ijlpr 2022; doi 10.22376/ijpbs/lpr.2022.12.6.SP24.L37-49

International Journal of Life science and Pharma Research

ISSN 2250-0480

Review Article

Overview On Neurogenic Bowel Dysfunction in Pediatrics

Ahmed Abdelsamie Fadl [1], Alruwaili, Nawaf Raji D [2], Nebras Omar Jaber Bedair [3], Ibrahim Hassan Almousaedi [4], Hadeel Salem Alwagdani [5], Shahad Bassam Sindi [6], Alhanoof Abdulhakeem Hazazi [7], Amal Abutaleb M Qaysi [8], Ahlam Shary J Hazazi [8], Mukhtar, Marah Wasel A [9], Howra Wasel Alhashim [10], Attar, Abdulhakeem Abdullah M [11], Fatimah Abdulkareem Bin Amer [12] and Alomair Abdulrahman Mohammed[13]

[1]Pediatric senior registrar at Doctor Samir Abbas Hospital,Department of pediatrics, Alazhar University hospitals, Cairo. SCFHS number: 12JM0036858

[2]Al-Qurayyat General Hospital, Al-Qurayyat

[3]King salman bin abdulaziz medical city Madinah

[4]Ghmrah PHCC, Rijal Almaa sector, Aseer, KSA

[5]Alaziziyah Children Hospital, Jeddah

[6]Pediatric resident, Khulais General Hospital ، Makkah

[7]Almaarefa university

[8]Medical intern, University of Tabuk

[9]Medical Intern, Umm alqura university

[10]Medical intern, Arabian gulf university

[11]Taif Children Hospital

[12]KING FAHAD HOSPITAL IN HOFOUF

[13]King faisal university

Abstract: Neurogenic bowel dysfunction (NBD) is fecal incontinence or constipation is prevalent in kids with both congenital and acquired neurological disorders. NBD results from loss of normal sensory or motor control and may include both the upper and the lower gastrointestinal (GI) tract Constipation and fecal incontinence are frequent symptoms of neurogenic bowel dysfunction (NBD), which makes it a challenging condition to treat. Also, they have a major impact on quality of life and dignity. Bowel dysfunction is less studied compared to neurogenic bladder, generally prevelant in spina bifida, but practically as prevalent in other neurological conditions. Generally, the objective of neurogenic bowel management is to achieve complete emptying of the rectum on a systematic basis. This may be accomplished through with a multidimensional approach containing of conservative, medical and even surgical approaches. A modified Delphi procedure was used to construct a statement document. This paper discusses the various causes of paediatric NBD. To enhance clinical management, numerous therapeutic techniques are given. Due to both the improved survival rate and better diagnosis, there are more children and teenagers with NBD. The number of children and adolescents with NBD is growing as a result of better diagnosis and a greater survival rat NBD can cause either faecal incontinence or constipation, or both, with a fair amount of predictability. However, each patient will experience NBD in a different way depending on a variety of underlying conditions and coexisting conditions. Due to the status of the affected child and caregivers, management of NBD should be individualized using a combined multidisciplinary therapy.

Keywords: Neurogenic, Central Nervous System, Pediatrics**,** Neurological Disorders and Sphincter

***Corresponding Author**

Ahmed Abdelsamie Fadl , Pediatric senior registrar at Doctor Samir Abbas Hospital,Department of pediatrics, Alazhar University hospitals, Cairo. SCFHS number: 12JM0036858

Received On 01 September 2022

Revised On 04 October 2022

Accepted On 11 October 2022

Published On 23 October 2022

Citation Ahmed Abdelsamie Fadl , Alruwaili, Nawaf Raji D , Nebras Omar Jaber Bedair , Ibrahim Hassan Almousaedi , Hadeel Salem Alwagdani , Shahad Bassam Sindi , Alhanoof Abdulhakeem Hazazi , Amal Abutaleb M Qaysi , Ahlam Shary J Hazazi , Mukhtar, Marah Wasel A , Howra Wasel Alhashim , Attar, Abdulhakeem Abdullah M , Fatimah Abdulkareem Bin Amer and Alomair Abdulrahman Mohammed , Overview On Neurogenic Bowel Dysfunction in Pediatrics.(2022).Int. J. Life Sci. Pharma Res.12(6), L37-

1. INTRODUCTION

Neurogenic bowel dysfunction (NBD) is fecal incontinence or constipation resulting from central nervous system (CNS) disease or damage [1, 2]. It is a widespread problem for people with neurological disorders; variations in bowel motility and sphincter control can present a most important problem for people with spinal cord injury (SCI) and multiple sclerosis (MS) amyotrophic lateral sclerosis (ALS), spina bifida, [3] myelomeningocele (MMC), Parkinson disease (PD), stroke, and diabetes mellitus which extremely influences quality of life [3, 4]. Constipation and fecal incontinence are frequent symptoms of neurogenic bowel dysfunction (NBD), which makes it a challenging condition to treat. Chronic constipation and fecal incontinence often coexist, sometimes with "overflow" diarrhea (where solid stool obstructed higher up the rectum or colon only permits watery stool past it, which is then very hard for even a neurologically intact anal sphincter to retain). This leading to annoying situation for both patients and caregivers, particularly in a neurogenic scenario, frequently well-defined as neurogenic or neuropathic bowel dysfunction (NBD) [5]. This regularly happens if muscles in the rectum and anus are not working to store and hold back a bowel movement due to muscle injury or nervous system damage, as well as a loss of rectal sensation [6]. NBD results from loss of normal sensory or motor control and may include both the upper and the lower gastrointestinal (GI) tract [6]. Neurogenic bowel dysfunction (NBD) can restrict knowingly with a person's education, work, and social life and presents a main challenge to quality of life, independence, and community reintegration after SCI. Loss of bowel control is a source of anxiety and distress [7, 8]. Bowel dysfunction can affect patients' psychological, physical, and social well-being because of constipation, FI, the extended time spent on defecation, and the social restrictions that bowel dysfunction imposes on the patient [9, 10]. A careful primary valuation delivers an idea of symptom severity and is critical for successful rehabilitation. The backbone of treatment remnants a traditional method of management fecal incontinence or enhancing the mechanics of defecation using laxatives and irrigation approaches. When successful, this approach progresses both evacuation and incontinence symptoms, with related developments in quality of life and independence [11, 12]. Even though bowel dysfunction is a communal occurrence, to date there have been moderately few studies addressing bowel management.

1.1 Prevalence

Bowel dysfunction is less studied, but practically as prevalent in other neurological conditions. Neurogenic bowel dysfunction (NBD) affects ~80% of spinal cord injury (SCI) patients to some degree up to 95% report constipation, fecal incontinence is experienced at least once per year by 75% and daily by 5%, with 33% experiencing regular abdominal pain associated with the level of injury [13-15]. It is also prevalent among patients with other neurological conditions such as multiple sclerosis (MS) and spina bifida (SB) [16]. Up to 30% of MS patients can experience fecal incontinence (FI) [17]. About one-third of MS patients suffer from constipation and one-quarter are incontinent at least once per week [18]. In patients with PD, constipation, in specific difficulty with defecation, occurs in 37% [19]. One quarter of stroke survivors experience constipation and 15% suffer with fecal incontinence [20]. Bowel dysfunction is recorded in 0.7–29.6% of children and adolescents, and it may be caused by functional abnormalities, congenital anatomical deformities, reasons involving the digestive tract and the nervous system, or a combination of these factors [21].

1.2 Causes

The term 'neurogenic bowel' includes the manifestations of bowel dysfunction resulting from sensory and/or motor disturbances due to central neurological disease or damage [22]. The complicated regulation of the gastrointestinal system depends on the coordinated relationship of neural impulses and muscle contractions. Constipation and/or fecal incontinence progress when there is a difficulty with the regular bowel functioning, which might be for numerous causes [23]. There are two chief types of nervous system within the lower gastrointestinal (GI) tract: the intrinsic enteric nervous system and the extrinsic nervous system. The intrinsic enteric nervous system controls gut motility directly, while the extrinsic nerve pathways effect gut contractility indirectly by adapting this intrinsic enteric response [23]. In almost all cases of neurogenic bowel dysfunction, it is the extrinsic nervous supply that is affected while the intrinsic enteric nervous supply remains intact. Patients with spinal cord lesions, either congenital or acquired, have an anatomically intact rectal ampulla, anal canal, and sphincter but experience constipation and/or incontinence due to damage of their enteric nervous system, reduced sensation, and limited mobility [24]. Pediatric NBD is typically brought on by congenital issues like spina bifida (SB). Acquired forms brought on by injury, infection, etc. resemble adult clinical images more [25]. The following list of etiologies is presented roughly in order of paediatric relevance (based on the frequency of each cause in childhood and its propensity to result in NBD in children).

1.3 Myelodysplasia

Also referred to as SB, is a condition that causes the embryonic neural tube to malform and the vertebral column to not completely close. This phrase refers to a group of neural tube abnormalities (NTDs), which include myelomeningocele, lipo-myelomeningocele, meningocele, and spina bifida occulta. One of the most frequent congenital deformities affecting the spine and brain, myelomeningocele (MMC), may affect any level of the spinal column (lumbo-sacral 47%, lumbar 26%, sacral 20%, thoracic 5%, and cervical spine 2%) [26]. The neurological abnormalities caused by SB vary and depend on the protruding neuronal components inside the sac. In myelomeningocele, the neural roots or spinal cord segments herniate through the imperfectly closed vertebrae and are thus vulnerable to harm before birth (or afterward, up until the sac is medically repaired). However, there is little connection between the neurological abnormalities caused and the bony vertebral level. Additionally, during childhood, from birth to puberty, a neurological lesion may become dynamic because to variations in the growth rates of the vertebral bodies and the spinal cord. In 85% of children with MMC, there is associated hydrocephalus (with an Arnold-Chiari, or Chiari type-II, malformation), which frequently necessitates ventriculo-peritoneal shunting of extra cerebrospinal fluid to lessen its pressure on the brain. The prevalence of MMC and other neural tube abnormalities has been greatly decreased thanks to widespread obligatory fortification of dietary staples with folic acid and voluntary folic acid intake prior to conception and during the first trimester of pregnancy [27].

1.4 Sacral Agenesis (SA)

SA also known as Caudal Regression Syndrome, involves the total or partial absence of the lowest five vertebrae. When a child fails to complete toilet training on schedule, urinary and/or faecal incontinence are frequently described and identified. A thorough physical examination should also involve palpating the spine to the tip of the coccyx (to rule out a bone deformity), neurological testing of the lower limbs, and gait analysis. Careful investigation may reveal flattened buttocks [28,29].

1.5 Anorectal Malformation

Anorectal malformation (ARM), formerly known by the more specific term imperforate anus, is thought to affect between 1 in 2000 and 1 in 5000 live births .In 38% of cases, spinal cord pathology is present, either alone or in conjunction with other congenital malformations [30]. The Vertebral, Anorectal Malformation, Cardiac, Tracheo-Esophageal Fistula, Renal, and Limb Anomalies (VATER or VACTERL association) is a set of frequently coexisting abnormalities .According on whether the blind-ending rectum finishes above, at, or below the levator ani muscle, ARM has historically been categorised as high, moderate, or low .Before, pulling the rectum through to the anal margin during an imperforate anus repair for high lesions using a perineal technique frequently led to an injured pudendal nerve .This problem has been removed thanks to the development of the posterior sagittal anorectoplasty (PSARP) surgical technique [31].

1.6 Cerebral Palsy

A congenital neurological disorder called cerebral palsy (CP) is described as a non-progressive damage to the brain or a deformity of the brain that occurs during prenatal or postnatal development[32]. The most frequent neurological disorder seen in children is cerebral palsy (CP), which has an incidence of 1.5 per 1000 live births CP refers to a range of conditions that affect mobility and posture development to varying degrees Constipation affects up to 90% of children with CP, and 47% of them experience some degree of faecal incontinence [33]. Instead of being caused by a fundamental intrinsic neuropathy of these structures, these effects are caused by aberrant higher-level regulation of the intestine and/or sphincter. The recommended treatment options for NBD depend on the intellectual disability of the patient, which affects about half of CP patients [34,35]. NBD has outher cause as muscular dystrophies, mitochondrial disorders, acquired brain Injury, acquired pelvic Injury, acquired spinal cord injury, autism, down syndrome, multiple sclerosis, transverse myelitis and meningitis retention syndrom Children and teenagers complain from NBD, frequently accompanied by neurogenic bladder dysfunction. NBD has been more thoroughly assessed in various neurological illnesses, and early management is typically implemented. In contrast, it is frequently overlooked, undertreated, or untreated in other conditions, both uncommon and prevalent. Due to the prevalence of common congenital disorders like cerebral palsy and Down syndrome, as well as all types of uncommon acquired neurological damage like post-traumatic stress disorder, Guillain-Barré syndrome, transverse myelitis, etc., all children with special needs must be evaluated for NBD. In order to provide customised diagnostic routes and management, future research must take these clinical circumstances into account[36].

2. PATHOPHYSIOLOGY

The intrinsic enteric neural system, which is found inside the gut wall, and the extrinsic nervous system, which consists of sympathetic and parasympathetic innervation, are the two primary types of neurological systems in the lower gastrointestinal (GI) tract [37]. The extrinsic neural pathways affect gut contractility indirectly by altering this intrinsic enteric response, whereas the intrinsic enteric nervous system directly regulates gut motility [38]. The extrinsic nerve supply, rather than the intrinsic enteric nervous supply, is nearly always the part of neurogenic bowel dysfunction that is compromised. The causes of a neurogenic bowel dysfunction (NBD) in children and adolescents are different from adult forms. In most cases, pediatric NBD is caused by congenital problems such as spina bifida (SB). Acquired forms caused by trauma, infection, etc., are more like adult clinical pictures [39]. Despite having anatomically normal rectal ampulla, anal canal, and sphincter, patients with spinal cord lesions-whether congenital or acquired-experience incontinence or constipation as a result of enteric nervous system impairment, decreased feeling, and restricted mobility. These children may also have problems with anal squeeze pressure, anorectal sensitivity, and anal resting pressure. Rectal compliance may also be affected [40,41] due to the rectum's hyperreactivity, which affects colorectal motility, transit time, and bowel emptying and frequently results in constipation, faecal incontinence, or a combination of the two. Children with spina bifida experience bowel dysfunction because, despite having a typically functioning spinal rectoanal inhibitory reflex (RAIR), they lack the urge to urinate. Bowel soiling happens when the internal sphincter relaxes. Increased colonic transit time, a lack of sphincter relaxation, and rectal distension cause constipation [41,42]. A general decline in activity and, depending on the severity of the spinal injury, abdominal muscular weakness that affects the capacity to push out faeces are additional factors contributing to bowel dysfunction in children with spinal cord disorders [43]. The majority of kids get constipation, which manifests as frequent, tiny, and firm faece. The pathophysiologic mechanisms of constipation are obstructed defecation, weak abdominal muscles, diminished rectal feeling, and delayed colonic transit time. Both incomplete and complete lesions can cause blocked defecation or fecal incontinence. fecal incontinence happens due to areflexic or atonic anal sphincter, uninhibited rectal contractions, poor rectal sensibility, and lack of anal sphincter tone and contraction (conus and cauda equina lesions)[44]. The pathophysiology of bowel dysfunction in patients with PD is relatively different from that of SCI or MS. Dystonia of the striated muscles of the pelvic floor and external anal sphincter describes the defecation dysfunction; this an etiological factor is reinforced by the observation that pelvic floor dysfunction is improved with L-Dopa [45,46]. As well as the pelvic dysfunction, colonic transit time is usually extended in patients with idiopathic PD [45].

2.1 Impact of Anatomical Location of Nerve Damage

According to the site of the brain damage and the site of damage along the spinal cord and severity of the damage, colorectal function as well as the kind and amount of future symptoms are defined. immediately after the injury the patient complain of a spinal shock that can last up to 6 weeks it makes it difficult to determine the level of injury along the spinal cord. Depending on the severity of the illness or damage to the conus medullaris, neurogenic bowel symptoms can be

classified into two patterns :1. Supraconal disorder, often known as "spastic bowel," "hyperreflexic bowel," or "upper motor neuron bowel syndrome" This pattern involves a loss of supraspinal inhibitory input, which results in hypertonia of the colorectum, and is observed in patients with sickness or injury above the conus medullaris. Reduced colonic compliance, excessive segmental peristalsis, and insufficient propulsive peristalsis are caused by an increase in the tonus of the colonic wall, pelvic floor, and anus [47-49]. The transit slows down throughout the colon as the peristaltic and haustral motions become less efficient [50,51]. Stool retention brought on by the external anal sphincter's (EAS) spastic constricted state makes the problem much worse. Constipation is the most common gut symptom as a result of the interaction of various physiological reactions to supraconal damage. The reflex that initiates a bowel movement still functions, but the child may not feel it coming, resulting in a sudden unplanned passage of stool whenever the rectum is full when the anal sphincter is unable to be consciously relaxed. High resting anal tone, the presence of the anal/anocutaneous reflex (which causes the anus to contract reflexively in response to perianal skin stroking), and the presence of the bulbospongiosus/bulbocavernosus reflex are all symptoms of these illnesses (reflex contraction of anus in response to squeezing the glans penis or clitoris). 2. Lower motor neuron type infraconal disease or areflexic bowel. After a lower spinal cord injury, the bowel may become flaccid. Infraconal lesions result from injury to parasympathetic cell bodies in the conus medullaris or their axons in the cauda equina, which disrupts autonomic motor neurons. This is characterised by decreased rectoanal inhibitory reflex (RAIR) amplitude and loss of colorectal tone, which causes a cyclical pattern of insensate rectal filling and progressive rectal distension that ultimately results in faecal incontinence. Due to weak anal sphincters and lax pelvic floor muscles, which permit excessive descent of pelvic contents, lowering the anorectal angle and widening the rectal lumen, the incontinence is further made worse by a decrease in resting and squeeze anal pressures [51]. When the bowels are flaccid, the colon moves less, there is fewer peristalsis, and the anal sphincter is more relaxed than usual. This may cause frequent stool leakage and constipation. These patients typically have no or little anal tone at rest, as well as no anal/anocutaneous or bulbospongiosus/bulbocavernosus responses.

2.2 Symptoms

The foremost symptoms of NBD are fecal incontinence and constipation. Fecal incontinence is the accidental movement of solid, liquid, or mucous stools. This often happens when the rectal and anal muscles fail to preserve and retain bowel movements due to muscle damage, nervous system damage, and loss of rectal sensation [52]. Constipation is well-defined as a reduction in the number of bowel movements, but deficiency of daily bowel movements is not constantly constipation, as some people only have three bowel movements a week. Symptoms of constipation include difficulty bowel movements, rare bowel movements, and hard bowel movements [53]. Cameron et al. lately established in a mixed cohort of patients with SCI or MS, patients with the most bothersome bowel symptoms also had higher acuity urinary incontinence and lower urinary tract symptoms [54].

2.3 Diagnosis and Assessment

A GI history should be taken from the patient and any careers they may have. Details of bowel habit before injury or neurological disease onset should be investigated [55]. Existing symptoms should be cautiously assessed, containing bowel movement frequency, stool consistency (the Bristol Stool Form scale can be helpful), occurrences of fecal or flatus incontinence or urgency, maneuvers wanted for bowel management, time spent using the restroom, episodes of fecal impaction, use of laxatives and anti-diarrheal, and the requirement for pads or plugs [56]. Furthermore, it is important to consider any associated illness, UTIs, hemorrhoids, stomach discomfort, rectal hemorrhage and prolapse, anal fissures, and autonomic dysreflexia are examples of NBD symptoms [57]. There are standard instruments such as the Cleveland constipation score and St Mark's incontinence score may possibly be used dependent on predominant symptoms and recently a condition specific score has been developed for neurologic patients [58,59]. Scoring systems may be supportive in measuring symptoms. Digital rectal examination is an important component of assessment and should examine rectal fullness, resting anal tone, and capacity for a voluntary contraction, this will also give a rough evaluation of anal sensitivity [56]. Perineal sensitivity may be examined by pinprick. Examination should as well involve looking for complications of chronic constipation, specifically anal fissures, complicated hemorrhoids, rectal bleeding, and prolapse [55]. Evidently, patients with alarm symptoms should have essential colonic imaging performed. Alarm symptoms in this patient group are more complicated to identify, but any deterioration of determined bowel dysfunction, weight, or blood loss necessitates investigation [60].

3. MANAGEMENT AND TREATMENT

Generally, the objective of neurogenic bowel management is to achieve complete emptying of the rectum on a systematic basis, thus reducing the risk of fecal impaction, urgency, and incontinence [61]. This may be accomplished through with a multidimensional approach containing of conservative, medical and even surgical approaches. Patient education and training are essential to success regardless of the strategies employed [62].

3.1 Conservative Treatments

3.1.1 Dietary Patterns

Fiber Changing diet to contain higher fiber content is generally suggested as a first step in a bowel management program [63]. Generally, high fiber diet is recommended to prevent constipation [64]. One small case series revealed that rising dietary fiber improved colonic transit time in a cohort of SCI patients [65]. Even though the prevalent recommendation, there is essentially very restricted data on dietary fiber particularly in the managing of NBD. Fluid intake should also be improved while taking bladder constraints into account [66]. The fluid/fiber ratio is also important: insufficient fluid intake with the fiber can make constipation worse. Sufficient fluid intake enhances the influence of osmotic laxatives and fiber and is also essential for bowel health in general. Fiber absorbs large amounts of water in the intestine, so a high-fiber diet can cause constipation if many fluids are not also taken [67]. Be aware that insoluble fiber causes flatulence and bloating. A well-balanced diet should be encouraging, which contains fruits, vegetables, and enough of water, and constipating foods such as cheese and white rice should be limited [63]. Use caution while consuming substances that cause stools to become loose, such

as coffee, alcohol, and foods that contain the sugar sorbitol . Establishing a regular eating schedule is the most crucial stage in achieving good bowel motility, regardless of the diet's composition [68]. No matter what the diet content, the most significant step in achieving optimum bowel motility is to create a standard eating pattern [69]. The use of opiates, non-steroidal anti-inflammatory medicines (NSAIDs), and antibiotics, as well as bladder anticholinergics and non-steroidal anti-inflammatory drugs (NSAIDs), may all lead to bowel dysfunction [68].

3.1.2 Physical Activity

Like diet, there is no common opinion about the effects of increased physical activity on managing constipation. Even though the lack of a strong evidence bases for these conservative interventions, they have been found to be beneficial in patients with NBD. Frequent activity can assist reduce constipation by promoting the bowel's peristaltic motility [63].

3.2 Abdominal Massage

In an uncontrolled clinical study, abdominal massage was found to have optimistic effects on select aspects of NBD (abdominal distention, fecal incontinence, colonic transit time) in patients with SCI [70].

3.3 Valsalva Manoeuvre

To guarantee efficient propulsion, the Valsalva manoeuvre is performed by trying to exhale against a closed airway (closed glottis or pinched nose).

3.4 Digital Rectal Stimulation

The goal of digital rectal stimulation is to trigger the recto-colic reflex, which will cause a bowel movement, by inserting a gloved, lubricated finger into the anus and moving it in a circular motion for 20 to 30 seconds. The procedure can be performed once more five minutes later, but SCI patients should exercise caution because it could cause autonomic dysreflexia [71]. Digital evacuation of faeces entails manually eliminating formed stools present in the rectum utilising a hooking motion rather than contraction; doing a Valsalva manoeuvre concurrently may increase effectiveness. Enemas and suppositories may also help to stimulate reflex contraction; however, they should only be used if digital rectal examination reveals that faeces is present in the rectum and should be kept for at least 10 minutes. Glycerin (a lubricant), bisacodyl (a stimulant), and lecicarbon are choices (carbon dioxide releasing).

3.5 Scheduled Defecation

Establishing a routine for bowel care is also extremely important. Patients should try to defecate at a scheduled time, either daily or on alternate days [55]. Generally, to advantage from the diurnal "body clock," scheduled defecation should be tried once per day at nearly the same time every day (or, if not possible, on alternate days) [63]. Opportunities of success may be increased by scheduling defecation to occur when bowel contractions are strongest: on waking and after a meal/warm drink [55]. Also, position during toileting can be used to increase bowel efficiency. Gravity can be best manipulated in a seated position, on a toilet or commode, if this is practical for the patient [69].

3.6 Transanal Irrigation Methods

IT WORKS by injecting water into the colon and rectum through the anus to trigger a reflex colorectal voiding, transanal irrigation (TAI) aids in the evacuation of faeces from the bowel. A single-use device, such as a catheter or a single-use cone, is used to introduce the water. Cones are favoured if the patient can keep the device in place by hand or sphincter tone while injecting the fluid. The choice of cone or catheter relies on the patient's preferences, hand function, and anal sphincter integrity. The contents of the rectum and some of the more proximal colon are discharged once the device is removed. Regular use of TAI assists in the restoration of regulated bowel function and gives the patient control over when and where ejection occurs. Effective evacuation of the colon and rectum in the case of faecal incontinence delays the arrival of fresh faeces by about two days, preventing leaking between irrigations [72]. Constipation sufferers who regularly clear their rectosigmoid region may be able to avoid blockages by facilitating movement across the entire colon. When compared to conservative bowel care, TAI had better quality-adjusted life years and around 60% of patients continued receiving treatment at long-term follow-up, which resulted in lower incidence of stoma surgery, UTIs, and episodes of faecal incontinence [73]. Compared to continuing with routine bowel treatment, this resulted in cost savings of $21,768 per patient[73].

3.7 Electrical Stimulation

Another technique that has been investigated in some individuals who have undergone unsuccessful conservative therapy is electrode implantation. The electrodes are implanted using this method on the sacral roots. Both the afferent fibres going to the brain and the sacral efferents are hypothesised to be affected by this sacral nerve stimulation.[68]. A sacral anterior root stimulator must be implanted via a laparotomy, which is a more invasive procedure. To avoid autonomic dysreflexia, this may be followed by a posterior rhizotomy, which is then followed by the implantation of electrodes on the efferent sacral roots[74]. Although these implants are more frequently done for bladder control, there is evidence that they have good effects on bowel function as well [74]. They are hardly ever employed, nevertheless, because of the methodology's expensiveness, technical complexity, and intrusiveness. Although they were first identified many years ago, alternative kinds of neuromodulation have only been examined in individuals with NBD with little success and widespread uptake [75].

3.8 Surgical Antegrade Colonic Irrigation

Children with NBD, particularly those who have spina bifida, have been treated with antegrade irrigation via an appendicostomy, with over 80% of patients experiencing long-term success [76]. Unfortunately, adult findings have been less encouraging, with tract stenosis developing as the predominant issue[77]. This method's time-consuming washing out of the entire colon is another drawback [68]. An alternative method of irrigation is through a percutaneous endoscopic colostomy. In this method, the distal bowel is washed out using a tube that has been inserted into the sigmoid colon. Although most patients benefit from the method, there can be significant

consequences that make it a less practical strategy over time [68,78].

3.9 Stoma Formation

Since it is invasive and not just reversible, surgical stoma creation is usually viewed as a last resort. However, it can be quite effective for patients who have strong upper-limb function and when faecal incontinence predominates [68]. Reduced bowel management time and higher quality of life are linked to stoma development [79]. Unfortunately, problems may occur in as many as 37.5% of patients (including rectal mucus discharge, diversion colitis, and post-surgical adhesions [62, 80, 81]. If a loop ileostomy is not performed, laxatives or stoma irrigation may still be required [68]. For patients who need faecal diversion because of complex perianal wounds, a left-sided colostomy may be the most appropriate placement. For those with good colonic motility, this strategy should be avoided because it is linked to poor colonic emptying. Although right-sided colostomies are less likely to result in these issues, they do produce more liquid stools, more stoma care needs, and a higher chance of leakage [82].

3.10 Pharmacologic Therapy

Oral or rectal pharmacologic therapies may be utilized to strengthen conservative management [83].

3.11 Rectal Medications

Rectal medications (suppositories, enemas) chemically stimulate the anal sphincter reflex to evacuate stool, and thus, the presence of an intact reflex is typically needed. They treat the dual problem of constipation and fecal incontinence [84]. Numerous cross-sectional studies determine those rectal medications are used to treat more severe cases of NBD as those using rectal medications were accompanying with cervical injuries, poorer quality of life [85, 86].

3.12 Suppositories

The suppository acts as a contact irritant to improve gastric motility, increase the fecal water content, and reduce transit-time within the large intestine [87]. Bisacodyl (Dulcolax) and glycerin suppositories are commonly used in traditional management of NBD that stimulate the bowel reflex [83]. Sodium bicarbonate (Lecicarbon) is a newer effervescent suppository, releasing bubbles of carbon dioxide to stimulate reflex rectal activity, which has a faster beginning of action than fat-based bisacodyl suppositories (15–20 min compared to 30–40 min) but similar efficacy [88]. suppositories can accomplish rectal stimulation but can be complicated to keep in place in patients with lax anal tone [89].

3.13 Enemas

An enema is a method of delivering liquid into the rectum to remove stool. Although enemas are often used for acute constipation in people with neurotypical bowels, regular enemas can be an important part of a bowel management program for people with neurodevelopmental bowel disorder (NBD). If a suppository is not working, they are usually used as a backup [63].

3.14 Oral Medications

Oral laxatives are the next step up the ladder in the management of NBD. High-quality information be present in the form of various RCTs validating the favorable effect of laxatives in individuals with NBD [63]. Oral laxatives are the first-line treatment for constipation; they are appropriate to both areflexic and reflexic bowel management. Polyethylene glycol (PEG)/macrogol has been found to be superior to lactulose in one RCT including pediatric NBD. Other generally used oral laxatives involve bisacodyl and senna (colonic stimulants), docusate (stool softener), and ispaghula husk (Fybogel, bulk-forming) [90]. Oral medications may direct constipation but may not certainly treat fecal incontinence. This may be due to the less expected timing of results following oral medications.

3.15 Prokinetic Drugs

When oral laxatives are not successful, prokinetic drugs may be an alternate. Evidence for prokinetic drug studies was found for prucalopride, metoclopramide and neostigmine in SCI (1 RCT for prucalopride, 2 RCTs and one observational study for neostigmine, and two observational studies for metoclopramide) [84]. Neostigmine is a reversal cholinesterase inhibitor that has also been studied in NBD. Considerable advancement in total bowel evacuation time with intramuscular neostigmine-glycopyrrolate as compared to placebo was noted in a trial in patients with SCI [91,92].

3.16 Surgical Management

3.13.1 Sacral Nerve Modulation

With the invasive implantation of electrodes along the sacral nerve roots, sacral nerve modulation (SNM) is a step up from transcutaneous electrostimulation techniques. It produces more targeted effects (i.e., it is possible to focus on either the rectum or the anal sphincter or both), but this is offset by higher risks of nerve damage and introducing infection. Initially designed to treat lower urinary tract symptoms, particularly in neuropathic situations, SNM is now also utilised to treat bowel dysfunction. SNM stimulates the somatic and autonomic nervous systems, while its precise mechanism of action is unclear [93,94] and only a few studies have suggested that it may also have effects on the central nervous system [95]. It has been hypothesised that its effects on cases of constipation are caused by an increase in the frequency and amplitude of antegrade pressure sequences, although it is yet unknown whether they are mediated by a central or peripheral mode of action. SNM is now solely recommended for faecal incontinence in adults because randomised controlled trials have not demonstrated a benefit for chronic constipation in this population [96]. Although it is questionable whether this is sufficient to justify the risks and high costs, SNM has demonstrated some sustained benefit in children and young adults with refractory functional constipation [97]. SNM is not, however, FDA-approved in the USA for treating gastrointestinal dysfunction in young patients (under the age of 18). (nor under the age of 16 for bladder dysfunction). Additionally, it might not be theoretically possible for the more prevalent causes of NBD, such as spina bifida and spinal cord injury, which include anatomical abnormalities of the spinal cord. A few studies have studied the role of SNM in neurogenic patients, and improvements in SCI have been noted [98].

3.13.2 Bowel Surgery

Surgical management of NBD is considered as a beneficial alternative in selected cases [99]. The purpose of surgery for NBD, just as with its conservative and pharmacological management, is to evacuate the colon at a time and place of the patient choosing, it should also minimize the average time the patient needs to spend in the bathroom every week [63]. Surgical methods for bowel management are typically employed only after breakdown of the maximum scale of conventional conservative and pharmacological medical treatments, which now involves trans anal irrigation (TAI) [100]. Various studies show that surgical treatment of NBD can be effective in supporting an enhanced QoL if properly indicated and with patients carefully selected [63]. However, a recent study found that roughly 40% of paediatric and adult patients with NBD attributable to myelomeningocele require surgery (due to failure of medical treatment) in order to attain faecal control [101]. Most reports on surgical treatment of NBD deal with adult patients and very little has been published on children and adolescents on this topic. The majority of the advantages and disadvantages of surgery for these individuals, apply to all age groups. However, it's crucial to keep in mind that young patients are still developing (both physically and emotionally) and will likely need to use the surgically set way of colon emptying for the rest of their lives. In order to create a suitable, individualised solution that enables the paediatric patient to integrate as best possible with their age-appropriate peers, the proposed surgical options must also respect the paediatric patient's developmental age and any comorbidities, as well as the family dynamics and environment [102]. The surgical treatment for NBD in kids mainly entails establishing artificial "upstream" access for antegrade administration of colonic irrigation enemas, either by the Malone's antegrade continence enema (ACE) surgery or through tube cecostomy. This may be helpful for patients who have NBD-related stool impaction [103] or who, because of coexisting conditions, lack the coordination, physical dexterity, or drive to self-administer retrograde washouts by TAI [104]. A catheter that is intermittently inserted or an indwelling tube can be used by many teenagers to autonomously deliver their antegrade enemas. Colostomy (faecal diversion) is the last surgical option for children, however Malone's ACE operation is by far the most popular one [105]. Sadly, several additional reconstructive methods that are available to people with NBD in adulthood, including artificial anal sphincter implantation [106], are typically inappropriate for a developing youngster.

3.13.3 Malone Antegrade Continence Enema Procedure

Malone's ACE approach has been demonstrated to be a secure surgical technique, with low mortality but a few minor side effects [107]. 80 %of adult patients who successfully used the Malone antegrade continence enema (MACE) through a neo-appendicostomy reported an improvement in QoL [108]. A considerable increase in faecal continence and QoL scores was seen after the MACE was effectively applied to children with spina bifida [109,110]. The current standard in situ appendicostomy for the MACE produces a continent catheterizable appendiceal channel to the cecum by creating a valve mechanism at the cecal end (to reduce leakage of feces onto the skin) and bringing the decapitated end of the appendix up to a convenient site on the abdominal wall such as the umbilicus or hidden under a cosmetic skin-flap elsewhere that also serves to reduce the risk of stomal stenosis. Beside this technique, other open surgical modifications have been performed in the pediatric age group such as the cecal extension (when the appendix is not long enough), the Yang-Monti ileo-cecostomy (using a short section of detubularized retubularized ileum to create an alternative channel when a suitable appendix is not available) and cecal or colon flap channels (again if an adequate appendix is not available) [110]. MACE channels are often constructed at the same time as urinary reconstructive surgery such as a Mitrofanoff procedure for associated neurogenic bladder. If the appendix is not long enough, or cannot be extended sufficiently, to create both channels, this may give rise to surgical dilemmas regarding the optimum use of the appendix, and the need to use such modifications. However, the rate of surgical revisions required after some of these modifications appears to be higher than for a standard MACE [111]. In the subsequent laparoscopic adaptation, there is usually no attempt at the technically difficult creation of a valve mechanism, yet the rates of fecal leakage via such stomas are still surprisingly low [112,113]. If investigation such as a colonic transit study suggests mega-rectum and/or distal colonic delay with feces impacting in the recto-sigmoid, then a “distal ACE” (e.g., in the transverse or descending colon) can produce a more effective evacuation of feces and reduce the risk of retention of the irrigant compared to the conventional cecal ACE [114].

3.13.4 Tube Cecostomy

Another modification of the MACE is the utilization of a Chait® (Cook Medical LLC, IN, USA) cecostomy tube, or a “button” device, placed as either a percutaneous endoscopic cecostomy (PEC), under fluoroscopic guidance, or via laparoscopy. It has been proven significantly to improve fecal continence and QoL in patients with NBD [115]. The drawback of such tubes is that they must be replaced on a regular basis and sooner if they clog, detach, or break .As with a traditional ACE, the Chait® tube or button device can be inserted more distally in the colon as a percutaneous endoscopic colostomy in cases of delayed colonic transit (for example, at the descending/sigmoid junction) [116]. In children with spina bifida (SB), MACE and tube cecostomy outcomes are comparable [102]. But regardless of how they are carried out, both operations pose a significant danger of compromising the vital ventriculo-peritoneal shunt in kids with hydrocephalus associated with spina bifida [117].

3.13.5 Bowel Diversion

Colostomy involves creating a stoma by bringing a portion of the large intestine to the surface of the abdomen .The patient wears an external bag over the stoma to collect faeces .The patient's unwillingness to accept the procedure from a psychological standpoint may be the biggest obstacle to performing a bowel stoma in any age group .This is especially true for the paediatric population, when kids and parents may worry about excrement, flatus, or smell leaks, its effects on physical integrity and self-image, and the potential for peer bullying. In contrast, ostomy (either colostomy or ileostomy) as a bowel diversion results in comparable or even better QoL outcomes in a subset of patients when compared to conventional bowel management techniques. The upstream colon can be retrogradely irrigated in a manner similar to TAI for people who wish their stoma to act at a convenient moment (see Section Transanal Irrigation.). However, there have been a sizable number of postoperative problems documented. The main benefit of diversion is a shorter time required to empty the bowels. Patients who have ostomy surgery, frequently as a "last resort," are typically very satisfied with the improvement that results, and a sizable percentage of

patients later report wishing that they had been counselled about this option sooner [118] rather than saving it for alleged failures of care [119,120]. Additionally, colostomy in adults with faecal incontinence was associated with a decrease in hospitalizations [121]. Early colostomy insertion following spinal cord damage has also been demonstrated to promote independence and raise bowel management acceptance [122,123]. Occasionally, an ostomy is necessary to prevent the perineum from being soiled while the chronic decubitus pressure sores heal.

3.13.6 Bowel Resection

For some cases of functional constipation and/or faecal incontinence that have not responded to conservative treatment, bowel resection has been suggested [124, 125]. The majority (up to 80%) of these children's outcomes were reported to be positive [126]. With the exception of sporadic small resections during MACE or ostomy construction, however, bowel resection does not play a role in the surgical therapy of NBD [127]. To promote a quicker and more thorough bowel evacuation, several authorities advise routinely taking into account bowel resection at the moment MACE is created .Others, on the other hand, contend that bowel resection should only be performed in a select few circumstances where there is a solid indication.

3.17 Colostomy

A colostomy comprises taking part of the large intestine to the abdomen's surface to form a stoma. Stool is collected in an external bag worn by the patient over the stoma [63]. It is believed in extremely intractable cases or when stool incontinence complicates other problems, such as pressure injury management. Colostomy formation early after spinal cord injury has also been shown to improve independence and increase acceptability of bowel management [128]. Bowel diversion with colostomy is a final alternative option for patients who have consumed all other treatments. In these patients, colostomy was shown to reduce hospitalizations due to bowel dysfunction as well as improve independence and quality of life [129].

4. CONCLUSION

This revies concluded that neurogenic bowel dysfunction is a pathophysiological phrase that refer to a variety of symptoms that can significantly affect a patient's quality of life. The symptoms that follow include persistent constipation and fecal incontinence, which have a significant influence on the patient's capability for social or occupational function. Most patients with bowel dysfunction are managed by a range of methods, involving conservative, medical and even surgical approaches. The goal of management is to avoid fecal incontinence, constipation, minimizing time spent toileting, preventing complications and optimizing quality of life.

5. AUTHOR CONTRIBUTION STATEMENT

All the authors read and approved the final version of the manuscript.

6. CONFLICT OF INTEREST

Conflict of interest declared none.

7. REFRENCES

1. Coggrave M, Norton C. Neurogenic bowel. Handb Clin Neurol. 2013;110:221-8. doi: 10.1016/B978-0-444-52901-5.00018-6, PMID 23312643.
2. Gor RA, Katorski JR, Elliott SP. Medical and surgical management of neurogenic bowel. Curr Opin Urol. 2016 Jul 26;26(4):369-75. doi: 10.1097/MOU.0000000000000299, PMID 27152922.
3. Johns JS, Krogh K, Ethans K, Chi J, Querée M, Eng JJ et al. Pharmacological management of neurogenic bowel dysfunction after spinal cord injury and multiple sclerosis: A systematic review and clinical implications. J Clin Med. 2021 Feb 22;10(4):882. doi: 10.3390/jcm10040882, PMID 33671492, PMCID PMC7926827.
4. Beierwaltes P, Church P, Gordon T, Ambartsumyan L. Bowel function and care: Guidelines for the care of people with spina bifida. J Pediatr Rehabil Med. 2020;13(4):491-8. doi: 10.3233/PRM-200724, PMID 33252093.
5. Coggrave M. Neurogenic continence. Part 3: Bowel management strategies. Br J Nurs. 2008;17(15):962-8. doi: 10.12968/bjon.2008.17.15.30698, PMID 18983017.
6. Bharucha AE. Fecal incontinence. American College of Gastroenterology. [(accessed on 11 January 2021)]. Available from: https://gi.org/topics/fecal-incontinence/.
7. Coggrave M, Norton C, Wilson-Barnett J. Management of neurogenic bowel dysfunction in the community after spinal cord injury: A postal survey in the United Kingdom. Spinal Cord. 2009;47(4):323-30; quiz 331. doi: 10.1038/sc.2008.137, PMID 19015665.
8. Coggrave MJ, Norton C. The need for manual evacuation and oral laxatives in the management of neurogenic bowel dysfunction after spinal cord injury: A randomized controlled trial of a stepwise protocol. Spinal Cord. 2010;48(6):504-10. doi: 10.1038/sc.2009.166, PMID 19949417.
9. Glickman S, Kamm MA. Bowel dysfunction in spinal-cord-injury patients. Lancet. 1996;347(9016):1651-3. doi: 10.1016/s0140-6736(96)91487-7, PMID 8642958.
10. Lynch AC, Wong C, Anthony A, Dobbs BR, Frizelle FA. Bowel dysfunction following spinal cord injury: a description of bowel function in a spinal cord-injured population and comparison with age and gender matched controls. Spinal Cord. 2000;38(12):717-23. doi: 10.1038/sj.sc.3101058, PMID 11175370.
11. Kumar L, Athanasakos E, Emmanuel AV. Evaluation and treatment of neurogenic bowel dysfunction–a review. Eur Neurol Rev. 2016;11(2):109-15. doi: 10.17925/ENR.2016.11.02.109.
12. Preziosi G, Emmanuel A. Neurogenic bowel dysfunction: pathophysiology, clinical manifestations and treatment. Expert Rev Gastroenterol Hepatol. 2009;3(4):417-23. doi: 10.1586/egh.09.31, PMID 19673628.
13. Krogh K, Nielsen J, Djurhuus JC, Mosdal C, Sabroe S, Laurberg S. Colorectal function in patients with spinal cord lesions. Dis Colon Rectum. 1997;40(10):1233-9. doi: 10.1007/BF02055170, PMID 9336119.

14. Glickman S, Kamm MA. Bowel dysfunction in spinal-cord-injury patients. Lancet. 1996;347(9016):1651-3. doi: 10.1016/s0140-6736(96)91487-7, PMID 8642958.
15. Finnerup NB, Faaborg P, Krogh K, Jensen TS. Abdominal pain in long-term spinal cord injury. Spinal Cord. 2008;46(3):198-203. doi: 10.1038/sj.sc.3102097, PMID 17621311.
16. Chia YW, Fowler CJ, Kamm MA, Henry MM, Lemieux MC, Swash M. Prevalence of bowel dysfunction in patients with multiple sclerosis and bladder dysfunction. J Neurol. 1995;242(2):105-8. doi: 10.1007/BF00887825, PMID 7707085.
17. Rao SS, Tuteja AK, Vellema T, Kempf J, Stessman M. Dyssynergic defecation: demographics, symptoms, stool patterns, and quality of life. J Clin Gastroenterol. 2004;38(8):680-5. doi: 10.1097/01.mcg.0000135929.78074.8c, PMID 15319652.
18. Bakke A, Myhr KM, Grønning M, Nyland H. Bladder, bowel and sexual dysfunction in patients with multiple sclerosis—a cohort study. Scand J Urol Nephrol Suppl. 1996;179:61-6. PMID 8908666.
19. Krogh K, Ostergaard K, Sabroe S, Laurberg S. Clinical aspects of bowel symptoms in Parkinson's disease. Acta Neurol Scand. 2008;117(1):60-4. doi: 10.1111/j.1600-0404.2007.00900.x, PMID 18095955.
20. Doshi VS, Say JH, Young SH-Y, Doraisamy P. Complications in stroke patients: a study carried out at the Rehabilitation Medicine Service, Changi General Hospital. Singapore Med J. 2003;44(12):643-52. PMID 14770260.
21. Rajindrajith S, Devanarayana NM, Benninga MA. Review article: Faecal incontinence in children: Epidemiology, pathophysiology, clinical evaluation and management. Aliment Pharmacol Ther. 2013;37(1):37-48. doi: 10.1111/apt.12103, PMID 23106105.
22. Chung EA, Emmanuel AV. Gastrointestinal symptoms related to autonomic dysfunction following spinal cord injury. Prog Brain Res. 2006;152:317-33. doi: 10.1016/S0079-6123(05)52021-1, PMID 16198710.
23. Brookes SJ, Dinning PG, Gladman MA. Neuroanatomy and physiology of colorectal function and defaecation: From basic science to human clinical studies. Neurogastroenterol Motil. 2009;21;Suppl 2:9-19. doi: 10.1111/j.1365-2982.2009.01400.x, PMID 19824934.
24. Mosiello G, Safder S, Marshall D, Rolle U, Benninga MA. Neurogenic bowel dysfunction in children and adolescents. J Clin Med. 2021 Apr 13;10(8):1669. doi: 10.3390/jcm10081669, PMID 33924675, PMCID PMC8069792.
25. Krogh K, Christensen P, Laurberg S. Colorectal symptoms in patients with neurological diseases. Acta Neurol Scand. 2001;103(6):335-43. doi: 10.1034/j.1600-0404.2001.103006335.x, Google Scholar, PMID 11421845.
26. Northrup H, Volcik KA. Spina bifida and other neural tube defects. Curr Probl Pediatr. 2000;30(10):313-32. doi: [Green Version]. PMID 11147289, Google Scholar.
27. Bauer SB. The management of the myelodysplastic child: A paradigm shift. BJU Int. 2003;92;Suppl 1:23-8. doi: 10.1046/j.1464-410x.92.s1.2.x, Google Scholar, PMID 12969005.
28. Guzman L, Bauer SB, Hallett M, Khoshbin S, Colodny AH, Retik AB. Evaluation and management of children with sacral agenesis. Urology. 1983;22(5):506-10. doi: 10.1016/0090-4295(83)90230-3, PMID 6649205, Google Scholar.
29. Borrelli M, Bruschini H, Nahas WC, Figueiredo JA, Prado MJ, Spinola R et al. Sacral agenesis: Why is it so frequently misdiagnosed? Urology. 1985;26(4):351-5. doi: 10.1016/0090-4295(85)90182-7, PMID 4049612, Google Scholar.
30. Mosiello G, Capitanucci ML, Gatti C, Adorisio O, Lucchetti MC, Silveri M et al. How to investigate neurovesical dysfunction in children with anorectal malformations. J Urol. 2003;170(4 Pt 2):1610-3. doi: 10.1097/01.ju.0000083883.16836.91, PMID 14501674, Google Scholar.
31. Peña A. Posterior sagittal approach for the correction of anorectal malformations. Adv Surg. 1986;19:69-100. Google Scholar, PMID 3942039.
32. Richards CL, Malouin F. Cerebral palsy: Definition, assessment and rehabilitation. Handb Clin Neurol. 2013;111:183-95. doi: 10.1016/B978-0-444-52891-9.00018-X, Google Scholar, PMID 23622163.
33. Ozturk M, Oktem F, Kisioglu N, Demirci M, Altuntas I, Kutluhan S et al. Bladder and bowel control in children with cerebral palsy: case-control study. Croat Med J. 2006;47(2):264-70. PMID 16625691, Google Scholar.
34. Von Wendt L, Similä S, Niskanen P, Järvelin MR. Development of bowel and bladder control in the mentally retarded. Dev Med Child Neurol. 1990;32(6):515-8. doi: 10.1111/j.1469-8749.1990.tb16977.x, PMID 2365145, Google Scholar.
35. Böhmer CJ, Taminiau JA, Klinkenberg-Knol EC, Meuwissen SG. The prevalence of constipation in institutionalized people with intellectual disability. J Intellect Disabil Res. 2001;45(3):212-8. doi: 10.1046/j.1365-2788.2001.00300.x, Google Scholar, PMID 11422645.
36. Siiiiiiiiiiiiiiiiiiiiiiiiiiiite.
37. Brookes SJ, Dinning PG, Gladman MA. Neuroanatomy and physiology of colorectal function and defaecation: From basic science to human clinical studies. Neurogastroenterol Motil. 2009;21;Suppl 2:9-19. doi: 10.1111/j.1365-2982.2009.01400.x, PMID 19824934, Google Scholar.
38. Krogh K, Lie HR, Bilenberg N, Laurberg S. Bowel function in Danish children with myelomeningocele. APMIS Suppl. 2003;109(109):81-5. PMID 12874955, Google Scholar.
39. Krogh K, Christensen P, Laurberg S. Colorectal symptoms in patients with neurological diseases. Acta Neurol Scand. 2001;103(6):335-43. doi: 10.1034/j.1600-0404.2001.103006335.x, PMID 11421845.
40. Krogh K, Lie HR, Bilenberg N, Laurberg S. Bowel function in Danish children with myelomeningocele. APMIS Suppl. 2003;109(109):81-5. PMID 12874955, Google Scholar.
41. Krogh K, Mosdal C, Laurberg S. Gastrointestinal and segmental colonic transit times in patients with acute and chronic spinal cord lesions. Spinal Cord. 2000;38(10):615-21. doi: , Google Scholar, PMID PubMed[Green Version].
42. Di Lorenzo C, Benninga MA. Pathophysiology of pediatric fecal incontinence. Gastroenterology. 2004;126 (Suppl. S1):S33-40. doi: , Google Scholar, PMID PubMed.
43. Emmanuel AV, Chung EA, Kamm MAF, Middleton F. Relationship between gut-specific autonomic testing

and bowel dysfunction in spinal cord injury patients. Spinal Cord. 2009;47(8):623-7. doi: [Green Version]. PMID 19274057, Google Scholar.
44. Johns JS, Chapter 139. Neurogenic bowel, essentials of physical Medicine and rehabilitation. 4th ed. Elsevier, ISBN 9780323549479; 2020. p. 786-91. doi: 10.1016/B978-0-323-54947-9.00139-5.
45. Ashraf W, Wszolek ZK, Pfeiffer RF, Normand M, Maurer K, Srb F, et al. Anorectal function in fluctuating (on-off) Parkinson's disease: evaluation by combined anorectal manometry and electromyography. Mov Disord. 1995;10(5):650-7. doi: 10.1002/mds.870100519, PMID 8552119.
46. Singaram C, Ashraf W, Gaumnitz EA, Torbey C, Sengupta A, Pfeiffer R, et al. Dopaminergic defect of enteric nervous system in Parkinson's disease patients with chronic constipation. Lancet. 1995;346(8979):861-4. doi: 10.1016/s0140-6736(95)92707-7, PMID 7564669.
47. Banwell JG, Creasey GH, Aggarwal AM, Mortimer JT. Management of the neurogenic bowel in patients with spinal cord injury. Urol Clin North Am. 1993;20(3):517-26. doi: CrossRef, PMID 8351776, Google Scholar.
48. Camilleri M, Bharucha AE. Gastrointestinal dysfunction in neurologic disease. Semin Neurol. 1996;16(3):203-16. doi: 10.1055/s-2008-1040977, PMID 9085470, Google Scholar.
49. Stiens SA, Bergman SB, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: Clinical evaluation and rehabilitative management. Arch Phys Med Rehabil. 1997;78(3);Suppl:S86-S102. doi: 10.1016/s0003-9993(97)90416-0, PMID 9084372, Google Scholar.
50. Krogh K, Nielsen J, Djurhuus JC, Mosdal C, Sabroe S, Laurberg SColorectal function in patients with spinal cord lesions. Dis Colon Rectum. 1997;40(10):1233-9. doi: 10.1007/BF02055170, Google Scholar, PMID 9336119.
51. Leduc BE, Giasson M, Favreau-Ethier M, Lepage Y. Colonic transit time after spinal cord injury. J Spinal Cord Med. 1997;20(4):416-21. doi: 10.1080/10790268.1997.11719549, Google Scholar, PMID 9360223.
52. Bharucha AE. Fecal incontinence. American College of Gastroenterology. [(accessed on 11 January 2021)]. Available from: https://gi.org/topics/fecal-incontinence/.
53. Wald A. Constipation and defecation problems. American College of Gastroenterology. [(accessed on 11 January 2021)]. Available from: https://gi.org/topics/constipation-and-defection-problems/.
54. Cameron AP, Rodriguez GM, Gursky A, He C, Clemens JQ, Stoffel JT. The severity of bowel dysfunction in patients with neurogenic bladder. J Urol. 2015;194(5):1336-41. doi: 10.1016/j.juro.2015.04.100, PMID 25956470. This cross-sectional analysis of a prospective neurogenic bladder database highlights the characteristics of neurogenic bowel dysfunction in this population. One important finding was that patients with worse bladder function experienced worse bowel dysfunction.
55. Emmanuel A. Neurogenic bowel dysfunction. F1000 Fac Rev-1800. 2019 Oct 28;8:F1000Res. doi: 10.12688/f1000research.20529.1, PMID 31700610, PMCID PMC6820819.
56. Preziosi G, Emmanuel A. Neurogenic bowel dysfunction: pathophysiology, clinical manifestations and treatment. Expert Rev Gastroenterol Hepatol. 2009;3(4):417-23. doi: 10.1586/egh.09.31, PMID 19673628.
57. Correa GI, Rotter KP. Clinical evaluation and management of neurogenic bowel after spinal cord injury. Spinal Cord. 2000;38(5):301-8. doi: 10.1038/sj.sc.3100851, PMID 10822403.
58. Agachan F, Chen T, Pfeifer J, Reissman P, Wexner SD. A constipation scoring system to simplify evaluation and management of constipated patients. Dis Colon Rectum. 1996;39(6):681-5. doi: 10.1007/BF02056950, PMID 8646957.
59. Staskin D, Kelleher C, Avery K, et al. Initial assessment of urinary and faecal incontinence in adult male and female patients—patient reported outcome assessment. Incontinence. Proceedings of the fourth international consultation on incontinence. Plymouth: Health Communications Publications Limited; 2009. p. 331-412.
60. Cotterill N, Madersbacher H, Wyndaele JJ, Apostolidis A, Drake MJ, Gajewski J, et al. Neurogenic bowel dysfunction: Clinical management recommendations of the Neurologic Incontinence Committee of the Fifth International Consultation on Incontinence 2013. Neurourol Urodyn. 2018;37(1):46-53. doi: 10.1002/nau.23289, PMID 28640977.
61. Wyndaele JJ, Kovindha A, Igawa Y, Madersbacher H, Radziszewski P, Ruffion A, et al. Neurologic fecal incontinence. Neurourol Urodyn. 2010;29(1):207-12. doi: 10.1002/nau.20853, PMID 20025022.
62. Cotterill N, Madersbacher H, Wyndaele JJ, et al. Neurogenic bowel dysfunction: Clinical management recommendations of the Neurologic Incontinence Committee of the Fifth International Consultation on Incontinence 2013. Neurourol Urodyn. 2018;37(1):46-53. doi: 10.1002/nau.23289, PMID [CrossRef], Google Scholar.
63. Mosiello G, Safder S, Marshall D, Rolle U, Benninga MA. Neurogenic bowel dysfunction in children and adolescents. J Clin Med. 2021 Apr 13;10(8):1669. doi: 10.3390/jcm10081669, PMID 33924675, PMCID PMC8069792.
64. Vande Velde S, Van Biervliet S, Van Renterghem K, Van Laecke E, Hoebeke P, Van Winckel M. Achieving fecal continence in patients with spina bifida: a descriptive cohort study. J Urol. 2007;178(6):2640-4; discussion 2644. doi: 10.1016/j.juro.2007.07.060, PMID 17945290.
65. Cameron KJ, Nyulasi IB, Collier GR, Brown DJ. Assessment of the effect of increased dietary fibre intake on bowel function in patients with spinal cord injury. Spinal Cord. 1996;34(5):277-83. doi: 10.1038/sc.1996.50, PMID 8963975.
66. Clinical practice guidelines: Neurogenic bowel management in adults with spinal cord injury. Spinal Cord Medicine Consortium. J Spinal Cord Med. 1998;21(3):248-93. doi: 10.1080/10790268.1998.11719536, PMID 9863937.
67. Institute of Medicine. Dietary reference intakes for water, potassium, sodium, chloride, and sulfate. Washington, DC: National Academies Press; 2005. p. 638.
68. Emmanuel A. Managing neurogenic bowel dysfunction. Clin Rehabil. 2010;24(6):483-8. doi:

10.1177/0269215509353253, PMID [CrossRef], Google Scholar.
69. Emmanuel A. Managing neurogenic bowel dysfunction. Clin Rehabil. 2010;24(6):483-8. doi: 10.1177/0269215509353253, PMID 20511302.
70. Ayaş S, Leblebici B, Sözay S, Bayramoğlu M, Niron EA. The effect of abdominal massage on bowel function in patients with spinal cord injury. Am J Phys Med Rehabil. 2006;85(12):951-5. doi: 10.1097/01.phm.0000247649.00219.c0, PMID 17117000.
71. Faaborg PM, Christensen P, Krassioukov A, et al. Autonomic dysreflexia during bowel evacuation procedures and bladder filling in subjects with spinal cord injury. Spinal Cord. 2014;52(6):494-8. doi: 10.1038/sc.2014.45, PMID [CrossRef], Google Scholar F1000 [recommendation].
72. Emmanuel AV, Krogh K, Bazzocchi G, et al. Consensus review of best practice of transanal irrigation in adults. Spinal Cord. 2013;51(10):732-8. doi: 10.1038/sc.2013.86, PMID [CrossRef], Google Scholar.
73. Emmanuel A, Kumar G, Christensen P, et al. Long-term cost-effectiveness of transanal irrigation in patients with neurogenic bowel dysfunction. PLOS ONE. 2016;11(8):e0159394. doi: 10.1371/journal.pone.0159394. (PMC Free article). PMID [CrossRef], Google Scholar.
74. Creasey GH, Grill JH, Korsten M, et al. An implantable neuroprosthesis for restoring bladder and bowel control to patients with spinal cord injuries: A multicenter trial. Arch Phys Med Rehabil. 2001;82(11):1512-9. doi: 10.1053/apmr.2001.25911, PMID [CrossRef], Google Scholar.
75. Minardi D, Muzzonigro G. Lower urinary tract and bowel disorders and multiple sclerosis: Role of sacral neuromodulation: a preliminary report. Neuromodulation. 2005;8(3):176-81. doi: 10.1111/j.1525-1403.2005.05236.x, PMID [CrossRef], Google Scholar.
76. Curry JI, Osborne A, Malone PS. The MACE procedure: Experience in the United Kingdom. J Pediatr Surg. 1999;34(2):338-40. doi: 10.1016/s0022-3468(99)90204-x, PMID [CrossRef], Google Scholar.
77. Gerharz EW, Vik V, Webb G, et al. The value of the MACE (Malone antegrade colonic enema) procedure in adult patients. J Am Coll Surg. 1997;185(6):544-7. doi: 10.1016/s1072-7515(97)00125-7, PMID [CrossRef], Google Scholar.
78. Cowlam S, Watson C, Elltringham M, et al. Percutaneous endoscopic colostomy of the left side of the colon. Gastrointest Endosc. 2007;65(7):1007-14. doi: 10.1016/j.gie.2007.01.012, PMID [CrossRef], Google Scholar F1000 [recommendation].
79. Branagan G, Tromans A, Finnis D. Effect of stoma formation on bowel care and quality of life in patients with spinal cord injury. Spinal Cord. 2003;41(12):680-3. doi: 10.1038/sj.sc.3101529, PMID [CrossRef], Google Scholar.
80. Frisbie JH, Ahmed N, Hirano I, et al. Diversion colitis in patients with myelopathy: clinical, endoscopic, and histopathological findings. J Spinal Cord Med. 2000;23(2):142-9. doi: 10.1080/10790268.2000.11753522, PMID [CrossRef], Google Scholar F1000 [recommendation].
81. Randell N, Lynch AC, Anthony A, et al. Does a colostomy alter quality of life in patients with spinal cord injury? A controlled study. Spinal Cord. 2001;39(5):279-82. doi: 10.1038/sj.sc.3101156, PMID [CrossRef], Google Scholar.
82. Safadi BY, Rosito O, Nino-Murcia M, et al. Which stoma works better for colonic dysmotility in the spinal cord injured patient? Am J Surg. 2003;186(5):437-42. doi: 10.1016/j.amjsurg.2003.07.007, PMID [CrossRef], Google Scholar.
83. Martinez L, Neshatian L, Khavari R. Neurogenic bowel dysfunction in patients with neurogenic bladder. Curr Bladder Dysfunct Rep. 2016 Dec;11(4):334-40. doi: 10.1007/s11884-016-0390-3. PMID 28717406, PMCID PMC5510247.
84. Johns JS, Krogh K, Ethans K, Chi J, Querée M, Eng JJ et al. Pharmacological management of neurogenic bowel dysfunction after spinal cord injury and multiple sclerosis: A systematic review and clinical implications. J Clin Med. 2021 Feb 22;10(4):882. doi: 10.3390/jcm10040882, PMID 33671492, PMCID PMC7926827.
85. Coggrave M, Norton C, Wilson-Barnett J. Management of neurogenic bowel dysfunction in the community after spinal cord injury: A postal survey in the United Kingdom. Spinal Cord. 2009;47(4):323-30; quiz 331. doi: 10.1038/sc.2008.137, PMID 19015665.
86. Inskip JA, Lucci V-EM, McGrath MS, Willms R, Claydon VE. A community perspective on bowel management and quality of life after spinal cord injury: the influence of autonomic dysreflexia. J Neurotrauma. 2018;35(9):1091-105. doi: 10.1089/neu.2017.5343, PMID 29239268.
87. Kienzle-Horn S, Vix JM, Schuijt C, Peil H, Jordan CC, Kamm MA. Efficacy and safety of Bisacodyl in the acute treatment of constipation: A double-blind, randomized, placebo-controlled study. Aliment Pharmacol Ther. 2006;23(10):1479-88. doi: 10.1111/j.1365-2036.2006.02903.x, PMID 16669963.
88. Peña A, De La Torre L, Belkind-Gerson J, Lovell M, Ketzer J, Bealer J et al. Enema-Induced spastic left colon syndrome: an unintended consequence of chronic enema use. J Pediatr Surg. 2021;56(2):424-8. doi: 10.1016/j.jpedsurg.2020.10.027, PMID 33199058.
89. Leibold S, Ekmark E, Adams RC. Decision-making for a successful bowel continence program. European journal of pediatric surgery : official journal of Austrian Association of Pediatric Surgery [et al] = Zeitschrift fur Kinderchirurgie. 2000;10;Suppl 1:26-30.
90. Rendeli C, Ausili E, Tabacco F, Focarelli B, Pantanella A, Di Rocco C et al. Polyethylene glycol 4000 vs. lactulose for the treatment of neurogenic constipation in myelomeningocele children: A randomized-controlled clinical trial. Aliment Pharmacol Ther. 2006;23(8):1259-65. doi: 10.1111/j.1365-2036.2006.02872.x, PMID 16611288.
91. Korsten MA, Rosman AS, Ng A, Cavusoglu E, Spungen AM, Radulovic M, et al. Infusion of neostigmine-glycopyrrolate for bowel evacuation in persons with spinal cord injury. Am J Gastroenterol. 2005;100(7):1560-5. doi: 10.1111/j.1572-0241.2005.41587.x, PMID 15984982.
92. Rosman AS, Chaparala G, Monga A, Spungen AM, Bauman WA, Korsten MA. Intramuscular neostigmine and glycopyrrolate safely accelerated bowel evacuation in patients with spinal cord injury and defecatory disorders. Dig Dis Sci. 2008;53(10):2710-3. doi: 10.1007/s10620-008-0216-z, PMID 18338263.

93. Gourcerol G, Vitton V, Leroi AM, Michot F, Abysique A, Bouvier M. How sacral nerve stimulation works in patients with faecal incontinence. Colorectal Dis. 2011;13(8):e203-11. doi: 10.1111/j.1463-1318.2011.02623.x, PMID 21689312, Google Scholar.
94. Sheldon R, Kiff ES, Clarke A, Harris ML, Hamdy S. Sacral nerve stimulation reduces corticoanal excitability in patients with faecal incontinence. Br J Surg. 2005;92(11):1423-31. doi: 10.1002/bjs.5111, PMID 16044426, Google Scholar.
95. House JG, Stiens SA. Pharmacologically initiated defecation for persons with spinal cord injury: Effectiveness of three agents. Arch Phys Med Rehabil. 1997;78(10):1062-5. doi: 10.1016/s0003-9993(97)90128-3, PMID 9339153, Google Scholar.
96. Patton V, Stewart P, Lubowski DZ, Cook IJ, Dinning PG. Sacral nerve stimulation fails to offer long-term benefit in patients with slow-transit constipation. Dis Colon Rectum. 2016;59(9):878-85. doi: 10.1097/DCR.0000000000000653, Google Scholar, PMID 27505117.
97. Van der Wilt AA, van Wunnik BPW, Sturkenboom R, Han-Geurts IJ, Melenhorst J, Benninga MA et al. Sacral neuromodulation in children and adolescents with chronic constipation refractory to conservative treatment. Int J Colorectal Dis. 2016;31(8):1459-66. doi: [Green Version]. PMID 27294660, Google Scholar.
98. Holzer B, Rosen HR, Novi G, Ausch C, Hölbling N, Schiessel R. Sacral nerve stimulation for neurogenic faecal incontinence. Br J Surg. 2007;94(6):749-53. doi: 10.1002/bjs.5499, Google Scholar, PMID 17410558.
99. Cotterill N, Madersbacher H, Wyndaele JJ, Apostolidis A, Drake MJ, Gajewski J, et al. Neurogenic bowel dysfunction: Clinical management recommendations of the Neurologic Incontinence Committee of the Fifth International Consultation on Incontinence 2013. Neurourol Urodyn. 2018;37(1):46-53. doi: 10.1002/nau.23289, PMID 28640977.
100. Mosiello G, Marshall D, Rolle U, Crétolle C, Santacruz BG, Frischer J et al. Consensus review of best practice of transanal irrigation in children. J Pediatr Gastroenterol Nutr. 2017;64(3):343-52. doi: 10.1097/MPG.0000000000001483, PMID 27977546.
101. Kelly MS, Wiener JS, Liu T, Patel P, Castillo H, Castillo J; et al. Neurogenic bowel treatments and continence in children and adults with myelomeningocele. J Pediatr Rehabil Med. 2020;13(4):685-93. doi: 10.3233/PRM-190667, PMID 33325404, Google Scholar.
102. Ambartsumyan L, Rodriguez L. Bowel management in children with spina bifida. J Pediatr Rehabil Med. 2018;11(4):293-301. doi: 10.3233/PRM-170533, PMID 30507592, Google Scholar.
103. Gor RA, Katorski JR, Elliott SP. Medical and surgical management of neurogenic bowel. Curr Opin Urol. 2016;26(4):369-75. doi: 10.1097/MOU.0000000000000299, PMID 27152922, Google Scholar.
104. Mosiello G, Marshall D, Rolle U, Crétolle C, Santacruz BG, Frischer J et al. Consensus review of best practice of transanal irrigation in children. J Pediatr Gastroenterol Nutr. 2017;64(3):343-52. doi: 10.1097/MPG.0000000000001483, PMID 27977546, Google Scholar.
105. Johnston AW, Wiener JS, Todd Purves JT. Pediatric neurogenic bladder and bowel dysfunction: Will my child ever be out of diapers? Eur Urol Focus. 2020;6(5):838-67. doi: 10.1016/j.euf.2020.01.003, PMID 31982364, Google Scholar.
106. Wong WD, Jensen LL, Bartolo DCC, Rothenberger DA. Artificial anal sphincter. Dis Colon Rectum. 1996;39(12):1345-51. doi: 10.1007/BF02054522, Google Scholar, PMID 8969658.
107. Griffiths DM, Malone PS. The Malone antegrade continence enema (MACE). J Pediatr Surg. 1995;30(1):68-71. doi: 10.1016/0022-3468(95)90613-4, PMID 7722834, Google Scholar.
108. Ok JH, Kurzrock EA. Objective measurement of quality of life changes after ACE Malone using FICQOL survey. J Pediatr Urol. 2011;7(3):389-93. doi: [Green Version]. PMID 21527223, Google Scholar.
109. Brinas P, Zalay N, Philis A, Castel-Lacanal E, Barrieu M, Portier G. Use of Malone antegrade continence enemas in neurological bowel dysfunction. J Visc Surg. 2020;157(6):453-9. doi: 10.1016/j.jviscsurg.2020.03.007, Google Scholar, PMID 32247623.
110. Bani-Hani AH, Cain MP, Kaefer M, Meldrum KK, King S, Johnson CS et al. The Malone antegrade continence enema: single institutional review. J Urol. 2008;180(3):1106-10. doi: 10.1016/j.juro.2008.05.062, Google Scholar, PMID 18639902.
111. VanderBrink BA, Cain MP, Kaefer M, Meldrum KK, Misseri R, Rink RC. Outcomes following Malone antegrade continence enema and their surgical revisions. J Pediatr Surg. 2013;48(10):2134-9. doi: 10.1016/j.jpedsurg.2013.05.010, Google Scholar, PMID 24094969.
112. Lynch AC, Beasley SW, Robertson RW, Morreau PN. Comparison of results of laparoscopic and open antegrade continence enema procedures. Pediatr Surg Int. 1999;15(5-6):343-6. doi: 10.1007/s003830050595, Google Scholar, PMID 10415282.
113. Kim J, Beasley SW, Maoate K. Appendicostomy stomas and antegrade colonic irrigation after laparoscopic antegrade continence enema. J Laparoendosc Adv Surg Tech A. 2006;16(4):400-3. doi: 10.1089/lap.2006.16.400, Google Scholar, PMID 16968193.
114. Malone PS, Curry JI, Osborne A. The antegrade continence enema procedure why, when and how? World J Urol. 1998;16(4):274-8. doi: 10.1007/s003450050066, Google Scholar, PMID 9775427.
115. Bevill MD, Bonnett K, Arlen A, Cooper C, Baxter C, Storm DW. Outcomes and satisfaction in pediatric patients with Chait cecostomy tubes. J Pediatr Urol. 2017;13(4):365-70. doi: 10.1016/j.jpurol.2017.04.008, PMID 28545800.
116. Rawat DJ, Haddad M, Geoghegan N, Clarke S, Fell JM. Percutaneous endoscopic colostomy of the left colon: A new technique for management of intractable constipation in children. Gastrointest Endosc. 2004;60(1):39-43. doi: 10.1016/s0016-5107(04)01286-6, PMID 15229423, Google Scholar.
117. Kelly MS. Malone antegrade continence enemas vs. cecostomy vs. transanal irrigation—What is new and how do we counsel patients? Curr Urol Rep. 2019;20(8):41. doi: 10.1007/s11934-019-0909-1, PMID 31183573, Google Scholar.
118. Beckers GMA, de Meij TGJ. Peritonitis after endoscopic caecostomy with a Chait trapdoor catheter: what lessons can be learned? J Pediatr Gastroenterol Nutr.

2012;55(2):217-8. doi: 10.1097/MPG.0b013e318231eefb, PMID 21857245, Google Scholar.

119. Hocevar B, Gray M. Intestinal diversion (colostomy or ileostomy) in patients with severe bowel dysfunction following spinal cord injury. J Wound Ostomy Continence Nurs. 2008;35(2):159-66. doi: 10.1097/01.WON.0000313638.29623.40, PMID 18344790, Google Scholar.
120. Coggrave MJ, Ingram RM, Gardner BP, Norton CS. The impact of stoma for bowel management after spinal cord injury. Spinal Cord. 2012;50(11):848-52. doi: 10.1038/sc.2012.66, PMID 22710944, Google Scholar.
121. Branagan G, Tromans A, Finnis D. Effect of stoma formation on bowel care and quality of life in patients with spinal cord injury. Spinal Cord. 2003;41(12):680-3. doi: 10.1038/sj.sc.3101529, Google Scholar, PMID 14639447[Green Version].
122. Cooper EA, Bonne Lee B, Muhlmann M. Outcomes following stoma formation in patients with spinal cord injury. Colorectal Dis. 2019;21(12):1415-20. doi: 10.1111/codi.14753, Google Scholar, PMID 31274228.
123. Boucher M, Dukes S, Bryan S, Branagan G. Early colostomy formation can improve independence following spinal cord injury and increase acceptability of bowel management. Top Spinal Cord Inj Rehabil. 2019;25(1):23-30. doi: 10.1310/sci18-00026, PMID 30774287, Google Scholar.
124. Gasior A, Reck C, Vilanova-Sanchez A, Diefenbach KA, Yacob D, Lu P et al. Surgical management of functional constipation: an intermediate report of a new approach using laparoscopic sigmoid resection combined with Malone appendicostomy. J Pediatr Surg. 2018;53(6):1160-2. doi: 10.1016/j.jpedsurg.2018.02.074, Google Scholar, PMID 29588075.
125. Kilpatrick JA, Zobell S, Leeflang EJ, Cao D, Mammen L, Rollins MD. Intermediate and long-term outcomes of a bowel management program for children with severe constipation or fecal incontinence. J Pediatr Surg. 2020;55(3):545-8. doi: 10.1016/j.jpedsurg.2019.10.062, PMID 31837840, Google Scholar.
126. Halleran DR, Sloots CEJ, Fuller MK, Diefenbach K. Adjuncts to bowel management for fecal incontinence and constipation, the role of surgery; appendicostomy, cecostomy, neoappendicostomy, and colonic resection. Semin Pediatr Surg. 2020;29(6):150998. doi: 10.1016/j.sempedsurg.2020.150998, Google Scholar, PMID 33288138.
127. Siminas S, Losty PD. Current surgical management of pediatric idiopathic constipation: A systematic review of published studies. Ann Surg. 2015;262(6):925-33. doi: 10.1097/SLA.0000000000001191, PMID 25775070, Google Scholar.
128. Boucher M, Dukes S, Bryan S, Branagan G. Early colostomy formation can improve independence following spinal cord injury and increase acceptability of bowel management. Top Spinal Cord Inj Rehabil. 2019;25(1):23-30. doi: 10.1310/sci18-00026, PMID 30774287.
129. Rosito O, Nino-Murcia M, Wolfe VA, Kiratli BJ, Perkash I. The effects of colostomy on the quality of life in patients with spinal cord injury: a retrospective analysis. J Spinal Cord Med. 2002;25(3):174-83. doi: 10.1080/10790268.2002.11753619, PMID 12214904.