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Physiology, Defecation

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Introduction

Defecation is the term given for the act of expelling feces from the digestive tract via the anus. It is a complex function that requires coordinated involvement from the gastrointestinal system, the nervous system, as well as the musculoskeletal system.[1] The frequency of **defecation** within a 24-hour period varies depending on age and diet, but most people tend to have a bowel movement 1 to 3 times daily.[2]

Cellular

The lining of the anus and rectum primarily columnar epithelium. This shifts to squamous epithelium in an area known as the transitional zone, located just superior to the dentate line.[3] Two sphincters control the act of **defecation**. The internal anal sphincter consists of smooth muscle cells under involuntary control, whereas the external anal sphincter consists of voluntarily-controlled striated muscle cells.[3]

Development

Defecation begins as an involuntary process early in life. Through the process of toilet training, children learn to control the urge to defecate and only perform the action when it is socially acceptable to do so. The age for acquiring this skill depends on the age that toilet training began for the child, as well as the method of training used.[4][5]

Organ Systems Involved

The colon is responsible for propelling feces toward the rectum and beginning the urge to defecate. The external anal sphincter and the puborectalis muscle relax to allow the passage of feces out of the rectum. Valsalva maneuver and abdominal muscle contraction are performed to increase intra-abdominal pressure and expel feces more rapidly.[1] Rectal afferent nerves are responsible for the sensation of rectal fullness and the urge to defecate. Sacral nerves S2-S4 supply innervation to the muscles most involved in the act of **defecation** via the pudendal nerve.[1][6]

Function

Defecation is necessary to expel undigested portions of food in addition to metabolic waste products like stercobilin from the body in the form of stool. Stool also contains bacteria and cellular debris from the gastrointestinal tract.

Mechanism

Colonic mass movements and peristalsis move intestinal contents distally into the rectum. Rectal filling activates mechanoreceptors in the rectal wall causing awareness of the need to defecate. As stool reaches the rectum, a small amount is allowed to pass through to the anal canal by an

involuntary relaxation of the internal anal sphincter. This action, known as the rectoanal inhibitory reflex, is necessary for anal sampling, which is the process of determining if the rectal contents are of the gaseous, solid, or liquid form.[1][7] At this time, if **defecation** is not socially acceptable or convenient, the rectal wall relaxes, and the need to defecate subsides temporarily. If it is a proper time to defecate, the person generally either sits or squats depending on their environment. Next, contraction of the abdominal muscles and performing the Valsalva maneuver while simultaneously relaxing the external anal sphincter and puborectalis muscle will expel feces from the body due to the pressure gradient generated between the rectum and anal canal. After fecal expulsion, the closing reflex occurs, which involves the external anal sphincter regaining its tone to maintain continence at rest.[1]

Related Testing

Colonic and anorectal manometry can be used to assess the contraction ability of the colon and anal sphincter muscles, which is especially useful in the setting of constipation or fecal incontinence.[1][8] Stool analysis can be performed to assess fecal content, which is most commonly performed to identify bacteria, blood, or other abnormal substances in the stool.[9] If abnormalities are present on fecal analysis, a flexible sigmoidoscopy or colonoscopy can be performed to investigate further possible pathology of the colon and rectum.[10]

Pathophysiology

The pathophysiology associated with **defecation** can subdivide into the following three conditions: diarrhea, constipation, and fecal incontinence.

Diarrhea

Diarrhea is an increase in stool frequency, liquidity, or volume.[11] It further subcategorizes into the following types:

- Secretory
- Osmotic
- Inflammatory
- Functional

Secretory diarrhea is caused either by decreased absorption or increased secretion of electrolytes and water by the intestinal epithelium; this commonly occurs in the setting of certain infections by certain bacteria like *Vibrio cholerae* or with certain malignancies that increase secretion such as a VIPoma or carcinoid tumor.[11] There are many medications that can increase intestinal fluid secretion as well, such as zidovudine and irinotecan among a long list of others.[12]

Osmotic diarrhea is characterized by a substance such as lactose or gluten drawing water into the bowel down the concentration gradient. This type of diarrhea will frequently occur in malabsorption syndromes such as lactose intolerance or celiac disease. It can also result from the ingestion of osmotically active substances like magnesium or sulfate, which are common ingredients in laxatives.[11]

Osmotic and secretory diarrhea is distinguishable from one another by calculating the fecal osmotic gap.

Fecal osmotic gap = 290 – 2 * (stool sodium + stool potassium)

A high osmotic gap (greater than 125 mOsm/kg) is characteristic of osmotic diarrhea whereas a low osmotic gap (less than 50 mOsm/kg) is more indicative of a secretory type.[13] Osmotic diarrhea is likely to decrease in severity and volume with fasting because fasting removes the causative osmotic load. However, secretory diarrhea will be unaffected by a fasting period.[13]

Inflammatory diarrhea will occur in inflammatory bowel diseases such as Crohn disease or ulcerative colitis, as well as in certain invasive intestinal infections like *Clostridium difficile* or *Shigella.*[14] In inflammatory diarrhea, the stool will contain blood, white blood cells along with mucous.[11]

The most common form of functional diarrhea is irritable bowel syndrome, which is one of the functional gastrointestinal disorders. This syndrome is often considered a diagnosis of exclusion with the aid of Rome criteria.[15] Its exact etiology is unknown, but it may involve alterations in the microbiome of the intestine in addition to the rapid transit time of contents through the digestive tract.[16]

Constipation

Constipation involves a decrease in **defecation** frequency, generally occurring 3 or fewer times per week. This can lead to hardening of the stool and straining when attempting **defecation**.[17] Constipation can be a side effect of many medications, often those that slow intestinal motility such as medications with anticholinergic properties. It can also be a result of a low-fiber diet or disorders that disrupt the necessarily coordinated muscle contractions for **defecation** such as multiple sclerosis or spinal cord injury.[17]

Fecal Incontinence

Fecal incontinence occurs most frequently in the elderly and is the inability to control the passage of stool. It can be congenital, such as in the setting of spinal cord defects or anorectal malformations, or it can be an acquired condition, often associated with an injury occurring during labor or anorectal surgery.[18]

Clinical Significance

Changes in **defecation** habits (bowel habits) is a common symptom in many disease processes and a common side effect to many therapies. Additionally, these changes could indicate alarming conditions such as colorectal malignancy. The occurrence likelihood increases if the alteration in bowel motion frequency correlates with symptoms of rectal bleeding, loss of weight, anemia, palpable abdominal mass, and others.[19][20]

Stool form alteration can be indicative of disease as in the case of steatorrhea. This term refers to excess fat in the stool which accompanies malabsorption related to the number of gastrointestinal conditions.[21]

Stool content also can provide helpful diagnostic information to the clinician regarding an underlying disease process.

Questions

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References

1. Palit S, Lunniss PJ, Scott SM. The physiology of human **defecation**. Dig. Dis. Sci. 2012 Jun;57(6):1445-64. [PubMed: 22367113]

2. Heaton KW, Radvan J, Cripps H, Mountford RA, Braddon FE, Hughes AO. **Defecation** frequency and timing, and stool form in the general population: a prospective study. Gut. 1992 Jun;33(6):818-24. [PMC free article: PMC1379343] [PubMed: 1624166]
3. Choby BA, George S. Toilet training. Am Fam Physician. 2008 Nov 01;78(9):1059-64. [PubMed: 19007052]
4. Blum NJ, Taubman B, Nemeth N. Relationship between age at initiation of toilet training and duration of training: a prospective study. Pediatrics. 2003 Apr;111(4 Pt 1):810-4. [PubMed: 12671117]
5. Yu SW, Rao SS. Anorectal physiology and pathophysiology in the elderly. Clin. Geriatr. Med. 2014 Feb;30(1):95-106. [PMC free article: PMC3910254] [PubMed: 24267605]
6. Sweetser S. Evaluating the patient with diarrhea: a case-based approach. Mayo Clin. Proc. 2012 Jun;87(6):596-602. [PMC free article: PMC3538472] [PubMed: 22677080]
7. Moon C, Zhang W, Sundaram N, Yarlagadda S, Reddy VS, Arora K, Helmrath MA, Naren AP. Drug-induced secretory diarrhea: A role for CFTR. Pharmacol. Res. 2015 Dec;102:107-112. [PMC free article: PMC4684461] [PubMed: 26429773]
8. Juckett G, Trivedi R. Evaluation of chronic diarrhea. Am Fam Physician. 2011 Nov 15;84(10):1119-26. [PubMed: 22085666]
9. Hodges K, Gill R. Infectious diarrhea: Cellular and molecular mechanisms. Gut Microbes. 2010 Jan;1(1):4-21. [PMC free article: PMC3035144] [PubMed: 21327112]
10. Ford AC, Lacy BE, Talley NJ. Irritable Bowel Syndrome. N. Engl. J. Med. 2017 Jun 29;376(26):2566-2578. [PubMed: 28657875]
11. Jamshed N, Lee ZE, Olden KW. Diagnostic approach to chronic constipation in adults. Am Fam Physician. 2011 Aug 01;84(3):299-306. [PubMed: 21842777]
12. Alavi K, Chan S, Wise P, Kaiser AM, Sudan R, Bordeianou L. Fecal Incontinence: Etiology, Diagnosis, and Management. J. Gastrointest. Surg. 2015 Oct;19(10):1910-21. [PubMed: 26268955]
13. Wu GJ, Xu F, Lin L, Pasricha PJ, Chen JDZ. Anorectal manometry: Should it be performed in a seated position? Neurogastroenterol. Motil. 2017 May;29(5) [PubMed: 27910245]
14. Osborn NK, Ahlquist DA. Stool screening for colorectal cancer: molecular approaches. Gastroenterology. 2005 Jan;128(1):192-206. [PubMed: 15633136]
15. Rastogi A, Wani S. Colonoscopy. Gastrointest. Endosc. 2017 Jan;85(1):59-66. [PubMed: 27658908]
16. Thiruppathy K, Mason J, Akbari K, Raeburn A, Emmanuel A. Physiological study of the anorectal reflex in patients with functional anorectal and **defecation** disorders. J Dig Dis. 2017 Apr;18(4):222-228. [PubMed: 28261913]
17. Guaderrama NM, Liu J, Nager CW, Pretorius DH, Sheean G, Kassab G, Mittal RK. Evidence for the innervation of pelvic floor muscles by the pudendal nerve. Obstet Gynecol. 2005 Oct;106(4):774-81. [PubMed: 16199635]
18. Lacy BE, Patel NK. Rome Criteria and a Diagnostic Approach to Irritable Bowel Syndrome. J Clin Med. 2017 Oct 26;6(11) [PMC free article: PMC5704116] [PubMed: 29072609]
19. Goddard AF, James MW, McIntyre AS, Scott BB., British Society of Gastroenterology. Guidelines for the management of iron deficiency anaemia. Gut. 2011 Oct;60(10):1309-16. [PubMed: 21561874]
20. Hamilton W, Sharp D. Diagnosis of colorectal cancer in primary care: the evidence base for guidelines. Fam Pract. 2004 Feb;21(1):99-106. [PubMed: 14760054]
21. DiMagno EP, Go VL, Summerskill WH. Relations between pancreatic enzyme outputs and malabsorption in severe pancreatic insufficiency. N. Engl. J. Med. 1973 Apr 19;288(16):813-5. [PubMed: 4693931]

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