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Neurogenic Bowel Dysfunction and Management after Spinal Cord Injury

Damien Debatisse*

Department of Neurophysiology IOM Neurosurgery, Technical University of Braunschweig, Brunswick, Germany

Abstract

People who have had a spinal cord injury (SCIs) often experience problems with their bowels. These include constipation, faecal incontinence, abdominal pain, bloating, diarrhea, nausea, vomiting and gas. Bowel problems can cause serious health issues such as dehydration, malnutrition, weight loss and skin breakdown. This paper will discuss the anatomy and physiology of the gut, how it works and what happens after an injury to the spinal cord. It will also explain the latest guidelines for assessing and managing people with neurogenic bowel.

Keywords: Spinal cord injury • Neurogenic bowel syndrome • Sympathetic nervous system • Parasympathetic nervous system

Introduction

Neurogenic Bowel Syndrome (NBS) is a chronic condition characterized by abnormal bowel function resulting from damage to the nervous system. NBS affects both men and women, although it is often diagnosed in people who are older than 50 years old. In addition to causing symptoms such as diarrhea, constipation and fecal incontinence, NBS may also cause pain, bloating, gas, nausea, vomiting and weight loss [1].

Description

The GI tract is innervated by both the autonomic and somatic nervous systems. The autonomic nervous system is made up of two divisions: the parasympathetic nervous and the sympathetic nervous systems. The parasympathetic nervous division controls smooth muscle contraction, glandular secretion and intestinal peristalsis. The sympathetic nervous division controls heart rate and blood pressure. Both divisions work together to ensure proper functioning of the digestive system. During times of crisis, the sympathetic nervous system (SNS) is activated, increasing the rate of digestion and shunting blood and nutrients to the body's working muscles. In addition, the parasympathetic nervous system (PNS) is inhibited, reducing the activity of the digestive organs. These two opposing systems work together to help keep the body functioning optimally during stressful situations [2].

SCI results in neurological dysfunction characterized by the dysmobility of various segments of gut (primarily colonic) and weakness of the pelvic floor muscles. Supra-sacral SCI typically results colonic hyper reflexivity, which is opposed by hyper reflexivity of the external sphincter, puborectalis muscle and pelvic floor musculatures, resulting in rectosphinatte dyssynergia and high colonic pressures. Conversely, conal or sub conal SCI causes hypo reflexivity or flaccidity of the colon, rectum, sphincters, puborectalis and pelvic floor muscles, resulting in low pressure, but uncontrolled fecal incongruence [3].

While both types of SI can lead to fecal leakage, hyper reflexive bowels can also cause an autonomic crisis in persons with SCIs above T6 due to uninhibited sympathetic reflex activity along the greater splanctmic nerve in response to noxious stimuli below the level of injury. Bowel-related stimulation is the second leading cause of autonomic crises (only urological stimulation is more common) and may include gastro duodenal ulcers (gastric ulcer, duodenal), gallbladder disease (cholecystitis, choletolithiasis, cholelithasis), appendicitis, bowel obstruction, bowel impaction, bowel care reflexes (e.g., peristalsis) and hemorrhoids. Autonomic dysreflexia can be life threatening and requires immediate intervention as outlined by others [4].

Of note, autonomic dysreflexias from any source can profoundly decrease parasympathetic outflow and increase sympathetic outflow. Neurogenic bowel (NBD) from SCI is categorized under the Rome IV criteria as functional constipation (FCD) and defecatory dysfunction (DDD). Other SCI factors exacerbate problems related to FCD and DDD including decreased mobility, poor nutrition and hydration and use medications that are known to adversely affect GI motility (e.g., opioids, anticholinergic agents and anti-spasmodics).

A hypo reflexive neurogenic bladder pattern of dysfunction that occurs at or below the conical segments (conal/infraconal) may include injury of the cauda equine or sacral nerve roots. There are diminished or lost anococutaneous, bulbocaverneous and other lumbar sacral reflexes. External sphincter innervation is diminished or absent. Tone and strength of the pelvic floor muscles and the external anal sphincters are similarly affected. Inability to control defecation results in fecal incontinence. Hyporeflexic neurogenic bladder is exceptionally difficult to manage because of the lack of both PN and somatic reflex activity; however, studies using radio opaque markers showed that people with acute and chronically injured conal/infracoinal segments had significantly longer total gastrointestinal transit times (4.9 days and 3.6 days, respectively).

Constipation and feculent incontinence often occur and can present, individually or collectively after SCI as a consequence of dysfunctional bowel motility and sphincter tone. Both hyperreflextive and hypo reflexive NB increases the gut transit times which contribute to FC. Regardless of pattern of delay, constipate is a result of suspended stools movement throughout the colon, causing hard stools from the absorption of water and electrolytes through the intestinal lumen by inhibiting secret motor neurons in ENS; the pattern worsens under SNS activation associated AD. Likewise, DD occurs in both hyper reflexive and hyporeflexic NBs exemplified by the poor empthyng of the recto sigmoidal. Recto sphincter dysgynergia causes obstructed defecates with intermittent fecal in hyper reflexive NB [5].

Conclusion

As medical providers, it is important that we make sure that our patients with SCIs stay healthy, well and away from the hospital and without any illness

***Address for Correspondence:** Damien Debatisse, Department of Neurophysiology IOM Neurosurgery, Technical University of Braunschweig, Brunswick, Germany, E-mail: debatissed@gmail.com*

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as much as possible. We will also need to form close partnerships and educate them to achieve these goals. We are all aware of how difficult it is to deal with NB and entail setting expectations, patience, constant monitoring and follow-ups. We need to consider everyone's unique circumstances to successfully manage NB and enhance compliance. Our primary goal should be to help our patients have the best quality of live they can have. We must all work together to find new ways to improve our approaches to NB evaluation and management. More research and studies of NB are needed now and in the future so that we can develop more responsive ways to address patient needs.

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