Grand Rounds

Fecal Incontinence:

A Clinical Approach

ZARA R. COOPER, B.S.[1], AND SUZANNE ROSE, M.D., M.S.ED.[2]

Abstract

"Fecal incontinence" is defined as the involuntary loss of stool at any time of life after toilet training. It is a socially and psychologically devastating condition for patients and their families, and a topic which both patients and physicians are reluctant to approach. Although the true prevalence of fecal incontinence is unknown, studies have reported it to be as high as 2.2% in the general population, with significantly higher rates among nursing home residents and hospitalized elderly. Risk factors include advancing age, female gender and multiparity.

An understanding of pelvic floor anatomy and physiology is required to appreciate how diverse medical conditions can affect mechanisms involved in normal continence. The rectum serves as a storage reservoir until elimination can take place at a socially acceptable time and place. The pelvic floor muscles help to regulate the defecatory process and maintain continence. These muscles include the internal anal sphincter, the external anal sphincter and the puborectalis muscle. Each muscle contributes to normal continence, although the relative importance of each is controversial. Neurologic integrity and sensation are also key factors.

Conditions associated with fecal incontinence include diarrheal states, fecal impaction, idiopathic neurologic injury, surgical and obstetric injury, pelvic trauma, collagen vascular disease, and neurologic impairment related to stroke, diabetes, or multiple sclerosis. Evaluation of the patient with fecal incontinence includes a directed history and physical examination, with particular attention paid to integrity of the perineum and rectum, and a complete neurologic evaluation. Diagnostic tools such as stool studies, anorectal manometry, defecography, electromyography, pudendal nerve conduction, and endoanal ultrasound may be employed in an outpatient setting. Fecal incontinence may be treated conservatively by employing such methods as dietary restriction, stool bulking agents, and biofeedback. Surgery may be the best option for cases refractory to medical treatment, or for those patients with rectocele or obstetrical injury.

In this article, we review the presentation, epidemiology, pathophysiology, and etiology of fecal incontinence. Evaluation, including key components of directed history and physical examination, and the appropriate use of diagnostic studies and indications for treatment options are also addressed. **Key Words:** Fecal incontinence, functional bowel disorder, anorectal disorders, anorectal physiology.

Introduction and Epidemiology

FECAL INCONTINENCE is defined as the involuntary loss of stool at any time of life after toilet training. It is a devastating condition for patients and their caretakers, and a topic which physicians and patients are reluctant to discuss. The exact prevalence of fecal incontinence in the population is unknown. Current data suggest that the occurrence may be much greater than is generally thought. One survey found that almost 14% of adult patients visiting their primary care physician had experienced fecal incontinence, and that 10.5% of those had restricted their activity due to the condition (1). It is notable that only one-fifth of the affected patients had discussed the topic with their physicians. A community-based study of Wisconsin residents found that 2.2% of the general population had anal incontinence of gas, liquid or feces (2). Of these subjects, 30% were older than 65 and 63% were women. The same study found that independent risk factors for incontinence included advancing age, female sex, poor general health and physical limitations. Other reports have estimated that 10–17% of nursing home residents (3, 4) and between 13 and 47% of hospitalized elderly (5, 6) have some incontinence. In fact, it is the second most common cause for institutionalizing the elderly (7).

[1]Mount Sinai School of Medicine Class of 2000 and [2]Associate Dean for Student Affairs and Medical Education, Mount Sinai School of Medicine, New York, NY.

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Address correspondence to Suzanne Rose, M.D., M.S.Ed., Associate Dean for Student Affairs and Medical Education, Mount Sinai School of Medicine, Box 1257, One East 100th Street, New York, NY 10029.

Double incontinence of urine and feces is 12 times more common in geriatric patients than fecal incontinence alone (4). Women of all ages are disproportionately affected by this condition. The incidence is eight times higher in 45-year-old women than in men of the same age (8). The costs of this disorder are unknown. However, the recent explosion of the adult diaper market is indicative of its economic importance.

Anatomy and Physiology of Normal Continence

An understanding of pelvic floor anatomy and physiology is key to appreciating the etiology and treatment of fecal incontinence and disorders of the pelvic floor (Table 1). The pelvic floor muscles help to regulate the defecatory process and maintain continence. The rectum serves as a storage reservoir until elimination can take place at a socially acceptable time and place. The muscles of the pelvic floor include the internal anal sphincter, the external anal sphincter and the puborectalis muscle (Fig. 1). Each muscle contributes to normal continence, although the relative importance of each is controversial.

The internal anal sphincter (IAS) is continuous with the smooth muscle layer of the rectum, and is under the control of the enteric nervous system. The IAS is tonically contracted and generates 80% of the anorectal resting pressure to prevent involuntary defecation (9, 10). The sphincter relaxes during defecation, in response to stretch by as little as 20–40 mL of rectal contents; this relaxation is caused by the rectoanal inhibitory reflex (RAIR) (11). This sphincter is under autonomic control by sympathetic innervation from the hypogastric plexus, which initiates tonic contraction, and by sacral parasympathetic fibers, which in turn mediate rectoanal muscle relaxation. Dysfunction of the internal anal sphincter muscle leads to a transient increase in intraluminal pressure, followed by a decrease in pressure. These dynamic changes are mediated by the rectoanal inhibitory reflex via parasympathetic nerves. In the absence of neuropathy, nerve endings in the anal canal can differentiate between solid, liquid and gas in the rectum via what is known as the sampling reflex.

TABLE 1
Factors Required for Normal Continence

Normal sphincter function
Neurologic integrity
Rectal sensation
Normal stool volume and consistency
Normal colonic transit time
Rectal storage capacity
Psychological motivation

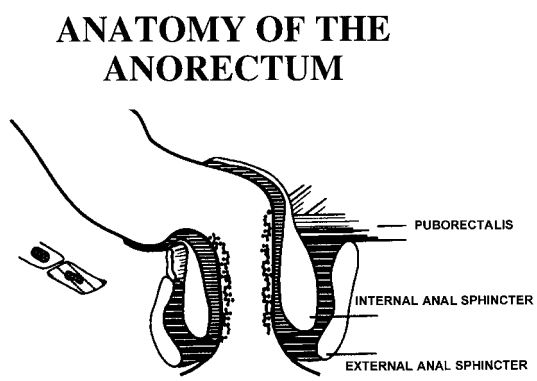

Fig. 1. Anatomy of the rectum.

The external anal sphincter (EAS) is a striated muscle under voluntary control, which contributes up to 20% of the resting pressure in the anal canal (9, 12). This muscle is innervated by the pudendal nerves (S2, S3, S4). Controversy exists as to whether the EAS is a single muscle or a triple-loop system consisting of deep, superficial and subcutaneous loops, each with its own innervation (13). It is partially contracted at rest, and undergoes tonic contraction approximately every fifteen minutes by reverse peristalsis (11). Inhibition of the EAS during defecation allows for the passage of stool. A spinal reflex stimulates contraction (14) of the EAS whenever external pressures increase, such as with erect posture and coughing (15, 16). It should be noted that external pressures decrease during sleep (17). Tactile stimulation of the perianal skin will elicit phasic contraction of the EAS, commonly referred to as the "anal wink," via the anocutaneous reflex.

The puborectalis muscle (PRM) seems to be most directly responsible for continence (11, 18). The PRM is also innervated by the pudendal nerve, and remains partially contracted at rest. It forms a U-shaped sling around the anorectal junction and connects to the bony pelvis. The PRM maintains a 92-degree angle (19) between the rectum and the anal canal, which obstructs the outlet and prevents the passage of solid stool. Voluntary contractions further narrow the anorectal angle. During normal defecation, relaxation of the PRM and descent of the pelvic floor widen this angle to approximately 135 degrees. Idiopathic fecal incontinence, spinal cord injuries and descending perineum syndrome are accompanied by a more obtuse angle. Other factors related to normal

continence include stool volume and consistency, stool transit time, neurologic integrity and rectal sensation, rectal storage capacity, and psychological motivation.

In normal subjects, the resting rectal pressures exceed pressures in the sigmoid colon, resulting in a retrograde pressure gradient which prevents the contents of the sigmoid from entering the rectum too quickly. Rectal incontinence may result from disruption of this pressure differential between the sigmoid and the rectum when colonic transit is so fast that the reservoir capacity of the rectum is overwhelmed. Anorectal pressures are determined by extrinsic pressures such as increased intra-abdominal pressure and intrinsic pressures generated by the bowel wall and muscles of the pelvic floor (2). A high-pressure zone is generated by the tonic contraction of the IAS, EAS, and PRM (20), preventing passage of stool from the sigmoid to the rectum and also preventing emptying of the rectum (21).

TABLE 2
Factors Associated with Fecal Incontinence

- Gender
- Age
- Abnormal rectal compliance
  - increased compliance
    - megarectum
    - fecal impaction
  - decreased compliance
    - inflammatory bowel disease (IBD)
    - radiation proctitis
    - coloanal anastomosis
- Diarrhea
- Sphincter insult
  - trauma, surgery
- Neuropathy
- Prolapse

Incontinence

Factors associated with fecal incontinence are listed in Table 2. Major incontinence is characterized by frequent and regular inability to control stool of any consistency. It is most often due to surgical and other trauma, or pelvic floor neuropathy (8) (Table 3). Incontinence is considered to be minor when the patient is unable to control passage of flatus or loose stool and this results in soiling of the undergarments. This is most often due to some surgical injury to the IAS or to overflow incontinence.

The rectum has both viscous and elastic properties that allow it to maintain low intraluminal pressure, even if the volume is large (22). Changes in compliance can affect continence. If compliance is decreased, smaller volumes of stool can cause increased pressure and impair rectal storage function. Incontinence accompanied by urgency and frequency may result. Conditions that may decrease compliance include inflammatory disease (23), radiation injury (24), and coloanal anastomosis. An anatomic obstruction, such as may occur with a fecal impaction, may result in overflow incontinence whenever liquid stool circumvents the offending obstruction. This situation may be associated with increased compliance.

In rare instances, profuse diarrhea can cause incontinence even in the presence of normal pelvic floor and sphincter mechanisms. Rapid colonic transit time and increased volume can overwhelm rectal storage capacity. Furthermore, diarrhea will stress normal continence mechanisms since it is more difficult to perceive and retain liquid stool than solid.

Impaired proprioception in the levators, puborectalis, and sphincters will decrease a patient's ability to sense rectal filling and cause loss of the normal warning that defecation is imminent (25). In mentally ill or geriatric patients, megarectum resulting in fecal impaction is often associated with decreased sensation. Intact autonomic pathways will still cause the IAS to relax in response to a fecal bolus. Consequently, the IAS will relax before the patient perceives rectal stretch (7). The result is fecal impaction and overflow incontinence (26), of which the patient may be unaware (11).

TABLE 3
Anorectal Abnormalities in Fecal Incontinence

Patient Categories	Reservoir	Sphincters	PRM	Sensation
Anal trauma	N	A	N	N
Idiopathic	N	A	A	N
Diabetes	N	A	?	A
Proctitis	A	N	N	N
Neurogenic	N	A	A	A

N = normal
A = abnormal

The effort to delay defecation initially requires powerful initiation of contraction of the EAS, which increases pressure within the anal canal. To prolong the delay, contraction must be sustained. One limited study by Read and Read (27) found the mean duration of contraction to be 3.2 minutes; others report mean time of 1–3 minutes for the EAS to fatigue completely (28). The inability to sustain a contraction (the fatigue phenomenon) is often more disabling to patients with incontinence than the inability to generate a peak effort. Marcello et al. (29) developed the fatigue rate index (FRI) to evaluate the fatigue rate of the EAS. They found that the FRI was approximately 2 minutes longer in controls than in incontinent subjects.

Anal trauma or surgery may cause incontinence, due to myopathic or neuropathic sphincter injury. Neurologic impairment caused by bilateral sacral denervation is also associated with diabetic autonomic neuropathy and neurologic disorders such as multiple sclerosis and meningomyelocele. Insult to the sphincter of the EAS and IAS does not necessarily cause incontinence. However, decreased IAS tone can impair resting pressures and lower the threshold for the sampling reflex which distinguishes solid from liquid and gas (30).

Idiopathic denervation of the PRM or EAS can also impair the sampling reflex, leading to sensory incontinence. The patient may know that he or she is passing contents, but be unable to prevent an accident. Idiopathic incontinence is most common in multiparous middle-aged or elderly women, and may be due to traction injuries (14) secondary to vaginal delivery (30) or excessive pelvic floor descent during defecation over a long period of time (31). Many patients have perineal descent and widening of the anorectal angle consistent with weakening of the striated muscles of the pelvic floor. It has been suggested that women are more susceptible to this type of injury because the morphology of the pelvic floor muscles may be hormone-dependent (32).

Age is also a known risk factor for fecal incontinence. Endosonography and histopathologic studies have demonstrated that age is related to sclerosis of the IAS (33, 34). Additionally, increased fiber density has been found in the EAS and pelvic floor muscles in study subjects older than 60 years of age (35). Degeneration of the sphincter causes a progressive decline in resting pressures and decreased sensation (36).

In the presence of a rectocele, the detached rectal mucosa prolapses during straining, which may cause excretion of mucous discharge from the mucosa for one to two hours following defecation (19). Incontinence of solid stool may also be seen. Procidentia is related to low rectal pressures and an obtuse anorectal angle, which can contribute to fecal incontinence. Most important, it appears that rectocele-induced straining at defecation causes a traction injury, which is most closely related to subsequent incontinence (14). It has been reported that patients with rectocele, but without evidence of neuropathy, remain continent (35). Additionally, after rectocele repair, patients with concomitant sphincter denervation remain incontinent (4).

Etiology

Despite similarities in the pathogenesis of fecal incontinence, the etiology of this disorder is highly variable (Table 4). As mentioned above, profuse diarrhea challenges normal continence secondary to an overwhelming increase in volume and impairment of rectal sensation. This may be associated with inflammatory bowel disease, radiation enteritis, laxative abuse, short gut syndromes, and acute diarrheal illness. However, it is most often seen with diabetes.

It has been estimated that 20% of diabetic patients experience fecal incontinence. Most have

TABLE 4
Conditions Associated with Fecal Incontinence

Diarrheal states
inflammatory bowel disease (IBD)
irritable bowel syndrome (IBS)
infectious diarrhea
laxative abuse
malabsorption
Anatomic defects
trauma
perineal/anorectal surgery
obstetric injury
Neurologic impairment
diabetes
multiple sclerosis
cauda equina syndrome
stroke
neoplasms
Overflow incontinence
fecal impaction
psychotropic drugs
antimotility agents
Collagen vascular disease
systemic sclerosis
Congenital abnormalities
myelomeningocele
spina bifida
Pelvic floor denervation
idiopathic
descending perineum syndrome
Aging

diarrhea and multiple neuropathies as well (37). The causes of diarrhea in diabetics with incontinence include autonomic dysfunction, bacterial overgrowth, ingestion of hexitols with osmotic diarrhea, and pancreatic insufficiency. Fecal incontinence may occur with the onset of diabetic diarrhea or it may follow years after the onset of diabetes has been documented. Nocturnal incontinence without warning is characteristic in these patients. Diarrhea in incontinent diabetics is rarely of high volume, but several studies indicate that one or more abnormalities of incontinence are present in the vast majority of these patients (38, 39). It is notable that unlike other causes of incontinence, fecal incontinence due to diabetes has a male predominance.

Patients with systemic sclerosis, or scleroderma, may experience gastrointestinal motility disturbances accompanied by symptoms of reflux, dysphagia, abdominal pain and defecatory difficulty. Incontinence receives less attention because it often goes unreported. Jaffin and colleagues (40) found that a tendency toward liquid and semi-formed stool and low resting pressure in the IAS in study subjects were likely contributors to incontinence in these patients. They also found that the RAIR was abnormal in 80% of subjects, but was normal in 70% of incontinent controls without systemic sclerosis. Because scleroderma affects smooth muscle more than striated muscles, EAS pressures in study subjects were normal or high when compared with incontinent controls without scleroderma.

Regrettably, many cases of fecal incontinence are iatrogenic in nature. Anal fistula repair requires sphincter division and is the most common operative cause of fecal incontinence (41). Additional procedural causes of incontinence include sphincterotomy, therapeutic anal dilation, and hemorroidectomy. The last has been associated with decreased intra-anal pressures (42, 43) and can also require removal of the anal cushions, with a subsequent decrease in rectal sensation (43). Prostatectomy has been associated with fecal incontinence in men. In one survey, Bishoff et al. (44) found the incidence of fecal incontinence to be 5% and 18% after radical perineal and retropubic prostatectomy, respectively. Fractures of the bony pelvis, which divide the PRM, may also be associated with incontinence.

Obstetric and birth trauma have long been recognized as significant factors in the etiology of fecal incontinence. Forceps, prolonged second stage (30), multiparity (30), and delivery of a large birth-weight infant (45) have all been associated with an increased risk of secondary incontinence.

There appear to be two major causes of incontinence related to childbirth: traumatic disruption of the sphincter muscles with neuropathy, resulting in early fecal incontinence, and isolated pudendal neuropathy, causing a late manifestation. Sultan et al. (46) reported that vaginal delivery is frequently associated with mechanical disruption of both the internal and external sphincters. This series found that 13% of primiparous and 23% of multiparous women who delivered vaginally had anal incontinence or fecal urgency when studied six weeks after delivery (46). Approximately 0.7% of vaginal deliveries with posterolateral episiotomies are associated with a third or fourth degree tear. These lacerations can cause sphincter and nerve injuries resulting in decreased resting and squeeze pressures. The internal anal sphincter appears to be injured more often and can be damaged even when the perineum remains intact. Episiotomies are usually repaired shortly after delivery, but inadequate primary repair with persistent incontinence may require secondary sphincter repair (3).

Stretching of the pudendal nerves during childbirth leads to progressive denervation of the anal sphincter muscles with resultant incontinence years later. Pudendal neuropathy causes a decrease in anal pressures, delayed conduction and decreased anal sensation (47). Idiopathic fecal incontinence in middle-aged women may be associated with prolonged pudendal latencies, which may be a late manifestation of childbirth injury.

It is noteworthy that the majority of patients with idiopathic fecal incontinence are women. Usually these are multiparous women with a history of vaginal delivery, high birth-weight infants, prolonged labor and/or forceps delivery. A history of a third or fourth degree perineal tear is not uncommon. As in incontinence related to obstetric injury, it is believed that pudendal nerve damage at delivery causes future dysfunction. In some reports, electromyograph (EMG) and histopathology studies have demonstrated denervation of the PRM and EAS muscles in up to 80% of patients with fecal incontinence (7).

Overflow incontinence is most commonly seen in elderly and institutionalized patients. Anatomical or functional outlet obstruction causes a leakage around the mass. The problem is often exacerbated by laxative abuse, which results in liquid stool seeping around the fecal bolus. Large neoplasms, such as lymphogranuloma, may also cause functional obstruction similar to that seen with constipation or encopresis in childhood.

Clearly, any neurologic deficit affecting the pelvic musculature can lead to incontinence. Neurogenic causes include multiple sclerosis, spinal cord injury, cauda equina syndrome of any etiology,

and diabetes, as mentioned above. Lesions in the frontal cortex, usually associated with stroke, can also impair pelvic muscle function.

Descending perineum syndrome, a condition closely linked to fecal incontinence (48), may develop in patients with a long history of straining at defecation. This syndrome presents with chronic perineal aching and a vague sensation of a lump in the anal canal. When the patient strains to defecate, the perineum descends below the ischial tuberosity. This is probably secondary to stretch of the pudendal nerve during chronic straining.

As mentioned above, surgical insult or trauma can also lead to incontinence. It is important to recognize that causes of trauma include sexual abuse and anal intercourse. In working with patients with fecal incontinence, the physician must be prepared to approach these matters in a sensitive and nonjudgmental manner.

TABLE 5
Evaluation of Fecal Incontinence

History
Stool patterns
frequency, duration
pattern of soiling
diurnal or nocturnal
urgency
diarrhea, constipation, straining
Associated symptoms or medical conditions
Physical examination
Rectal examination
perineal inspection
anocutaneous reflex
digital exam to assess resting tone or squeeze pressure
PRM testing
Neurologic examination
mental status
sacral reflexes
perineal sensation

Diagnosis and Evaluation

Because fecal incontinence is a socially devastating condition, patients are often reluctant to discuss their experiences. Signs and symptoms need to be elicited directly. Patients should be asked to characterize their bowel movements, including the frequency, duration and pattern of incontinence. Inquiry should be made about daytime and nocturnal soiling and associated symptoms, such as urgency, lack of warning, diarrhea, constipation and straining.

History and physical examination should be focused to identify potential causes and conditions that may exacerbate fecal incontinence (Table 5). Pseudoincontinence, or perineal soiling caused by prolapsed hemorrhoids, perineal fistula, discharge from sexually transmitted disease and pruritus ani should also be excluded by history and physical examination. A complete history will note previous anorectal surgery or trauma, back injury, a history of functional bowel or inflammatory bowel disorders, diabetes, scleroderma, multiple sclerosis, stroke or other neurologic conditions. The physician should elicit a history of laxative abuse or other medication use. A dietary history to eliminate lactose, fructose, and sorbitol intolerance may be useful. An obstetric history, including number of vaginal deliveries, prolonged labor, forceps deliveries, episiotomies, and birth weights must be included, if applicable. A mental status examination and psychosocial testing may be useful, especially with the elderly and with children, where impaired motivation for continence may be a factor. Any history of sexual abuse or anal intercourse should be approached in a sensitive and nonjudgmental manner.

The physical examination should begin with inspection of the undergarments for soiling. It should also include careful inspection of the perianal area for any scars and obvious lacerations, fissures, or hemorrhoids. Prolapse or a gaping anus is often accompanied by urinary incontinence as well. Integrity of the sacral reflex arc can be assessed via the anocutaneous reflex, or "anal wink." This maneuver involves lightly scratching the perianal skin to elicit EAS contraction. The reflex may be absent in diabetics and in those with spinal cord injuries. Patients should also be assessed for gait and lower extremity sensory and motor function, to rule out cauda equina syndrome. The digital examination is useful in identifying a rectal mass or fecal impaction. One may also assess resting tone and strength of the PRM and EAS contraction by sensing the degree of contraction against the examining finger. The PRM is usually palpable posteriorly. The examination can be completed with anoscopy and flexible sigmoidoscopy. The rectum should be examined for the presence of ulceration or inflammation. Biopsy may be necessary to identify mucosal inflammation, neoplasm or ischemic changes. Evaluation of patients with diarrhea should include stool studies. These may include quantitative 24–72 hour stool samples for volume with measurement of stool electrolytes, osmolality, and fat to determine if high-volume diarrhea is osmotic or secretory in nature (Table 6). Culture and microscopic examination for ova and parasites also may be indicated.

TABLE 6
Diagnostic Studies for Fecal Incontinence

Sigmoidoscopy
Stool studies if diarrhea is present
Anorectal manometry
test done with balloon catheters to test muscle function
resting pressures
squeeze pressures
sensation
compliance
sphincter response to distention
Proctography and defecography
defines anatomy and change of pelvic floor muscle position with defecation
Anorectal electromyography
examines muscle fibers to define muscular disorder
Pudendal nerve conduction
tests how long it takes the "message" to get from nerve to muscle
Endoanal ultrasound
assesses the integrity of the internal and external anal sphincters

Anal manometry is done with balloon catheters, to test the function of the sphincter muscles. Specifically, it can measure the resting tone of the IAS, squeeze pressures of the EAS and functional length of the high-pressure zone. In general, incontinent patients have lower squeeze and resting pressures than do controls (49, 50) (Fig. 2). Additional manometry studies can be used to assess the rectoanal inhibitory reflex, sensitivity, compliance and capacity (32). Manometry results correlate well with EMG studies used to assess the EAS.

EMG of the EAS can be performed with needles or surface electrodes. The sphincter can be examined in a circumferential pattern, by quadrant, or by the anterior/posterior positions. EMG can also be used to evaluate the PRM, by inserting a needle above the natal cleft. Denervation, conduction defects, and striated muscle function can all be evaluated with this study.

Pudendal nerve assessment (pudendal nerve terminal motor latency [PNTML]) tests how long it takes the "message" to get from the stimulated nerve to the muscle. Conduction of the pudendal nerve can be performed by using a St. Mark's electrode to stimulate the right and/or left pudendal nerves at the ischial tuberosities and then measuring the time to detect a muscle response. Such provocation of the pudendal nerve is most useful for assessment of neurogenic incontinence, obstetric injury and rectal prolapse. Patients with rectal prolapse and women with obstetric injury to the EAS may have prolonged PNTML (30).

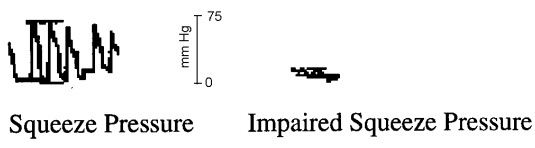

Fig. 2. Anal manometry. Pressure in millimeters of mercury. Normal is illustrated on the left.

Defecography, or the dynamic proctogram, defines the anatomy and change of the pelvic floor muscle position with defecation. It can identify abnormalities such as prolapse, perineal descent and intussusception. It can also assess PRM function. This procedure utilizes a paste of barium and potato powder to simulate fecal material. Video recording of strain, squeeze and defecation into a radiolucent commode allows real-time assessment of anatomic changes during defecation (51).

Endoanal ultrasound can identify structural defects of either the IAS or the EAS (52) (Fig. 3). This procedure is relatively noninvasive and is not time-consuming. Endosonographically, the sphincters appear distinctly as circular bands. Other pelvic floor structures, including the puborectalis muscle, the urethral sphincter, vagina and outlines of the bony pelvis and ischiorectal fossa, may be seen.

Treatment

The first step in the treatment of fecal incontinence is to address as many modifiable factors as possible. Dietary intolerance for sorbitol, lactose and fructose, should be considered, and substances should be eliminated as necessary, as well as increasing dietary fiber and adding bulking agents to solidify stool and enhance sphincter function. Conversely, patients with neurogenic

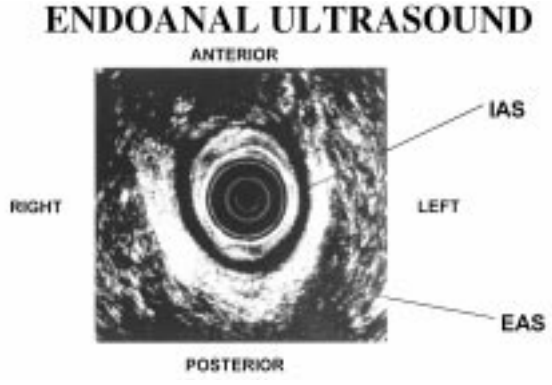

Fig. 3. Endoanal ultrasound. Note: Disruption of anterior external anal sphincter (EAS) and internal anal sphincter (IAS).

incontinence may benefit from a fiber-deficient diet, to decrease the volume of stool. Antacids containing magnesium should also be avoided, as they can cause diarrhea.

Opiate derivatives such as loperamide or diphenoxylate with atropine may decrease stool volume and frequency, improve stool consistency, or perhaps directly affect sphincter muscles. Some studies have shown that loperamide may be the more effective agent in improving anal pressures (53, 54) and may be the drug of choice when combined with a low-fiber diet to treat neurogenic incontinence.

In some cases, phosphate and warm tap-water enemas as needed can keep the rectum empty, and assist in regularly planned bowel movements. Constipation and overflow incontinence can be treated with hypertonic enemas, mineral oil enemas, and even manual disimpaction if necessary. In elderly or immobilized patients, enemas should be given once or twice daily until there are no more feces left in the colon or rectal vault. Follow-up should include enemas given at regular intervals, accompanied by a high fiber diet and generous fluid intake, to avoid recurrence. Psychotherapy may be indicated for those patients lacking motivation.

Biofeedback, an operant conditioning technique, has a response rate comparable to that of surgical treatments (55–57). It requires a motivated patient who can understand the process. In addition, the patient must have some rectal sensation and voluntary control of the EAS in order to generate a squeeze pressure. Biofeedback can be done by employing a manometer or anal EMG plug. Once the intra-anal sensor is in place, the response is recorded for the patient. A patient can look at tracings of muscle strength and work toward enhancing his or her response. The rectal balloon is inflated with volumes of air and the patient learns to recognize this as rectal distention (sensory discrimination). The smallest volume of distention sensed by the patient is determined and the threshold is then lowered progressively until the response becomes automatic and no further improvement occurs.

Patients also can be taught to increase EAS sphincter pressure, in response to rectal distention via visual feedback from manometric recordings, by practicing contracting the EAS. This technique can be used when rectal distention or urgency is sensed. Most adult patients respond after just one session.

The large number of surgical procedures employed for fecal incontinence suggests that no single technique is best in all cases. Sphincter repair is usually reserved for isolated defects and for cases where medical therapy has failed. Surgery is often considered as first-line therapy in rectal prolapse, as resuspension restores continence in two-thirds of the patients (58). Obstetrical injuries, trauma and acute disruption of the sphincter are usually managed by primary repair of the defect. Results are usually better with solid rather than liquid stool (14).

There are a number of surgical techniques available to address fecal incontinence that has been unresponsive to medical therapy. Direct apposition requires mobilization of the EAS, resection of scar tissue, muscle division, and an end-to-end repair. The procedure has a 33.5–77.5% success rate (59). Postanal repair has been useful for neurogenic and idiopathic incontinence. The levator ani, PRM and EAS are plicated posteriorly to restore the anorectal angle and tighten the anal canal. Anterior plication is the preferred approach, with a satisfactory outcome in 62% of patients with idiopathic incontinence and 71% with a traumatic etiology (60). Generally, surgery is less likely to be successful if neuropathy is present prior to repair.

Overlapping anterior sphincteroplasty can be employed to treat childbirth injuries. This procedure involves identification of healthy tissue on either side of the defect, division of scar tissue and overlapping repair of the sphincter. Normal PNTML is predictive of a good outcome with sphincterotomy (61).

Anal encirclement uses a silver wire or, more recently, a Silastic ring or rod to mechanically tighten the anus, creating a barrier to the passage of feces. These procedures are often unsatisfactory, with frequent complications including impaction, infection and erosion of the encircling material. Implantable artificial sphincters are also available.

Skeletal muscle transpositions using the gluteus maximus, gracilis or palmaris longus muscles have been performed for fecal incontinence. In graciloplasty, first described in 1952, the gracilis muscle is mobilized to preserve the proximal attachments and maintain the neurovascular supply. An implantable nerve stimulator may be utilized to achieve sustained contraction (62).

Colostomy is reserved for failures of medical, biofeedback, and surgical techniques. Colostomy has been advocated for immobile patients who suffer from severe skin breakdown and complications including sepsis related to fecal incontinence.

Conclusion

Fecal incontinence is a devastating, but potentially treatable, condition. It is a significant prob-

lem affecting up to 20% of hospitalized patients and more than 2% of the general community. This condition primarily affects women, and is often associated with sphincter disruption and/or nerve injury after vaginal delivery. It is also associated in older patients with such common conditions as diabetes and stroke. It is important to understand the pathophysiology of incontinence and to recognize specific abnormalities, using objective tests of anorectal function described here. Evaluation begins with sensitive history-taking and a physical examination; even the most conservative treatments may have a significant impact on a patient's quality of life.

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