy JJ. The surgical anatomy of the anal canal. *Br J Surg.* 1955;43:51–56.
54. Weber J, Denis B, Mihout J, Muller F. Effect of brain stem lesions on colonic and anorectal motility. *Dig Dis Sci.* 1985;30(5):419–425.
55. Connell AM, Frankel H, Guttman L. The motility of the pelvic colon following complete lesions of the spinal cord. *Paraplegia.* 1963;1:98–115.
56. Devroede G, LaMrache J. Functional importance of extrinsic parasympathetic innervation to the distal colon and rectum in man. *Gastroenterology.* 1974;66:273–280.
57. Kaufman PN, Krevsky B, Malmuc LS, et al. Role of opiate receptors in the regulation of colonic transit. *Gastroenterology.* 1988;94: 1,351–1,356.
58. Shafik A. Electrorectogram study of the neuropathic rectum. *Paraplegia.* 1995;33(6):346–349.
59. Roman C, Gonella J. *Physiology of the Gastrointestinal Tract.* 2nd ed. New York, NY: Raven Press; 1987.
60. Glick M, Meshkinpour J, Haldeman S, Hoehler F, Downey N, Bradley WE. Colonic dysfunction in patients with thoracic spinal cord injury. *Gastroenterology.* 1984;86:287–294.
61. Rousseau P. Treatment of constipation in the elderly. *Postgrad Med.* 1988;83(4):349–389.
62. Chapman R, Sillery J, Fontana D, Matthys C,

Saunders D. Effect of oral dioctyl sodium sulfosuccinate on intake–output studies of the human small and large intestine. *Gastroenterology.* 1985;89:489–493.
63. Dunn KL, Galka ML. A comparison of the effectiveness of Therevac SB and bisacodyl suppositories in SCI patient's bowel programs. *Rehabil Nurs.* 1994;19(6):334–338.
64. Stiens SA. Reduction in bowel program duration with polyethylene glycol based bisacodyl suppositories. *Arch Phys Med Rehabil.* 1995;76(7):674–677.
65. Stone JM, Wolfe V, Nino-Murcia M, Perkash I. Bowel care problems and anorectal function after SCI. *ASIA Abstracts Dig.* 1989;April:74.
66. Youle M, Read N. Effect of painless rectal distention on gastrointestinal transit of a solid meal. *Dig Dis Sci.* 1984;29(10):902–906.
67. Menardo G, Bausano G, Corazziari E. Large bowel transit in paraplegic patients. *Dis Colon Rectum.* 1987;30:924–928.
68. Wrenn K. Fecal impaction. *N Engl J Med.* 1989;321(10):658–662.
69. Stone JM, Wolfe VA, Nino-Murcia M, Perkash I. Colostomy as treatment for complications of spinal cord injury. *Arch Phys Med Rehabil.* 1990;71(7):514–518.
70. Tedesco F, DiPiro J. Laxative use in constipation. *Am J Gastroenterol.* 1985;80:303–309.
71. Geders JM, Gaing A, Baumann WA, et al. The effect of cisapride on segmental colonic transit time in patients with spinal cord injury. *Am J Gastroenterol.* 1995;90(2):285–288.
72. Carone R, Vercelli D, Bertapelle P. Effects of cisapride on anorectal and vesicourethral function in spinal cord injured patients. *Paraplegia.* 1993;31(2):125–127.
73. Chia YW, Lee TK, Kour NW, et al. Microchip implants on the anterior sacral roots in patients with spinal trauma: Does it improve bowel function? *Dis Colon Rectum.* 1996;39(6):690–694.
74. Binnie NR, Smith AN, Creasey GH, Edmond P. Constipation associated with chronic spinal cord injury: The effect of pelvic parasympathetic stimulation by the Brindley stimulator. *Paraplegia.* 1988;26(3):151–158.