Introduction

Faecal incontinence and constipation are common in people with neurological disease or injury. Up to 70% of people with multiple sclerosis (MS) report monthly episodes of incontinence (Chia et al., 1995) and up to 80% of spinal cord injury (SCI) individuals complain of constipation (Glickman and Kamm 1996). Consequently many people with neurological conditions suffer emotional distress and limit their participation in social activities and/or work.

The medical literature is confusing. ‘Neurogenic incontinence’ can denote both incontinence secondary to damage to the pudendal nerve during childbirth or be associated with major neurological injury or disease such as autonomic diabetic neuropathy. For the purpose of this chapter, ‘neurogenic bowel’ refers to incontinence and constipation that occurs in patients with any chronic pathological process affecting the nervous system.

Management of the neurogenic bowel must be undertaken with the recognition that one may be treading a fine line between helping constipation and precipitating faecal incontinence. An individualised bowel programme should be designed for each patient, based on physical and psychological conditions, and consistent with life style and personal goals. Neurogenic bowel management also carries significant resource implications.

Epidemiology of the neurogenic bowel

The prevalence of faecal incontinence and constipation varies from 1% to 20% of the general adult population depending which definitions are applied (see Chapter 3). The prevalence of bowel dysfunction in people suffering from neurological disease or injury is higher.

Stroke is the commonest cause of neurological damage in Western countries, with an annual incidence rate between 300 and 500 per 100,000 population. Faecal incontinence has been reported by 23% of 135 consecutive stroke patients within one year (Brocklehurst et al., 1985). Older patients, women and those with the most severe strokes seemed to be most at risk (Nakayama et al., 1997).

Multiple sclerosis affects approximately one million young adults worldwide. Two-thirds of patients with MS complain of bowel problems (Hinds et al., 1990). The prevalence of constipation and/or faecal incontinence ranges from 39% to 73% (Chia et al., 1995; Nordenbo et al., 1996; Sullivan and Ebers, 1983). Bowel dysfunction is a source of considerable psychological disability for MS patients. In a study of 890 patients, the main factors limiting the ability of MS sufferers to work were spasticity, incoordination and bladder and bowel symptoms (Bauer et al., 1965), indicating that bowel dysfunction is a major hurdle to full rehabilitation.

Spinal cord injury is relatively common, with approximately 400,000 SCI individuals currently living in the UK. Glickman and Kamm reported that 95% of 115 consecutive SCI outpatients required at least one therapeutic procedure to initiate defaecation, and 50% needed help to manage their bowel (Glickman and Kamm, 1996). Faecal incontinence is reported by 15–25% of patients discharged from rehabilitation units (Krogh et al., 1997; Subbarao, 1987). Bowel function is a source of distress in over half of patients, and this is associated with the time required for bowel management and the
frequency of incontinence (Glickman and Kamm, 1996). In many surveys of SCI, subjects rank bowel dysfunction as one of their major life-limiting problems (Glickman and Kamm, 1996; Hanson and Franklin, 1976; Stone et al., 1990; White et al., 1993).

**Parkinson’s disease** affects one in a thousand people, and constipation and evacuation disorders are common in these patients, with slow transit or evacuation difficulty-type constipation in up to 50% of patients (Edwards et al., 1992).

**Autonomic neuropathy** (disease or degeneration of the autonomic nervous system) is associated with a wide range of gastrointestinal manifestations, such as nausea, bloating, vomiting, abdominal pain, constipation and faecal incontinence. Most knowledge is based on studies of diabetes mellitus. Constipation has been reported in 12% to 88% of diabetic patients, with a debated direct correlation with the incidence of autonomic neuropathy (Clouse and Lustman, 1989; Feldman and Schiller, 1983). Twenty per cent of diabetics complain of faecal urgency and episodes of incontinence, with evidence of decreased rectal sensation or impaired function of the anal sphincters, or both (Wald and Tunuguntla, 1984; Caruana and Wald, 1991; Sun et al., 1996).

**Neurophysiology of the gastrointestinal tract**

The neurological control of the bowel is the result of an intricate balance between the extrinsic nervous system, the enteric nervous system, and the intestinal smooth muscle cells.

**The extrinsic nervous system**

The extrinsic nervous system of the bowel is the nervous system which is external to the bowel itself. It consists of autonomic, sensory and motor nerves (Figure 17.1). The autonomic (smooth muscle, involuntary) nervous system of the bowel comprises an integrated complex system of parasympathetic (predominantly stimulating motility) and sympathetic (predominantly inhibiting motility) fibres. The parasympathetic vagal nerves originate from the medulla within the brain and supply motor and sensory input to the proximal (upper) colon. The parasympathetic sacral nerves originate from the spinal level S2 to S4 and contain both motor and sensory fibres to distal (lower) colon.

The sympathetic nerve supply arises from between the tenth thoracic and third lumbar segments. It is composed of fibres that synapse with enteric ganglionic plexuses within the colon wall.

**Somatic (voluntary) nerves** provide both sensory and motor supply to the large bowel and pelvic floor, via the vagus, the nervi erigentes (from S2 to S4), direct sacral root branches (from S1 to S5) and the pudendal nerve.

**The enteric nervous system**

The enteric nervous system is the internal nervous system of the gut itself. It has an essential role in the control of motility, blood flow, water and electrolyte transport, and acid secretion in the digestive tract. This system is organised in ganglionated plexuses within the bowel wall itself and is separated from the autonomic nervous system. The enteric nervous system has several components: sensory receptors (mechano- and chemoreceptors), interneurons (processing input and controlling effector units, motor and secretory) and effector motor neurones involved in motility of the gut. Enteric nervous control of the bowel is modulated through connections from the autonomic nervous system to the brain. The enteric nervous system has the unique capacity to mediate reflex activity independently of input from the central nervous system. This is why even people with complete destruction of central neurological input to the bowel maintain some gut motility – the intrinsic nervous system keeps the bowel working to at least some extent (unlike the bladder, which can become totally atonic).

A neurogenic bowel can result from any pathological processes involving any component of the central nervous system, and/or the
extrinsic and the intrinsic innervation of the bowel. Bowel dysfunction can either be directly due to the neuropathological process (e.g. spinal cord injury), to common causes unrelated to the neurological disease (e.g. low dietary fibre, insufficient liquid intake, difficulty accessing the toilet), or to a combination of the neuropathological process and unrelated factors. Damage to the frontal lobe of the brain can lead to emotional disturbance, altered social relationships relating to bowel behaviour, lack of voluntary control of the pelvic floor or ignoring the call to stool because of reduced awareness. When sensory (afferent) input to the brain is impaired, a call to stool may be diminished and be responsible for constipation or faecal incontinence. Both can also coexist if incontinence is secondary to faecal impaction (misleadingly labelled ‘overflow diarrhoea’). Moreover, impaired anorectal sensation may co-exist with
impaired mobility and inability to cope with urgent defaecation. Thus, bowel function and continence can be altered as a result of multiple causes including: altered cognitive function, loss of mobility and independence in toileting, loss of control of the pelvic floor striated musculature, loss of ano-rectal and pelvic floor sensory function, colonic dysmotility and/or use of drugs and subsequent side effects on colonic motility and sensory function.

Assessment of the neurogenic bowel

Assessment of bowel dysfunction in neurologically impaired patients requires a holistic approach that takes into account the patient’s environment, physical and psychological disabilities, and general medical conditions (Table 17.1). This will usually require a multidisciplinary team.

Assessment of the environment is essential. Bowel management is often a source of difficulty in the reintegration into the home and the community for the disabled person. This will include assessment of available health and social care resources, so that impractical and unrealistic care plans are avoided. A social worker will often become involved to assess the local healthcare network and availability of community support for which an individual may qualify. Family and social supports should be assessed in collaboration with the patient, the general practitioner and the family. Home assessment will require a home visit by an occupational therapist. Some disabled people have bowel problems that are only related to toilet accessibility (see Chapter 23).

Assessment of the individual

Assessment of the individual with a neurological disorder or injury should include assessment of impairment/disability/handicap, psychological/cognitive function and compliance with previous and current treatment.

A neurological examination will yield information about the completeness, the level, and severity of the impairment and disability. This is made easier with a validated scoring system. A physiotherapist or a rehabilitation therapist should assess the handicap: i.e. sitting tolerance, upper body strength, hand and arm function, spasticity, and transfer skills.

A drug history should be obtained, as a number of drugs commonly prescribed to neurological patients have the potential to alter bowel function: for example, oxybutynin, diuretics, antacids, antidepressants and narcotic drugs may decrease bowel motility; whereas antibiotics and dantrolene sodium may provoke soft stool, diarrhoea and faecal incontinence.

An assessment of cognitive function includes the individual’s ability to learn and to direct others in assisting in the provision of bowel care. Awareness of the level of cognitive function allows the design of a realistic bowel programme. For people with severe cognitive impairment or learning disabilities, this assessment may need referral to specialised units (e.g. a neuropsychologist).

Assessment of mood can be made by the hospital anxiety and depression scale (Zigmond and Snaith, 1983). Referral to local psychiatric or psychological services may be considered for those patients where there is clinical suspicion of mood disturbance. Low mood or anxiety states may lead to inaccurate assessment of cognitive function. Coping strategies and compliance are important, as many bowel care regimes fail due to poor compliance and inadequate coping strategies (Wineman et al., 1994). This may be aggravated by altered cognitive function, mood disturbances, the level of support available or unrealistic goals.

Assessment of goals, lifestyle and commitments will enable the setting of appropriate aims for bowel care. For example, the bowel programme of a young woman who is wheelchair independent and works full-time will probably be different to that of a bedbound elderly man cared for by his wife. Self-rated quality of life can help to identify areas of specific need and monitor the outcome of bowel care programs. Patients and clinicians may disagree on perception of health and disability (Rothwell et al., 1997).

The whole individual assessment should be
repeated whenever progression or relapse of the neurological process occurs.

**Assessment of bowel function**

This involves a thorough history, full neurological and gastroenterological examination, and in some cases targeted investigation.

A gastrointestinal assessment should include symptoms and disease prior to the neurological disease or injury (e.g. abdominal surgery, inflammatory bowel diseases, irritable bowel syndrome, laxative dependency) and current gastrointestinal symptoms.

Symptom diaries, filled in over 1–2 weeks (Chapter 6), are useful adjuncts, particularly when memory is impaired. Constipation and faecal incontinence embrace a range of symptoms, with different meanings for different individuals. In neurological patients, constipation can be defined as small, unsatisfactory, inefficient or absent bowel movements after two or more attempts at toileting or bowel care. Faecal incontinence is the involuntary loss of faeces and/or flatus, and is generally characterised as urge incontinence or passive leakage. No published criteria have been validated in neurological patients.

### Table 17.1: The assessment of the neurogenic bowel

*The assessment of the neurogenic bowel is seen as a holistic approach to assessing the bowel of a neurological patient in her/his environment. In brackets is the most appropriate method (see text for details).*

<table>
<thead>
<tr>
<th>Environment Assessment</th>
<th>Individual Assessment</th>
<th>Bowel Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community and health care (community liaison team, social worker)</td>
<td>Impairment, disability and handicap</td>
<td>Past/current gastrointestinal symptoms (questionnaire, bowel diary)</td>
</tr>
<tr>
<td>Socio-familial (general practitioner, social worker)</td>
<td>Cognitive function, ability to learn and to direct others</td>
<td>Past/current bowel programme (relatives, carers)</td>
</tr>
<tr>
<td>Home, workplace (visits)</td>
<td>Mood, coping strategies, compliance, quality-of-life</td>
<td>Motor assessment</td>
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<tr>
<td></td>
<td>Concomitant diseases and drugs</td>
<td>Abdominal wall (physical examination)</td>
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<tr>
<td></td>
<td></td>
<td>Pelvic floor (physical examination, balloon expulsion, defaecography)</td>
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<tr>
<td></td>
<td></td>
<td>Anal sphincters (physical examination, ano-rectal manometry, endoanal ultrasonography)</td>
</tr>
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<td></td>
<td></td>
<td>Sensory assessment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sacral reflexes (physical examination)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ano-rectal thresholds (balloon distension, mucosal electrostimulation)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Motility assessment</td>
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<tr>
<td></td>
<td></td>
<td>Bowel transit (radio-opaque markers colonic transit study)</td>
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</tbody>
</table>
Details of past bowel care programmes should be obtained. It is important to clarify the reasons for failure if this failed, as frequently inadequate techniques, or wrong dosage or timing of drugs can be found. The patient should be asked about her/his satisfaction with the programme to determine whether there are specific problem areas. Duration of care and the occurrence of complications should be recorded (faecal incontinence, constipation, difficult evacuation, nausea, abdominal distension, pain, rectal bleeding, pressure sores, dysreflexia). The current bowel programme over the last two weeks should be detailed, including: diet, fibre intake (this may require a one-week food record and assessment by a dietitian), use of assistive methods for defaecation, and the dosage and timing of pharmacological agents. Family members and carers often provide helpful additional information.

Physical examination

This includes assessment of the abdominal wall, pelvic floor musculature, anal sphincter and rectum. It will focus on the neurological system.

Assessment of the abdominal wall (looking for hernias) and muscles is done at rest, during voluntary contraction of the muscles and involuntary response to coughing and sneezing. Assessment of the pelvic floor is performed at rest and during straining. A decreased tone and weak muscles will be seen when somatic motor neurones in the S2–S3 segments of the anterior horn of the spinal cord are impaired. Spastic muscles incapable of relaxation are present in some MS patients. Perineal descent is sometimes seen as a result of chronic straining, which can cause external rectal prolapse and eventually passive leakage of stool.

Assessment of the anal sphincters starts with examination of the sacral reflexes. These consist of motor responses in the pelvic floor and sphincter muscles, evoked by physical or electrical stimulation of sensory receptors (Uher and Swash, 1998). The anocutaneous reflex is a contraction of the external anal sphincter (EAS) in response to touch or pin stimulus to the perianal skin (all 4 quadrants to be assessed), which is mediated by the pudendal nerves (S2–S5). The bulbocavernous reflex is elicited by pinching or pricking the dorsal glans penis or by pressing the clitoris, and palpating for EAS contraction within the anal canal. Presence of both reflexes suggests the integrity of a conus-mediated (S2–S3) reflex activity. Anal cough reflex is a contraction of the EAS elicited by a rise in intra-abdominal pressure during vigorous coughing or sneezing. This reflex can be detected after spinal cord trans-section and might be initiated by stretch receptors in the EAS and/or surrounding muscles.

Digital examination of the anal sphincters will give a notion of the resting tone. Strength of the EAS and puborectalis can be assessed by requesting the patient to tighten the pelvic floor as if to prevent defaecation and to cough. Digital rectal examination should also exclude stool impaction (rock-hard stool), rectocoele or a tumour.

Sensory assessment of the lower limbs, pelvic floor and anus is performed with the pin-prick technique.

Further investigations of constipation and faecal incontinence

These will follow a thorough assessment, and the choice of test will depend on the clinical problem. Frequently neurological patients will benefit from objective assessment of their bowel dysfunction. Investigations assist in excluding treatable non-neurological causes of bowel dysfunction, and are used to identify those patients who may be candidates for more invasive management such as surgery. Investigation of other bowel symptoms such as rectal bleeding or weight loss may be indicated (see Chapter 7). Although haemorrhoids are frequent in neurological patients, rectal bleeding should never be attributed to them without proper investigations.

Ano-rectal physiology tests (see Chapter 8 for more detail)

Manometry is important to define weakness of one or both sphincter muscles. Manometry also allows recognition of the inability to
evacuate adequately from the rectum related to a lack of sphincter relaxation, also named ‘anismus’ or ‘pelvic floor dyssynergia’. Balloon expulsion test with a 50 ml water-filled rectal balloon will help identify those patients with pelvic floor dyssynergia (Jameson et al., 1994).

Balloon distension with an air-filled rectal balloon will provide a gross measurement of sensory function and compliance. The thresholds for the first perceived sensation (smallest volume of rectal distension), the sensation of urgency to defecate, and the maximum tolerable volume are measured. These thresholds depend on the neural balance between the ability to feel, retain and tolerate rectal content by inhibiting defaecation and by voluntary contracting the pelvic floor muscles (Caruana et al., 1991). Local diseases (e.g. proctitis) can also alter these thresholds. The recto-anal inhibitory reflex (RAIR) is induced by rapid rectal distension. A sudden fall in anal pressure is caused by internal anal sphincter relaxation. RAIR seems to be mediated by intramural myenteric neurones – it is not abolished by spinal transection but is abolished in Hirschprung’s disease.

Mucosal electrostimulation using an electrical stimulus passed across the anal and rectal mucosa can obtain a quantitative assessment of ano-rectal innervation and may help distinguish between functional and neurological disorders (Kamm and Lennard-Jones, 1990). When it is uncertain whether constipation is directly related to impaired central innervation of the gut, or is ‘idiopathic’, this test may be helpful. In patients with neurological disease affecting the hindgut innervation, the rectal mucosal electrical sensory threshold is usually grossly abnormal. Previous studies in patients with cord injury have demonstrated the sensitivity of electrical sensation testing in defining impaired innervation (Emmanuel and Kamm, 1999). In some patients its normality may provide a reassuring basis on which to try to behaviourally correct the constipation, in the expectation that the bowel dysfunction is reversible.

**Radiological investigations**

Endoanal ultrasonography and transit studies may be indicated in incontinence or constipation respectively (see Chapter 9). Defaecography and proctography are reserved for patients in whom abnormalities, such as slow or incomplete rectal emptying, failure of puborectalis and anal sphincter muscles relaxation, a rectal prolapse, a rectocele, or a megarectum are clinically suspected.

In summary, a methodical history will assess the patient’s environment, impairment, disability, handicap, coping strategies and compliance. Careful physical examination should show which element of the bowel function is essentially impaired. Targeted investigations will verify whether constipation is predominantly due to impaired colonic motility or inadequate evacuation, and faecal incontinence mostly due to a weak anal sphincter or to impaired sensory function.

**Autonomic dysreflexia**

Autonomic dysreflexia (also known as hyperreflexia or dysautonomia) is a potentially life-threatening complication (Banwell et al., 1993), that can occur in some people with a spinal cord injury at level T6 or above (those with injuries at T6 to T10 may also be susceptible). Patients usually present with elevated blood pressure and bradycardia. If not treated promptly and correctly, it may lead to seizures, stroke and death. The pathophysiology of dysreflexia is related to an overactivity of the sympathetic nervous system. Noxious stimuli to intact sensory nerves below the injury lead to relatively unopposed sympathetic outflow and blood pressure elevations. Parasympathetic outflow through the vagus nerve can cause reflexive bradycardia but cannot compensate for severe vasoconstriction. Anything that would have been painful, uncomfortable or physically irritating before the injury may cause dysreflexia. Symptoms are a restless feeling and shortening of breath, pounding severe headache, blurred vision and dizziness, nasal congestion, goose pimples, profuse
sweating, and flushing or blotching of the skin (particularly above the level of injury). Most causes are related to the bladder, such as a blockage in a drainage device, an infection, bladder spasms, or stones. The second most common cause is a full bowel. Any stimulus to the rectum, such as digital stimulation or manual evacuation, can also trigger dysreflexia. Treatment must be initiated quickly: head elevation, pressure release, adequate urinary drainage system, or emptying the bowel, as appropriate. Other possible causes are: fractures, appendicitis, sexual intercourse, period pains, pregnancy or labour. Blood pressure should be treated until the cause is found and eliminated.

Prevention is very important, as most causes can be avoided. Medications to treat dysreflexia and prevent increases in blood pressure may be given prior to bowel care: a rectal anaesthetic ointment – ten minutes before, glycerol trinitrate 250 mcg, nitroglycerine paste 2 inches – on the skin above level of injury, nifedipine 10 mg – not sublingual, as it can cause abrupt hypotension. Blood pressure should be monitored for at least two hours after it has stabilised at under 150 mmHg systolic. If dysreflexia persists, alternatives for bowel care must be considered (e.g. surgical approaches). Table 17.2 (overleaf) gives a patient information sheet about autonomic dysreflexia (reproduced by permission of Spain Rehabilitation Center, USA).

Management of the neurogenic bowel

Bowel management in neurological patients is currently empirical, based on clinical experience rather than research-based evidence. While there is a considerable literature on the prevalence and pathophysiology of bowel dysfunction in neurological disease or injury, there has been remarkably little research done on practical management. There are about 150 non-randomised non-controlled non-comparative and anecdotal trials on different methods of managing a neurogenic bowel. Most methods emphasise the importance of a high fibre diet, drinking more fluid, triggering the gastro-colic response by a hot drink, increasing physical activity, a scheduled regular bowel routine and discouraging the long-term use of laxatives, and frequently advocate rectal stimulation with suppositories. There is no evidence to support any of these recommendations (Wiesel et al., 2001). A practical approach is whenever possible to tailor the bowel management to the specific problems revealed by the assessment described in Table 17.1. However, there are a number of general principles that apply. Many people will need active bowel management, rather than waiting for problems or complications to develop.

Managing the bowel will consider the person, within the context of more general problems and the daily environment (Table 17.3, page 192). The bowel programme will be a personalised plan designed to help the neurological patient gain control of her/his bowel, taking into account attendant care, personal obligations and goals. It should provide predictable and effective elimination of stool, and is designed to be easily applied in the individual’s home setting. It should be reviewed as needed, and at least once a year to ensure ongoing relevance. A bowel diary is a key part of this review. ‘Bowel care’ is the term employed for assisted elimination of stool and may be part of the bowel programme. It consists mainly of conservative assistive and pharmacological methods. For a few patients, surgical methods will have to be considered.

Bowel programmes

A safe, private and pleasant environment is the first step in a successful bowel programme. This may require appropriate equipment such as hand rails, transfer board, a raised and padded commode seat, back support, safety straps, foot blocks, a suppository inserter or a digital stimulator, anal plugs, continence products, roll-in shower chair and other home adaptations (see Chapter 23). The type of equipment required is based on the individual’s functional status and the living environment as assessed by physiotherapists and occupational therapists. Preven-
tion of pressure sores should be addressed when bowel care is prolonged (for example the use of a padded seat or commode). Perineal cleaning can be problematic with limited dexterity, and will sometimes be the cause of minor soiling. A conventional U-shaped toilet seat (opening in either the front, the sides, or the rear) usually allows adequate access, and in particular cases a perineal shower or bidet can be helpful. Regional and national patient associations are a useful source of support and information on how to obtain or purchase equipment (see Appendix II). The Internet provides an opportunity for disabled people to get information and purchase products. However, material and information provided by commercial sites should be handled with caution, and in most instances suitable equipment should be available through health or social services.

Independent bowel care is ideal but not possible for some patients. The patient may be able to direct a carer. Carers who are involved in bowel management should obtain informed consent, especially when invasive procedures like digital examination and manual evacuation are required (Addison and Smith, 2000). Psychosocial difficulties are frequent in disabled patients. Emotional support for both patient and carer is important to improve compliance with and success of any bowel care regime. This may require formal counselling and regular respite for the carer. Excessive involvement in bowel management of the partner, family members, relatives or friends may not be appropriate in many cases.

Communication between the patient, carer, district nurse, general practitioner (GP) and the multidisciplinary assessment team is vital. A clear written bowel care regime with a single contact person in times of difficulty is useful. Often issues of dose titration or adjustment of routine can be dealt with over the phone, saving an immobile patient an unnecessary trip to outpatients or the GP’s surgery, and preventing inappropriate attendance at accident and emergency departments.

Bowel training, with behavioural modification of self-initiating defaecation and positive reinforcement of this process, have successfully prevented constipation and soiling in children with neurological bowel (Jeffries et al., 1982; King et al., 1994; Younoszai, 1992). Preliminary data on bowel training in cerebrovascular accidents patients (CVA) and SCI adults are encouraging (Badiali et al., 1997; Munchiando and Kendall, 1993; Stiens et al., 1997; Venn et al., 1992).

Scheduling bowel evacuation after a meal should be encouraged to take advantage of the gastro-colic response. Delaying bowel movements is frequent in disabled patients but is usually unwise. Eventually the rectum will adapt to increased bulk of stool, the urge to defaecate might progressively diminish, the rectum can even dilate (megarectum) and result in impaction. Although investigators have reported some impairment of the gastro-colic response in a few SCI and MS patients, results have been inconsistent and it is worth trying to utilise this response for many people.

Fibre intake should be adequate on the premise that the bowel will respond with a decrease in transit time and an increase in stool frequency. Contradictory results have been published in neurological and disabled patients when high fibre diet was consumed (Ashraf et al., 1997; Astarloa et al., 1992; Badiali et al., 1997; Cameron et al., 1996; Levine et al., 1992; Liebl et al., 1990). However, it has been shown that the average daily fibre intake is low in SCI (between 7 and 14 grams per day) (Kirk et al., 1997). There is indeed a fundamental difference between adequate (more than 25 g/day) and ‘excessive’ fibre diet. Consultation with a dietitian can be helpful to clarify misconceptions and provide sound advice.

An adequate fluid intake is recommended. Some individuals may try to solve common bladder problems (incontinence, urgency, frequent catheter bag emptying) by drastically reducing their fluid intake. Dehydration may occur, triggering fluid reabsorption from the faeces which can then harden and be difficult to evacuate. Similarly, prolonged colonic transit time (i.e. secondary to drugs) can result in excessive fluid absorption and hardened stool.
AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia (AD) is a potentially life-threatening condition that occurs in individuals with a spinal cord injury at level T6 or above. Patients usually present with elevated blood pressure and bradycardia. Noxious stimuli to intact sensory nerves below the injury lead to relatively unopposed sympathetic outflow and dangerous blood pressure elevations. Parasympathetic outflow through cranial nerve X (vagus) can cause reflexive bradycardia but can’t compensate for severe vasoconstriction.

Common signs and symptoms may include

<table>
<thead>
<tr>
<th>BRADYCARDIA</th>
<th>CHILLS WITHOUT FEVER</th>
</tr>
</thead>
<tbody>
<tr>
<td>HYPERTENSION</td>
<td>SEIZURES</td>
</tr>
<tr>
<td>POUNDING HEADACHE</td>
<td>SWEATING ABOVE LEVEL OF INJURY</td>
</tr>
<tr>
<td>NASAL CONGESTION</td>
<td>SKIN FLUSHING ABOVE LEVEL OF INJURY</td>
</tr>
<tr>
<td>BRONCHOSPASM</td>
<td>GOOSE BUMPS ABOVE LEVEL OF INJURY</td>
</tr>
<tr>
<td>BLURRED VISION</td>
<td>APPREHENSION OR ANXIETY</td>
</tr>
</tbody>
</table>

Follow the examination tree below to eliminate any noxious stimuli below level of injury. A drop in blood pressure will occur with the removal of the stimuli. Seizures, stroke, or death may occur if stimuli are not immediately removed.

Examination tree

Sit up and take blood pressure in both arms (repeat blood pressure every 3 minutes and between steps).

Important note – Normal systolic BP for an individual with an SCI above T6 can be in the 90-110 mm Hg range. If blood pressure is elevated, give medications as indicated. Use an antihypertensive with rapid onset and short duration while the causes of AD are being investigated.

Look for noxious stimuli below level of injury

Check Bladder for Distension: Catheterize bladder using 2% lidocaine jelly. If indwelling catheter already in place, inspect for kinks, folds, constrictions or obstructions. Irrigate or replace the catheter to ensure patency – RELIEF? – collect U/A and C/S (irritation may be due to infection). Assess for any urologic obstruction such as kidney or bladder stones.

Check Bowel: Anesthetize using lidocaine jelly 2% (wait 5 minutes) prior to checking for impaction. Remove impaction and recheck blood pressure – RELIEF? – Evaluate for high impaction.
Table 17.2: Autonomic dysreflexia – information sheet (continued)


*Treatment reminders*

1) Sit patient up. 
2) Check BP often and treat elevated systolic blood pressure (> 150) until cause is found and eliminated. 
3) Medications commonly used for elevated BP are:
   a) Nitroglycerine paste. Apply 1-2 inches to skin q2hrs above the level of injury. May wipe off if BP stable and reapply if needed. Avoid sublingual which can cause abrupt hypotension. 
   b) Nifedipine 10 mg capsule (immediate release form). May repeat in 20-30 minutes If needed. Avoid sublingual which can cause abrupt hypotension. 
   c) IV-Antihypertensives. These are secondary agents to be utilized In a monitored setting. 
4) Treat symptomatic hypotension by laying down the individual and elevating the legs. 
5) Anesthetize noxious stimuli prior to removal to prevent exacerbation of AD. 
6) Monitor symptoms and BP for at least 2 hours after the resolution of an AD episode. 
7) Admit the patient if response to treatment is poor or cause has not been identified. AD can lead to seizures, stroke, or death.

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*Acknowledgement:* Autonomic Dysreflexia poster published by The University of Alabama at Birmingham Rehabilitation Research and Training Center on Secondary Conditions of Spinal Cord Injury and the UAB Model SCI System of Care. UAB RRTC is supported by grant #H133B980016 from the National Institute on Disability and Rehabilitation Research, Office of Special Education and Rehabilitative Services, United States Department of Education, Washington, D.C.
Although target fluid intake will essentially depend on renal function and bladder management, adequate fluid intake is advised for the prevention of hard stool. A loaded bowel may interfere with urethral or suprapubic catheter drainage.

Bladder management is closely linked with the bowel. Frequently neurological patients have both a neurogenic bladder and a neurogenic bowel (Bauer et al., 1965; Hanson and Franklin, 1976; Kang et al., 1996). Moreover, bladder catheterisation may diminish the use of a toilet and lessen opportunities to empty the bowel. Co-operative management with a urological team will sometimes be needed.

**Assistive methods**

A number of methods may assist effective emptying of the rectum when this does not happen adequately or spontaneously.

Abdominal massage, push-ups, Valsalva manoeuvre (holding the breath and forcibly trying to exhale against a closed glottis, thereby creating raised intra-abdominal pressure and a bearing-down effect), deep breathing and forward-leaning position are some of the techniques used to assist bowel emptying. Although very little research has been done on the use of these techniques (Ernst, 1999), they may aid bowel evacuation by increasing abdominal pressure. In a survey of 277 individuals with SCI, the seated position was rated as faster, more effective and more convenient than bowel care completed in bed (Nelson et al., 1993). To minimise the risk of vesico-ureteral reflux, Valsalva should not be performed with a full bladder and is contraindicated for individuals with cardiac problems and hypertension. Prolonged straining may eventually predispose to haemorrhoids and even rectal prolapse.

**Digital ano-rectal stimulation** (Figure 17.2) triggers peristalsis of the left colon. It is performed by gently inserting a gloved lubricated finger into the rectum and slowly rotating the finger in a circular movement against the rectal mucosa. Rotation is continued until relaxation of the bowel wall is felt, flatus passes, stool passes, or the internal anal sphincter contracts. It should last 20 seconds, and stimulation longer

*Figure 17.2  Digital stimulation.*
than one minute is seldom necessary. Stimulation is repeated every 5 to 10 minutes until stool evacuation is complete, or no stool has been passed after at last two stimulations. Individuals who lack sufficient hand function for gloving and digital stimulation may be candidates for a digital stimulator. Applied mostly to people with high SCI injury (Glickman and Kamm, 1996), CVA patients may also benefit from a programme of daily digital stimulation (Munchiando and Kendall, 1993). It is as yet unproven if this method is effective in other neurological patients, but it should be considered.

**Manual evacuation** is the digital removal of faeces from the rectum. This method should only be performed when required, after informed consent, and by skilled persons. The Royal College of Nursing has recently reviewed the procedure and safety points (Addison and Smith, 2000). Manual evacuation is performed by inserting a gloved lubricated finger into the rectum to break up or hook stool and pull it out. Anaesthetic ointment should be used to decrease the noxious stimuli and avoid producing autonomic dysreflexia in those prone to this (see above). Many people with low SCI injury rely on this method once or more per day to stay continent (Glickman and Kamm, 1996), and there is often no viable alternative to the long-term use of manual evacuation in those with low SCI.

**Biofeedback** aims to condition patients to be more sensitive to a stimulus distending the rectum and to improve pelvic floor function. It has been shown that biofeedback is effective in constipation and faecal incontinence in some MS adults with limited disability and a non-progressive disease course (Wiesel et al., 2000).

**Management of constipation and faecal incontinence in neurological disease**

**Prevention and management of constipation**

Prevention of constipation should be the first objective. Some people will prefer constipation to faecal incontinence, either to avoid soiling or as being more socially acceptable. However, this is associated with complications such as impaction, ‘overflow diarrhoea’, bladder infections, increased spasticity, or the life-threatening autonomic dysreflexia in SCI patients. Patient and carers’ education should be undertaken to ensure regular bowel emptying by means of general measures and assistive methods.

Attention to diet and a balanced increase in diverse sources of fibre may be sufficient to relieve mild constipation in some patients. For further information see Murray and Emmanuel (2002). Nevertheless, when patients are complaining of constipation, pharmacological methods to assist bowel emptying will often be required. Bulk-forming agents, rectal stimulants, oral laxatives and enemas should be tried according to patient’s assessment and preferences. Surgical options should be discussed early in the management and considered at an appropriate time if conservative methods are not any longer realistic. In selected cases a progressive approach to the most suitable surgical method can be made by starting with retrograde enema, moving to antegrade colonic enema (ACE), and then perhaps considering a stoma. Complex surgery should be performed in specialised centres. Specific details of each of these methods are given below.
discourage its use (Sussman and Dorian, 1990). These agents (ispaghula, methylcellulose) have a role in the management of constipation, and patients should be encouraged to achieve adequate dietary fibre with a supplement of natural bran, if fibre intake is less than 25–30 g/day.

Faecal softeners include different oils (mineral, paraffin, arachis) which are given orally or by enema to penetrate or lower the surface tension and therefore soften stool. Randomised controlled trials are lacking to determine the role of stool softeners in the prevention and treatment of constipation (Hurdon et al., 2000). Many oral faecal softeners may increase the uptake and toxicity of drugs, interfere with the absorption of fat-soluble vitamins and give rise to lipoid pneumonia if inhaled (Gattuso and Kamm 1993). The authors regard the evidence for the efficacy of oral faecal softeners as unproved and unsafe, discouraging further use.

Rectal stimulants (e.g. glycerine, bisacodyl) can be used alone or in combination with digital stimulation when this latter alone is inefficient. They have the advantage of predictability in terms of time of response and can trigger a bowel movement fifteen to thirty minutes after insertion. A glycerin suppository acts as a mild local stimulus and lubricating agent. Bisacodyl is a contact irritant that acts directly on the colonic mucosa, producing peristalsis throughout the colon. Glycerin is used in individuals who experience adverse reactions to the bisacodyl suppository, have a fast response to bisacodyl, or are making a transition from bisacodyl to mechanical stimulation. Bisacodyl may be compounded with a vegetable oil or a polyethylene glycol (PEG) base. There is strong evidence that PEG–based bisacodyl suppositories can produce significantly more rapid onset of defaecation and shorten the total bowel care time in SCI patients when compared to hydrogenated vegetable oil–based bisacodyl suppositories (Frisbie, 1997; House and Stiens, 1997; Stiens, 1995; Stiens et al., 1998). Carbon dioxide generating suppositories produce reflex – explosive and unpredictable – defaecation in response to colonic dilatation, they are not recommended. It may be necessary to remove stool prior to the insertion of a suppository against the rectal mucosa. When hand function is insufficient a suppository inserter can be used (Figure 17.3).

Oral laxatives can be prescribed to increase bowel frequency if other measures have failed, and particularly when transit is documented to be slow. Any laxatives should be titrated to produce a satisfactory response without causing liquid stools and faecal incontinence. Every agent should be tried for a minimum of 3–4 weeks before progressing to the next agent. All laxatives share, to some degree, the potential for dose-dependent side effects, including abdominal cramping, diarrhoea and electrolyte imbalance. Although the chronic ingestion of anthranoid laxatives (senna) has been blamed for the development of the cathartic colon – an

![Figure 17.3 Suppository inserters.](image-url)
atonic non-functioning colon – there are no definitive studies which have demonstrated this (Gattuso and Kamm, 1994).

**Osmotic laxatives** include mixed-electrolyte solutions containing polyethylene glycol (PEG, e.g. Movicol) or nonabsorbable sugars (lactulose, lactitol and sorbitol) that cause distension and stimulation of the bowel. PEG solutions act more rapidly with stronger efficacy and less side effects than nonabsorbable sugars (Attar et al., 1999; Corazziari et al., 2000; DiPalma et al., 2000). After four weeks, patients treated with PEG had a higher bowel frequency than patients treated with lactulose (Attar et al., 1999). In two double-blind, placebo controlled, parallel group studies, patients treated with small daily doses of PEG reported higher bowel frequency, less frequently straining at defaecation and reduced consumption of laxative, when compared to the placebo group (Corazziari et al., 2000; DiPalma et al., 2000). Nonabsorbable sugars are associated with the production of gases due to colonic fermentation, bloating, cramping and flatulence. By lowering the stool pH they also are aggressive to the skin and present a greater risk of pressure sores if faecal incontinence is present. Besides, PEG solutions were recently found to be safe to treat faecal impaction (Culbert et al., 1998; Ferguson et al., 1999; Ungar 2000), which could be useful when manual evacuation by a trained person fails. Although no trial has yet specifically included neurological patients, the efficacy of PEG solutions for constipation, the possibility of fine-tuning the prescription and the good tolerance, are reasons to make this agent an attractive osmotic laxative.

**Saline oral laxatives** (salts of sodium, magnesium or potassium) contain relatively nonabsorbable cations and anions that osmotically increase intralumenal water content and stimulate motility. They induce complete bowel evacuation in two to six hours and may give rise to electrolyte imbalance and water retention.

**Stimulant oral laxatives** include polyphenolic compounds (bisacodyl, phenolphthalein, sodium picosulphate) and anthraquinone-containing substances obtained from senna. Phenolphthalein is best avoided because it undergoes an enterohepatic circulation and can cause a rash. Sodium picosulfate is hydrolysed by colonic bacterial flora. Bisacodyl is hydrolysed by intestinal enzymes and can act on both the small and the large intestine to stimulate motor activity and to inhibit glucose and sodium absorption. Its effect on the small bowel is a disadvantage, in comparison with that of anthranoid glycosides, and it could be difficult to adjust the dose to produce soft, formed stools. Senna increases propulsive activity by altering electrolyte transport and increasing intraluminal fluid; it also exerts a direct stimulant effect on the myenteric plexus, which increases motility. Senna is widely used among neurological patients, and is best taken in the evening or at bedtime with the aim of producing a normal stool next morning. Senna and Bisacodyl are reliable and effective agents, which can be titrated to produce a satisfactory response without causing the stools to become liquid and/or cause excessive urgency (Gattuso and Kamm, 1994; Schiller, 1999).

**Small-volume enemas** have been recently used to assist impaired defaecation. A 4 ml liquid suppository mini-enema is a combination of liquid docusate and glycerine in a PEG base. In SCI patients a mini-enema (Therevac) shortened the time between insertion of the stimulant and evacuation (Dunn and Galka, 1994; House and Stiens, 1997). Although availability of these mini-enemas and cost are currently problematic, larger trials are urgently needed.

**Large-volume enemas** (tap water, phosphate) should be reserved for special cases when all other conservative methods have failed. Sodium, potassium or phosphate enemas act directly on colonic mucosa, causing an influx of water and electrolyte which stimulate bowel evacuation in response to distension. Their onset of action is rapid and unpredictable (two to six hours). Frequently they result in abdominal cramping and watery bowel movements. There is a risk of electrolyte disturbances, recto-anal trauma, bowel perforation, bacteraemia and colonic infections. Large-volume enemas
should be if possible avoided in the routine management of the neurogenic bowel for reasons related to side effects, the need for independent self-administration, healthcare cost and the availability of other conservative or surgical methods.

**Retrograde colonic irrigation (bowel washout)** is sometimes used in Europe and Japan. Most methods of irrigation have been derived from colostomy irrigation techniques. Hand-warm tap water or saline solutions are instilled through a catheter incorporating an inflatable rectal balloon, or via an anal cone, and have been successfully used in spina bifida and meningomyelocele children (Blair et al., 1992; de Kort et al., 1997; Eire et al., 1998) (Figure 17.4: Shandling catheter). A closed rectal washout system which infuses liquid by gravity through a retention-cuffed speculum inserted in the rectum has been developed for people with faecal incontinence (Iwama et al., 1989). Seventy-nine per cent of 32 ambulatory patients who suffered mainly from soiling were helped by a daily rectal washout (Briel et al., 1997).

**Pulsed irrigation enhanced evacuation** is a mechanically-assisted method of clearing faecal impaction using intermittent pulsed irrigation of small amounts of warm tap water administered rectally (Kokoszka et al., 1994). The procedure is supposed to rehydrate faeces, to break up impacted stools and to promote peristalsis (Puet et al., 1997). However, safety, complexity, comfort and cost of this procedure have yet to be fully evaluated. No reports have yet been published on its efficacy for routine bowel care, and therefore it cannot be recommended for that purpose.

**Surgical options for constipation**

**An antegrade continence enema (ACE)** is made possible after a surgical procedure (Malone procedure) giving direct access to the caecum through the appendix or a ‘neo-appendix’ by means of a small stoma and use of a small catheter (Malone et al., 1990) (see Chapter 14). Enemas are usually performed daily or on alternate days, using tap water or a mixture of phosphate and saline. Bowel management and quality of life improved in some adults with severe neurogenic bowel dysfunction (Christensen et al., 2000). Response to washout is individual, and it is worth experimenting with volumes, temperatures and fluids, with or without the addition of enemas, to find the optimum. If constipation is severe in patients with intractable and disabling soiling, it might be worthwhile to treat these patients with an ACE. This procedure could also be a ‘bridge’ towards the acceptance of a stoma. The technique has relatively little morbidity except for abdominal pain during enema and stoma stenosis that may require dilation or surgical

![Figure 17.4 Shandling catheter.](image)
revision. It has been successful in neurological children for managing combined constipation and faecal incontinence (Driver et al., 1998; Liptak and Revell, 1992). Preliminary reports in adult patients with severe faecal incontinence or impaired bowel evacuation are encouraging (Christensen et al., 2000; Krogh and Laurberg, 1998; Teichman et al., 1998a; Yang and Stiens, 2000). Overall satisfaction with the ACE was high or very high. Faecal incontinence, toileting time, bowel medications, impact on social activities and quality of life were reduced. This procedure can be discussed for selected patients with intractable constipation and faecal incontinence (Bruce et al., 1999) in whom conservative management of bowel care has failed, or in bowel related dysreflexia (Teichman et al., 1998b).

A stoma to shorten and simplify bowel care and to improve quality of life has been advocated in SCI (Craven and Ettchells, 1998; Kelly et al., 1999; Saltzstein and Romano, 1990; Stone et al., 1990). When bowel emptying is ineffective using conservative methods or when bowel care requires unacceptable amount of time, inaccessible resources, or triggers complications (pressure sores, dysreflexia), a stoma is an appropriate alternative (see Chapter 15). Terminal ileostomy or terminal colostomy should be preferred rather than segmental resection of the bowel. Broad assessment of body image, lifestyle issue and independence in management of the stoma should be considered. A stoma has been used to deal with faecal incontinence in SCI patients (Frisbie et al., 1986).

Direct electrical stimulation of sacral anterior root has been attempted to promote bowel emptying in people with SCI. Stimulation is achieved by radio-frequency activation of a subcutaneous receiver (Brindley stimulator) that stimulates S2, S3, and S4 nerve roots bilaterally. Studies that have reported beneficial effects on colonic transit time, frequency of defaecation and time required for defaecation, have been based on observations and uncontrolled small series (Binnie et al., 1991; Chia et al., 1996; MacDonagh et al., 1990; Varma et al., 1986). This invasive procedure requires sacral dorsal rhizotomy in an attempt to prevent autonomic dysreflexia. This method has a high morbidity, especially when stimulation fails or devices have to be removed because of infection, leaving the patient with a flaccid bowel (from rhizotomy) which is extremely difficult to manage.

Direct electrical stimulation of posterior sacral nerves for treatment of faecal incontinence has also shown very promising preliminary results in some patients with weak, but structurally intact, anal sphincters (Malouf et al., 2000; Matzel et al., 1995). The posterior nerves are approached through the sacral foramen in a much less invasive technique than the anterior approach.

Management of faecal incontinence

Non-neurological causes of faecal incontinence should be addressed and treated: e.g. anal sphincter damage (obstetric or surgical), or infectious diarrhoea (e.g. *Clostridium difficile*). As previously mentioned, faecal incontinence might be secondary to constipation or impaction, and this should be managed first.

An anal plug has been marketed to help control faecal continence (see page 232). Impaired ano-rectal sensation is probably an important factor in enhancing tolerance of the plug and so it is particularly helpful for people with spinal lesions such as spina bifida.

Behavioural techniques, biofeedback and pelvic floor exercises may help where there is no structural sphincter damage, and may be useful as an adjunct to other treatments. Uncontrolled studies reported an efficacy of biofeedback for improving faecal continence in patients with myelomeningocele (Wald, 1983; Whitehead et al., 1981), spina bifida (Shepherd et al., 1983), and sacral agenesis (Benninga et al., 1994). In some incomplete SCI, preliminary results showed that biofeedback could be useful to recover continence (Monnerjahn C., Presentation at Annual Meeting of International Medical Society of Paraplegia, Copenhagen 1999). Biofeedback may thus play a role for those neurological patients in whom rectal sensation and the ability to contract voluntarily their
pelvic floor muscles are preserved, and who can co-operate in the process of training (Wald and Tunuguntla, 1984; Wiesel et al., 2000).

Loperamide has been shown to have mainly an antisecretory effect at the mucosal level, accompanied by motor effects when it reaches the myenteric opiate receptors (Awouters et al., 1993) (see Chapter 16). Patients who experience urge incontinence of faeces associated with loose stool, or who have a passive anal seepage of soft stool, may benefit from a low dose of loperamide, although there are no data on use in patients with neurological disorders. Lower doses (syrup formulation) than those used in diarrhoea may help selected patients by inhibiting colonic motility and rectal filling, especially when given on an intermittent basis when needed, such as before leaving the house. If used in this fashion, neurological patients need to be observed closely for the development of constipation and loperamide will usually only be used in conjunction with a bowel evacuation programme.

Summary

Management of the neurogenic bowel starts with the adaptation of the patient’s environment and his or her education (Table 17.2). A bowel programme will be designed to ensure satisfactory bowel care and prevent constipation and faecal incontinence. When constipation occurs, efforts are made to increase fluid and fibre intake and to use postprandial increases in colonic motility (gastro-colic response). Fibre is the most physiological approach in most patients. Rectal stimulants might be the first step, especially when colonic transit is normal but constipation predominantly terminal. An alternative to rectal stimulants is a small-volume enema. In case of failure, or when the rectal route is not practicable, an osmotic laxative should be tried. Stimulant laxatives should be deferred until patients do not respond to initial measures. When faecal incontinence occurs, behavioural techniques and pelvic floor exercises should be attempted. If constipation is not present, antidiarrheal drugs at appropriate dosage might be helpful. Surgical methods are considered when patients are unresponsive to medical therapy. Every method proposed should also consider the acceptability to the patient as well as cost aspects.

Educational strategies for the neurogenic patient

The success of any bowel management programme hinges on compliance. In a study addressing compliance with bowel medications, two-thirds of 114 participants were taking bowel medication when discharged from hospital. However, one month later 24% had reduced or stopped taking medication (Graham and Kunkle, 1996). A telephone survey questionnaire administered to 171 adults with a mean duration of SCI of 9 years and mean age of 40 years showed similar results on long-term outcomes of bowel management (Kirk et al., 1997). Bowel continence has been shown to be correlated to compliance in young adults with spina bifida (King et al., 1994). A variety of factors may contribute to nonadherence to a prescribed bowel programme, including impairment of cognitive function, misunderstanding of the prescription, inapplicability in the home setting, reluctance to cope with a disability, unwillingness to perform a complicated programme, acceptance of bowel symptoms, a lack of resources (home setting, carers, finance), and a lack of education about bowel function and management.

Education strategies should begin in the rehabilitation unit following full assessment. Education should centre on the patient, but carer(s) should also be included. The programme should describe the anatomy and the physiology of bowel function, the process of defaecation and bowel continence, the goals of a bowel programme, the safe and effective use of all methods prescribed for the bowel care, the bowel medications (type, purpose, dose, frequency, side effects and potential drug interactions), complications of the neurogenic bowel and ways to prevent and treat them, and methods to use in case of failure. Reinforcement
of the education should be provided when monitoring the effectiveness of the bowel programme. This approach has been successfully tested in SCI (Minton, 1983). See Appendix II for useful addresses and websites, such as www.spinal.co.uk/help/bowl.htm (sic).

**Conclusions**

Neurological disability is common and is often complicated by bowel problems such as constipation and faecal incontinence. Pathological involvement of the nervous system at any level may alter the motor function of the pelvic floor and abdominal muscles, bowel motility, the ascending afferent sensory function to the brain and the descending neuromodulation of visceral sensation from the brain. Although the neurophysiological pathways have been described, our understanding of enteric neurobiology is at a very early stage. This chapter outlines a targeted assessment and problem-solving approach to the management of neurogenic bowel dysfunction. The assessment of the neurogenic bowel is a methodical process, requiring multidisciplinary resources and a small number of investigations. It should take into account environmental and individual

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**Table 17.3: The management of the neurogenic bowel**

The management of the neurogenic bowel should offer the best method based on the assessment of the patient and his/her environment (see text for details).

**Environment management**

- Community and home (adapted, safe and pleasant, financial support)
- Carers (informed consent, ethical, skilled, empathetic)

**Individual management**

- Emotional (carers, relatives, professionals)
- Concomitant diseases, side-effects of drugs
- Bladder management (collaboration with urological team)

**Bowel Management**

- **Bowel programme**
  - Routine (scheduling, gastrocolic response)
  - Diet (healthy), fibre, fluid intake (accordance with the bladder)

- **Bowel care**

- **Assistive methods**
  - Massage, push-ups, Valsalva manoeuvre, deep breathing and forward-leaning position
  - Digital stimulation
  - Manual evacuation
  - Behavioural methods (biofeedback)

- **Pharmacological methods**
  - Laxatives (bulk-forming, faecal softeners, osmotic, stimulant)
  - Retrograde colonic irrigation (enema, pulsed irrigation enhanced evacuation)
  - Constipating drugs (Loperamide, codeine phosphate)

- **Surgical methods**
  - Antegrade continence enema procedure (Malone procedure)
  - Stoma (ileostomy, colostomy)
  - Sacral nerves neuromodulation (transcutaneous, direct anterior and direct posterior)
aspects. The assessment should identify which parts of bowel function are preserved. A bowel programme should be designed in accordance with patient’s disability, impairment, handicap, life goals and expectations.

The acceptability of a bowel program to the patient is crucial to its success. Compliance can be improved by education and reinforcement. Bowel management remains a challenge in rehabilitation. Many methods have been developed, but there is a lack of generalisation of knowledge and techniques between different patients groups. Some methods seem to be confined to SCI, others to spina bifida. There is a need for rigorous evaluation of the many methods that have entered the folklore without being subjected to trial.

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