Introduction

Faecal incontinence (FI) is a paediatric gastroenterological problem with profound personal and family impacts. The affected children present with a history of voluntary and/or involuntary passage of stools into the underwear (*Rajindrajith et al.*, 2010).

The characteristic aroma of faeces in these children predisposes them to stigmatisation, rejection and bullying at school, which subsequently result in school avoidance and social withdrawal (*Joinson et al.*, 2006).

History of fecal incontinence:

Faecal incontinence was originally described in children who were neurologically handicapped. Subsequently, it had been observed in a significant percentage of otherwise healthy children (*Bellman*, 1966). Irrespective of the differences in underlying pathology (organic or functional), these children have significantly lower quality of life and most of the time suffer silently (*Bongers et al.*, 2009). Therefore, it is not surprising that they develop behavioural, emotional and upbringing problems, learning difficulties, depression and also frequently subjected to maltreatment (*Van et al.*, 2005).

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AIM OF THE WORK

The present study aims at:

- 1. Identification of diagnostic methods for fecal incontinence.
- 2. Identification of the therapeutic stepwise approach for patients with fecal incontinence.

Chapter 1:

PHYSIOLOGY OF DEFECATION

Physiology

The rectum ampulla (anatomically also: *ampulla recti*) temporarily stores fecal waste. As the waste fills the rectum and expands the rectal walls, nervous system stretch receptors in the rectal walls stimulate the desire to defecate. This urge to defecate arises from the reflex contraction of rectal muscles, relaxation of the internal anal sphincter, and an initial contraction of the skeletal muscle of the external anal sphincter. If the urge is not acted upon, the material in the rectum is often returned to the colon by reverse peristalsis, where more water is absorbed and the faeces is stored until the next mass peristaltic movement of the transverse and descending colon. If defecation is delayed for a prolonged period the fecal matter may harden, resulting in constipation. If defecation occurs too fast, before excess liquid is absorbed, diarrhea may occur. When the rectum is full, an increase in intra-rectal pressure forces apart the walls of the anal canal, allowing the fecal matter to enter the canal. The rectum shortens as material is forced into the anal canal and peristaltic waves push the feces out of the rectum. The internal and external anal sphincters along with the puborectalis muscle allow the feces to be passed by muscles pulling the anus up over the exiting feces. When defecating, the external sphincter muscles relax. The anal and urethral sphincter muscles are closely linked. Experiments by

Dr. Harrison Weed at the Ohio State University Medical Center have shown they can only be contracted together, not individually, and that both show relaxation during urination. This explains why defecation is frequently accompanied by urination (*Widmaier et al., 2006*).

Voluntary and involuntary control

• Defecation may be involuntary or under voluntary control. Young children learn voluntary control through the process of toilet training. Once trained, loss of control called fecal incontinence, may be caused by physical injury, nerve injury, prior surgeries, constipation, diarrhea, loss of storage capacity in the rectum, intense fright, inflammatory bowel disease (Widmaier et al., 2006).

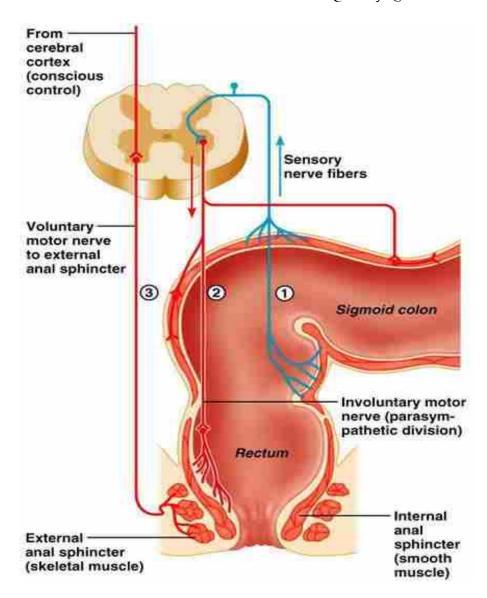


Figure (1): Mechanism of defecation.

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Chapter 2:

PATHOPHYSIOLOGY

Definition and Classification:

Faecal Incontinence (FI) denotes passage of stools into underwear in a child over the age of 4 years. This term replaces the previously used terms encopresis and faecal soiling. FI could be of functional or organic in origin. The common reasons for FI are listed in Table 1 (*Rajindrajith et al.*, 2013).

Table (1): Causes of faecal incontinence in children.

Functional causes	Organic causes
• Functional constipation associated faecal incontinence	Repaired anorectal malformations
• Functional non retentive faecal incontinence	Postsurgical Hirschsprung disease
	Spinal dysraphism
	Spinal cord trauma
	Spinal cord tumours
	Cerebral palsy
	• Myopathies affecting the pelvic floor and external anal sphincter

(Rajindrajith et al., 2013).

Fecal Incontinence leads to constant or episodic leaking of stools. Liquid stools in particular contain digestive enzymes that irritate and erode the skin, compromise integrity of the skin and affect the role of the skin as a protective barrier. Inddition, vigorousscrubbing, in attempts to remove soiling, strips away the protective horny layer of the epidermis. This impairs both its integrityand efficiency to function as a barrier. Epidermis of children is replaced fairly quickly (every 26 days) than that of adults (48days). Nevertheless, recurrent exposure to faecal matter can produce a vicious cycle of skin damage and inflammation, together with, loss of skin integrity (*Gray, 2004*).

More than 90% of encopresis is due to functional constipation where retained stool distends the rectum, resulting in stool leaking around a stool mass (Figure 2) (chonwald et al., 2004).

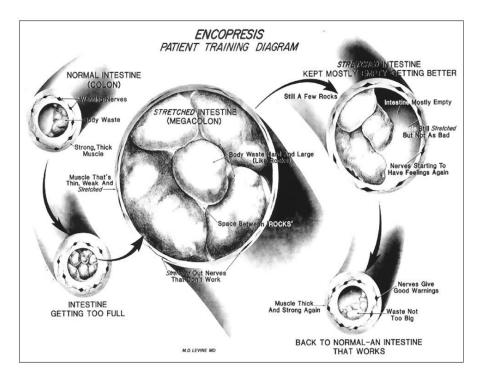


Figure (2): Process and effects of encopresis. Levine MD. Developmental and Behavioral Pediatrics, P. 419 3/e. © 2007.

1. Constipation-associated fecal incontinence -In most children with fecal incontinence, the problem is associated with, and probably caused by, underlying constipation (*Loening*, 2007). It is not clear why fecal incontinence develops in some children with constipation and not in others. Among children with constipation, there are no clear differences in pathophysiology or psychology between those with and without fecal incontinence. In both groups, constipation in children is frequently triggered by dietary changes, psychosocial stressors such as separation or schedule changes, and/or behavioral adaptations such as stool withholding. Functional constipation and associated fecal

incontinence is particularly common among children with attention deficit/hyperactivity disorder (ADHD) (Mckeown et al., 2013). And autism spectrum disorder (Peeters et al., 2013).

Three key milestones in a child's development have been recognized during which children may be at increased risk for the development of functional constipation and fecal incontinence (Di Lorenzo, 2011).

- The dietary switch to solid food
- Toilet training
- The start of school

A child who experiences a single painful or otherwise distressing bowel movement may decide to do everything possible to avoid passing stool. As a result, stool accumulates in the rectum and becomes hard and even more painful to pass. In some cases, the rectal enlargement causes loss of sensation and decreases the perception of the urge to defecate. With chronic rectal distention, the internal anal sphincter relaxes, allowing semisolid stool to leak onto the perianal skin and clothing. Thus, fecal incontinence frequently is the direct consequence of functional constipation and voluntary fecal retention (*Backer et al, 2006*).

The toilet training period is an especially important one for parents and the pediatrician to carefully monitor because of the potential for developing a chronic pattern of bowel and/or bladder problems Children who specifically avoid defecation may be at particular risk for developing fecal incontinence. In one series, 35 percent of children who refused toilet training developed chronic constipation and were at risk for primary fecal incontinence if they were not adequately treated (*Issenman et al., 2007*).

Incontinence of feces can occur both during the day and at night. Nocturnal incontinence is considered to be an indicator of severe accumulation of faeces in the rectum. Voluntary suppression of desire to pass stools leads to prolongation of total and segmental colonic transit times, aggravating faecal retention and leads to stool accumulation in the entire colon (megacolon). Furthermore, mechanical rectal distension is known to inhibit motor activities of right and left hemicolon and sigmoid colon by a reflex mechanism (*Bampton et al.*, 2010).

These intestinal reflexes further suppress defecation. Water is absorbed from the retained stools through rectal mucosa leading to hard stools. In addition, abnormally low chloride secretion due to abnormalities in non calcium mediated chloride channels in the rectal mucosa may further contribute to development of dry and hard fecal masses This process creates a vicious cycle of progressive accumulation of faeces and hardening of the faecal mass (*Bekkali et al.*, 2011).

Rectum and sigmoid colon gradually dilate causing megarectum and megacolon which lead in diminished propulsive

contractile forces of the rectal musculature. In addition, rectal sensitivity is blunted due to intrinsic rectal hyposensitivity or due to constant accumulation of faeces. Finally, semi-liquid faeces seeps between the faecal mass and rectal wall, and escapes through the anal canal when the sphincter muscles are relaxed. The volume of stools that leaks out is small and most of the time just stains the underwear (*Gladman et al.*, 2006).

2. Non retentive fecal incontinence - Fecal incontinence also can occur in the absence of constipation, in which case it is termed functional non retentive fecal incontinence. Because the definition of "non retentive" fecal incontinence is a diagnosis of exclusion, prevalence rates depend in large part on the definition used for fecal retention. According to several studies, up to 20 percent of children with fecal incontinence meet the criteria for functional non retentive fecal incontinence (Voskuijl et al., 2004).

In contrast, in one study in children older than the age of 5 presenting with fecal incontinence, only five percent of children met criteria for the non retentive form The disorder affects about 2 percent of children older than the age of 4, and is about four times more common in boys (*Loening-Baucke*, 2007).

Children with NRSI pass stools into inappropriate places without evidence of stool retention. The majority of them have complete evacuation of bowel, not just staining of the underwear as in retentive incontinence. The pathophysiology of NRSI is still far from clear. In patients with NRSI, total and segmental colonic transit times are within normal limits. Abnormalities in defecation dynamics, shown upon anorectal manometry, include inability to relax the external anal sphincter during defecation *(de Lorijn et al., 2004)*.

It is likely to be an acquired control mechanism in which after the loss of the first stool in the underwear, the child contracts the external anal sphincter to retain the rest of the stool. In contrast with retentive FI, the rectal compliance and sensitivity thresholds as measured by rectal barostat were normal in these children. In addition, barostat studies have also not revealed abnormalities in anorectal function in children with NRSI (Voskuijl et al., 2006).

Colonic transit, rectal compliance or sensitivity are responsible for NRSI. Psychosocial factors and deranged defecation dynamics may play a role in the pathogenesis (Voskuijl et al., 2007).

As for functional constipation, there is some association with behavioral and attention problems including attention deficit/hyperactivity disorder (ADHD) and autism spectrum disorder as well as with anxiety and depressive symptoms. Soiling episodes are generally linked to certain persons or situations (eg, visits to a divorced parent). However, up to 40 percent of children have never been toilet trained properly,

suggesting a long-standing physical or environmental cause (McKeown et al., 2013).

Risk factors for functional FI

Bio psycho social factors play a pivotal role in the onset and continuation of symptoms in children with FI (Figure 1). Two studies have identified low socioeconomic background as a risk factor for functional FI in children Inadequate toilet facilities and unclean or unhygienic toilets may be discouraging these children from using toilets, leading to stool withholding and retentive FI (*Rajindrajith et al.*, 2010).

Delay in seeking health care for defecation disorders, such as constipation, would also be a probable contributory factor for FI in such socioeconomic backgrounds. Other risk factors possibly contributing to retentive FI are living in urban areas and war affected zones. Hospitalisation of the child for another illness and bullying at school have also been suggested as risk factors for FI. Psychological and behavioural abnormalities (*Rajindrajith et al.*, 2010).

Like aggressive behavior, social withdrawal, anxiety, depression, behavior, and poor school and social performances were commonly noted in children with functional FI. Learning difficulties, upbringing problems and oppositional behaviour, were also noted to be higher in these children. Analysis of child behavior had shown that approximately one-third of children

with NRSI had psychological disturbances and behavioural problems (Cox et al., 2010).

Psychological stress is known to alter the output of the brain-gut axis in functional gastrointestinal diseases such as irritable bowel syndrome. Similarly, altered functions of braingut axis, triggered by psychological abnormalities, probably result in changes of anorectal functions in these children leading to FI (*Grover and Dorssman*, 2011).

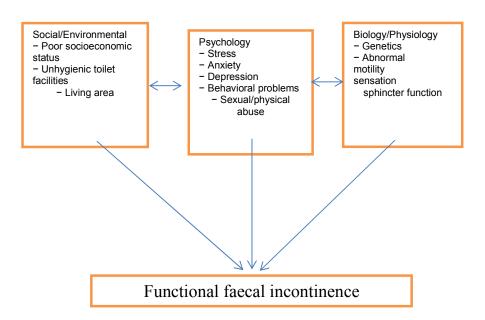


Figure (3): Biopsychosocial model of functional faecal incontinence. FI, faecal incontinence.

Chapter 3:

A- EPIDEMIOLOGY

It affects approximately 2.8 percent of 4-year-old children, 1.5 percent of 7- to 8-year-old children, and 1.6 percent of 10-to 11-year-old children. Another population-based study reported prevalence rates of 4 percent in children 4 to 17 years old (*Loening-Baucke*, 2011). In a study of 418 children with constipation, 68 percent of boys and 52 percent of girls presented with fecal incontinence (*van Ginkel et al.*, 2003).

Both epidemiological and hospital studies have shown FI in 75–90% of children with constipation A recent epidemiological survey has shown that 80% of children with FI are suffering from constipation (*Benninga et al.*, 2004).

Functional FI is either due to retentive (constipation associated) or functional non retentive FI. Epidemiological studies in the past have not attempted to differentiate these two entities. It is important to differentiate between retentive and non retentive FI as these two conditions different etiology and management. In a previous epidemiological survey we have shown that retentive FI (constipation associated FI is 4.5 times commoner than NRSI, underscoring the significance of constipation in the etiology of FI (*Bongers et al.*, 2009).