GI

TO HAVE AND TO HOLD:

PROBLEMS WITH FECAL CONTINENCE IN ADULTS

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"Maximilian Spielman, the great Mathematical Psycho-Proctologist ... founded the sciences of analogical proctoscopy and psychosymbolistic cosmography, developed the Rectimetric Index for 'distinguishing, arithmetically and forever, the sheep from the goats,' and explored the faint initial insights of what was to become Spielman's Law .... In three words Max Spielman ... showed the 'sphincter's riddle' and the mystery of the University to be the same. ONTOGENY RECAPITULATES COSMOGENY - what is it but to say that proctoscopy repeats hagiography?"

Giles Goat-Boy

JOHN BARTH, 1966

INTRODUCTION

Most adults can take fecal continence for granted. Those who cannot often lead tortured lives, characterized by anxiety and fear and punctuated by episodes of embarassment and shame. For fear of an episode of incontinence in a public place, patients often become reclusive, venturing out of their homes only when absolutely necessary. The social and psychological toll taken by incontinence can be staggering. The case history that follows illustrates some of the problems faced by incontinent individuals:

> A 31-year fold female was admitted to the hospital for evaluation of chronic diarrhea. Six years before admission, she developed abdominal pain and intermittent diarrhea (defined as the passage of two or more unformed stools daily). Two years later, she underwent an exploratory laparotomy because of persistence of abdominal pain. A cholecystectomy was performed showing chronic cholecystitis, and a benign cystic mass of calcified lymphoid tissue was excised from the head of the pancreas. After surgery, the pain disappeared, but she continued to have diarrhea. The diarrhea became more severe over the ensuing years with a stool frequency as high as 12 per day. No blood or mucus was ever noted in her stool, and her body weight was stable for the 12 months preceding admission. Diarrhea was most pronounced after breakfast but also occurred after other meals. No specific foods could be implicated. Treatments with opiates, anticholinergics and tranquilizers were unsuccessful in eliminating the diarrhea.
>
> Upon further questioning, it was apparent that the problems which most affected her life were fecal urgency and daily episodes (1-4 each day) of fecal incontinence, rather than the diarrhea itself. Fecal incontinence first became a problem about 1 year before admission when the diarrhea became more severe and more explosive in onset. She frequently soiled herself while rushing to get to the toilet or while sleeping, and carried spare clothing when away from home. She had quit her job as a cashier and restricted her travels and meals outside her home because of these problems.

In addition to restricting activities outside the home, incontinent individuals can suffer severe disruptions of their family lives. One patient of mine attributes his divorce to the strain placed on his marital relationship by constant nocturnal incontinence. The psychological toll of incontinence can also be staggering. Inability to control this basic biologic function leads to despair, hopelessness, and depression which colors all of one's life and one's relationships. There is no doubt that fecal incontinence is one of the most disabiling chronic gastrointestinal symptoms affecting individuals.

Incontinence is a problem that affects society too. The care of institutionalized, incontinent patients costs an estimated $8 billion in the United States each year (Ehrman, 1983). Fecal incontinence is often the major factor in the decision to institutionalize an elderly family member rather than to care for the relative at home. Moreover, once institutionalized, patients with incontinence require two to three times the amount of nursing care as other nursing home patients with comparable disabilities. If practical methods could be developed and applied widely to treat fecal incontinence, the cost savings might be dramatic.

PREVALENCE OF FECAL INCONTINENCE

Almost nothing is known about the prevalence of fecal incontinence in the general population in the United States. Although nearly 5% of subjects in two recent general surveys had chronic diarrhea, the number with coexisting incontinence was not defined (Drossman et al, 1982; Thompson, 1980). In England, the overall prevalence of fecal incontinence in the general population has been estimated as high as 4.3 per 1000 with higher rates among older individuals as shown in Table 1 (Thomas et al, 1984).

TABLE 1. ESTIMATED COMMUNITY PREVALENCE OF FECAL OR FECAL/URINARY INCONTINENCE*

SEX:	AGE:	PREVALENCE PER 1000
Male	15-64	4.2
Male	65+	10.9
Female	15-64	1.7
Female	65+	13.3
Total Sample		4.3

* From Thomas et al, 1984.

While fecal incontinence is relatively rare in the general population, the prevalence of incontinence in some populations is quite high. For instance, elderly institutionalized patients have a high frequency of incontinence usually both of feces and urine. One survey showed that 40% of women in geriatric units in one psychiatric hospital were incontinent of stool at least once during three weeks of observation (McLaren et al, 1981). In another study, 53% of patients in a geriatric unit were incontinent at least once during a week of observation, and 14% had more than 3 episodes (Smith, 1983).

In the second study, the patients with more frequent episodes were "all demented, very demanding or both, and were therefore unpopular with the staff" (Smith, 1983). This observation raises an important issue when discussing the prevalence of fecal incontinence, that is, that incontinence may be the result of physical barriers that are placed in the way of defecation in an appropriate place, not just a loss of the ability to control defecation (Millard, 1981). An "accident" may result from an unanswered call to the nurse for assistance as well as resulting from a true problem with the patient's continence mechanisms. Alternatively, a mobile patient with a continence problem may not have any episodes of incontinence in spite of severe urgency while under observation in a hospital room only a few steps away from a toilet. Thus, the presence or absence of incontinence must be judged against the opportunity for it to occur in a certain situation. This is particularly important in studies of therapy for incontinence.

Another group which appears to be at great risk for fecal incontinence is composed of patients with chronic diarrhea (Read et al, 1979). Liquid stools put a great stress on the continence mechanisms which may bring out latent problems with the neuromuscular machinery regulating continence. Patients with diarrhea and incontinence may view the incontinence as a measure of severity of the diarrhea and may not report it to physicians involved in their care. Without being asked directly, patients do not often volunteer a history of incontinence (Read, et al, 1979; Leigh and Turnberg, 1982). Whenever a patient is seen with a complaint of diarrhea, the physician should find out whether the patient has incontinence as well.

This is particularly true in diabetics with chronic diarrhea who have a high prevalence of co-existing fecal incontinence (Feldman and Schiller, 1983; Schiller, et al, 1982). As shown in Table 2, one-fifth of unselected diabetic outpatients interviewed while awaiting their doctors' appointments at Parkland or VA Diabetic Clinics had a history of fecal incontinence. A few of these diabetic patients were incontinent of solid as well as liquid stools.

TABLE 2.GASTROINTESTINAL SYMPTOMS IN 136 DIABETIC OUTPATIENTS AT PARKLAND MEMORIAL HOSPITAL AND THE DALLAS VA MEDICAL CENTER*

SYMPTOM:	N (%):
Constipation	82 (60)
Abdominal Pain	46 (34)
Nausea and Vomiting	39 (29)
Dysphagia	37 (27)
Diarrhea	30 (22)
Fecal Incontinence	27 (20)
No Gastrointestinal Symptoms	32 (24)

* From Feldman and Schiller, 1983

Some other medical problems that are thought to be causally related to the occurence of fecal incontinence are shown in Table 3. Occasionally one of these conditions present with fecal incontinence. More often incontinence complicates the course of these problems. For most of these conditions, the prevalence of incontinence is not known accurately. For some, the association is speculative and not supported by objective data. The role of some of these conditions in the pathogenesis of incontinence will be discussed later.

TABLE 3. SOME CONDITIONS ALLEGED TO BE CAUSALLY RELATED TO FECAL INCONTINENCE

NEUROLOGICAL PROBLEMS
- Dementia, Stroke, Sedation
- Spinal Cord/Cauda Equina Compression
- Tabes Dorsalis
- Multiple Sclerosis
- Peripheral Neuropathy
- Autonomic Neuropathy
  - Shy-Drager Syndrome
  - Diabetes Mellitus
- ? Idiopathic Incontinence

PELVIC PROBLEMS
- Trauma
  - Injury
  - Surgery (Including Hemorrhoidectomy)
  - Childbirth
- Perineal Descent
- Rectal Ischemia

MISCELLANEOUS
- Idiopathic Hypoparathyroidism
- Acute Myocardial Infarction

PHYSIOLOGY OF CONTINENCE AND DEFECATION

More than other gastrointestinal functions the maintenance of continence depends in a critical way on the anatomy of the pelvic region (Phillips and Edwards, 1965; Tagart, 1966; Schuster, 1968; Scharli and Kiesewetter, 1970; Duthie, 1975; Schuster, 1975; Dickinson, 1978; Duthie, 1982; Wunderlich and Parks, 1982). The arrangement of the rectum, anal canal and pelvic musculature presents a series of barriers to the passage of stool which work together to prevent incontinence. These barriers are reversible and can be removed to allow for defecation. To understand the physiology of continence one must understand the anatomical arrangements that produce these barriers and the reflexes and learned responses that reinforce these barriers when stressed and remove them when defecation is appropriate.

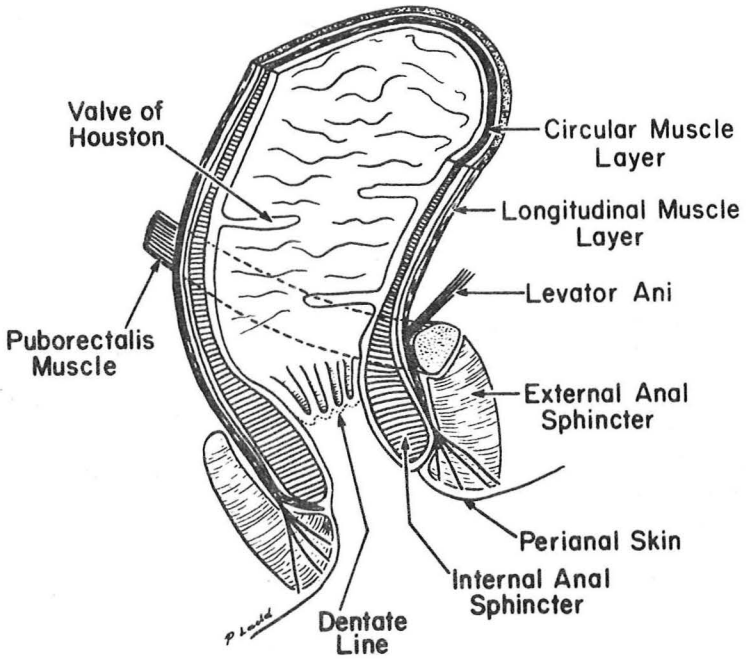

FIGURE 1. Anatomy of the Recto-anal Region.

Functional Anatomy. Like the rest of the intestine, the colon is composed of five major layers: the mucosa, submucosa, circular muscle, longitudinal muscle and serosa. Throughout most of the colon the longitudinal muscle is arranged in three discrete bands, the taenia coli. As the sigmoid colon sweeps into the pelvis to become the rectum, the longitudinal muscle layer becomes continuous around the circumference of the rectum. The rectal mucosa is thrown up into a series of transverse bands, the valves of Houston (Figure 1). At the

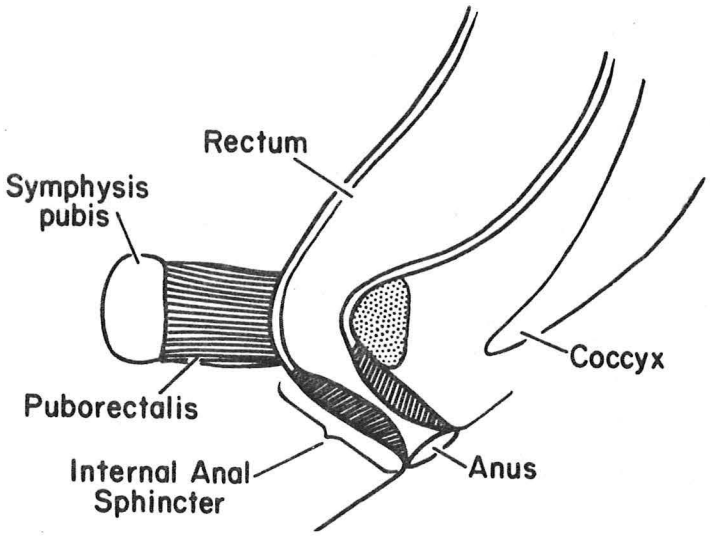

FIGURE 2. Side View of Rectum and Internal Anal Sphincter.

distal end of the rectum, the circular muscle layer becomes 2-3 times thicker than usual forming the internal anal sphincter (Figure 2). The rectum inserts into the anal canal and the columnar epithelium of the colon joins the squamous epithelium of the anal canal at the dentate line (Figure 1).

The external anal sphincter, a voluntary striated muscle surrounds the internal sphincter and extends distally to the subcutaneous tissue surrounding the anus (Figure 3). It is composed of both "red" and "white" fibers and shares the electromechanical properties of antigravity muscles elsewhere in the body (Fay et al, 1976).

As the distal rectum passes inferiorly through the pelvis on it way to join the anal canal, it passes through a muscular diaphragm, the pelvic floor. This

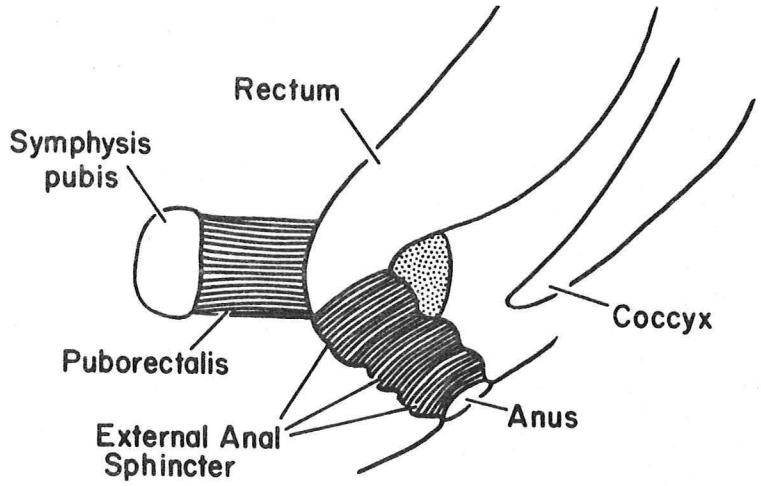

FIGURE 3. Relationship of External Anal Sphincter and Rectum.

striated muscle forms a continuous sheet dividing the abdominal cavity from the perineum. It is pierced by orifices for the rectum, urinary bladder and vagina and surrounds each of these organs (Figure 4). The pelvic floor is composed of the same skeletal muscle fiber types as the external anal sphincter and shares similar physiological properties (Vereecken et al, 1982). Named components of the pelvic diaphragm include the levator ani, pubococcygeus, iliococcygeus and the puborectalis muscle (Shafik, 1975).

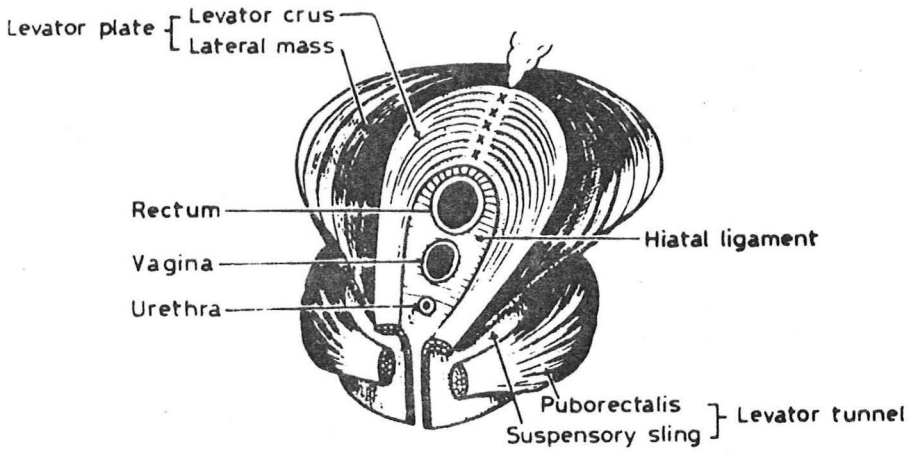

FIGURE 4. Relationship of Pelvic Floor (Levator Ani Muscle) and Pelvic Viscera (From Shafik, 1975).

The puborectalis muscle consists of a group of fibers in the pelvic diaphragm which extend from the pubic arch around the posterior part of the rectum and back to the pubic arch (Figure 4). When this muscle contracts, it draws the rectum forward, producing an angulation between the axis of the rectum and the axis of the anal canal of approximately 90^{0} (Figure 5). This angle and the tendency of the anal canal to take the shape of a slit oriented in the anterior-posterior direction produce a "flutter-valve effect" (Figure 6) which allows increases in intraabdominal pressure (such as produced by coughing) to compress the walls of the rectum and anal canal against each other, creating an obstruction of the lumen at this point (Phillips and Edwards, 1965). When defecation is imminent, the puborectalis muscle relaxes, straighting out the recto-anal angle and allowing increases in intraabdominal and intraluminal pressure to expel feces (Figure 7). (This angle is also straightened by flexion of the hips and this probably accounts for the sitting or squatting position used by most people to defecate [Tagart, 1966]). Of all the muscles responsible for maintaining continence, the puborectalis muscle is the most important.

The innervation of the muscles in this region and the nerves supplying sensory structures in the area are also important in maintaining continence (Figure 8) (Denny-Brown and Robertson, 1935; Schuster, 1968). The skeletal muscle of the pelvic diaphragm and of the external anal sphincter is absolutely dependent on the somatic nerve supply from the sacral levels of the spinal cord. The puborectalis is supplied by direct branches from the third and fourth sacral nerves which ramify on the superior surface of the pelvic diaphragm (Percy et al, 1980). The external anal sphincter is supplied by branches from the pudendal nerve.

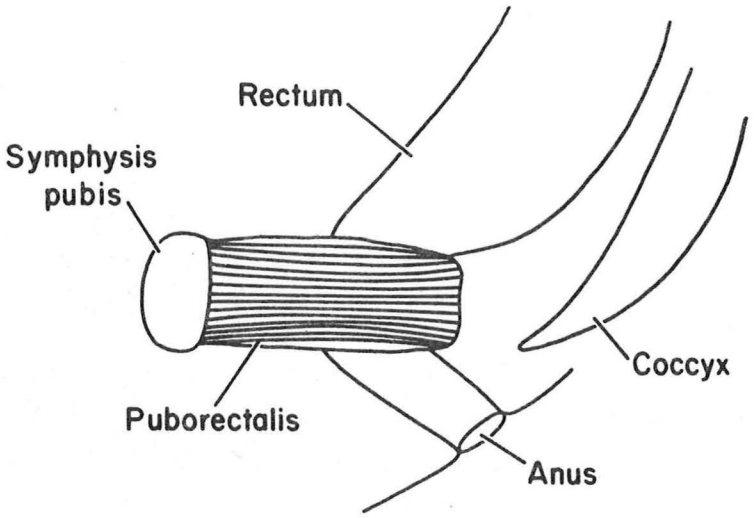

FIGURE 5. Relationship of Puborectalis Muscle, Rectum and Anal Canal at rest or when continence is threatened.

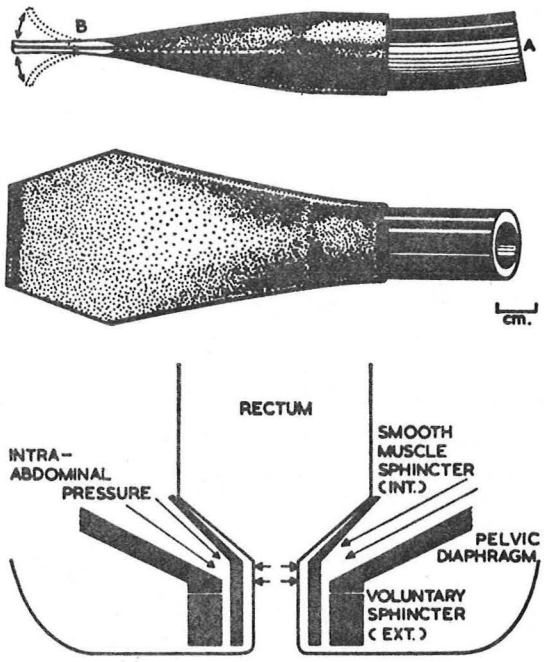

FIGURE 6. Flutter-Valve Mechanism at Recto-Anal Angulation. Increases in intraabdominal pressure can be transmitted to walls of rectum and anal canal.

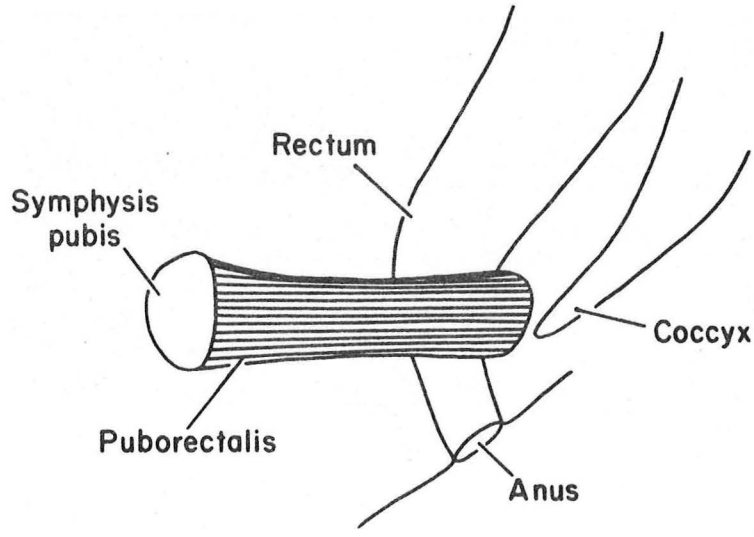

FIGURE 7. Relationship of Puborectalis Muscle, Rectum and Anal Canal during Defecation when Puborectalis relaxes.

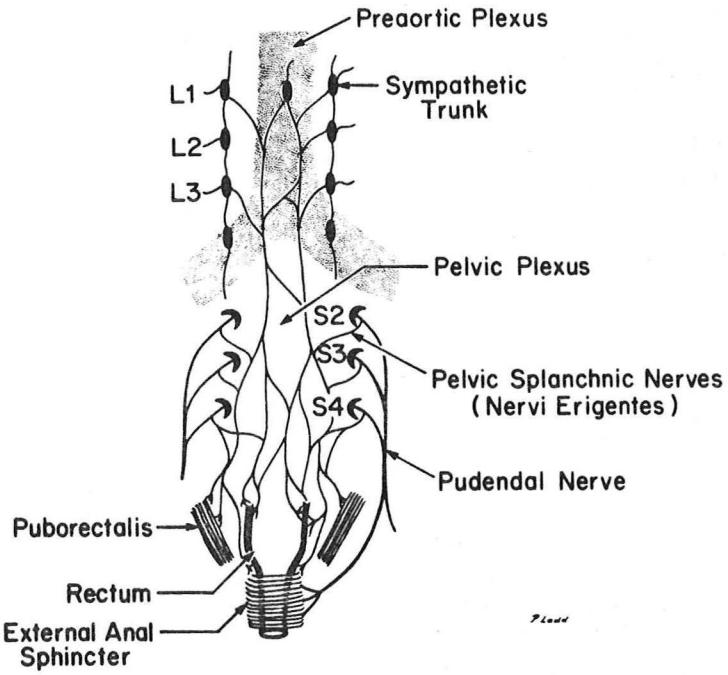

FIGURE 8. Innervation of muscles involved with continence. Sympathetic fibers from the lumbar cord and preaortic plexus descend to join parasympathetic nerves from the sacral cord in the pelvic plexus. These nerves supply the sigmoid colon, rectum and internal anal sphincter. Somatic nerves from sacral roots supply the puborectalis directly and the external anal sphincter via the pudendal nerves.

The rectum and internal anal sphincter are supplied by extrinsic autonomic nerves arising from the lumbosacral spinal cord and distributed through the pelvic plexus (Figure 8) (Frenckner and Ihre, 1976). The intrinsic nervous system of the gut including ganglia in the myenteric plexus is present in the rectum but ganglion cells are not found in the internal anal sphincter. It is likely though that nerve fibers originating in the myenteric plexus in the rectum also innervate the internal anal sphincter.

The sensory innervation of this area also appears to be important to the preservation of continence (Garry, 1933). The rectal mucosa and muscle appears to be innervated like the rest of the gut and to be sensitive only to distention. In contrast the epithelium of the anal canal is richly supplied with a variety of sensory receptors including those for pain, touch and temperature. The voluntary muscles of the pelvic diaphragm have the usual array of stretch receptors but these receptors may be of unusual importance in generating conscious feelings from this area.

Dynamics of Continence. Continence depends on a variety of dynamic responses to the movement of feces (Schuster, 1975; Wunderlich and Parks, 1982). These are summarized in Table 4.

TABLE 4. DYNAMIC COMPONENTS OF CONTINENCE

1. Delivery of Colon Contents to Rectum.
2. Rectal Compliance and Accommodation.
3. Internal Anal Sphincter Responses.
4. Rectal/Pelvic Sensation.
5. Skeletal Muscle Responses.

Abnormalities in any of these dynamic components might impair continence. Unfortunately, relatively little is known about these individual components in health or disease.

Delivery of colon contents to the rectum is the end result of the entire process of digestion and absorption. Problems with absorption or motility in the small intestine can result in passage of more material than normal into the colon. Disturbances in colon water and electrolyte transport or transit can result in abnormally large volumes of material entering the rectum. In health approximately 1000-1500 ml of fluid enters the colon each day and 100 ml leaves as feces (Krejs and Fordtran, 1983). Most of the abstraction of water from colon contents occurs in the right and transverse colon but an important fraction occurs in the left colon and is responsible for the conversion of liquid colonic contents to solid stool. The motility patterns that are responsible for the movement of colon contents from the cecum to the rectum are not well described in humans but presumably involve slow mixing contractions and more vigorous mass movements from the transverse colon to the sigmoid (Christensen, 1981; Sarna et al, 1982; Misiewicz, 1984).

In most people, the rectum is usually empty (McNeil and Rampton, 1981). When feces enter the rectum, they must be accommodated there if defecation is to be delayed. This is accomplished by the process of rectal compliance (Ihre, 1974; Arhan et al, 1976; Denis et al, 1979; Suzuki and Fujioka, 1982; Devroede, 1982). As shown in Figures 9 and 10, the rectum has both viscous and elastic properties. The viscous properties of the rectum allow it to accept a distending

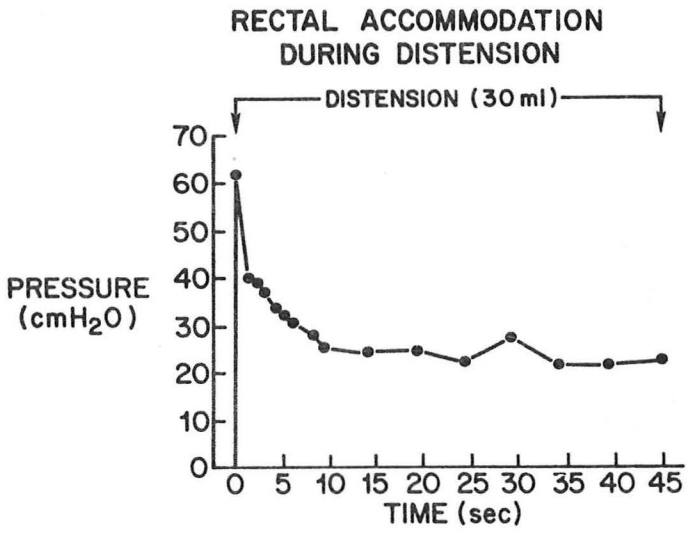

FIGURE 9. Viscous Properties of the Rectum. An intrarectal balloon was inflated with 30 ml of air and pressures were monitored. The pressure in the balloon decreased indicating relaxation of the rectal wall (After Devroede, 1982).

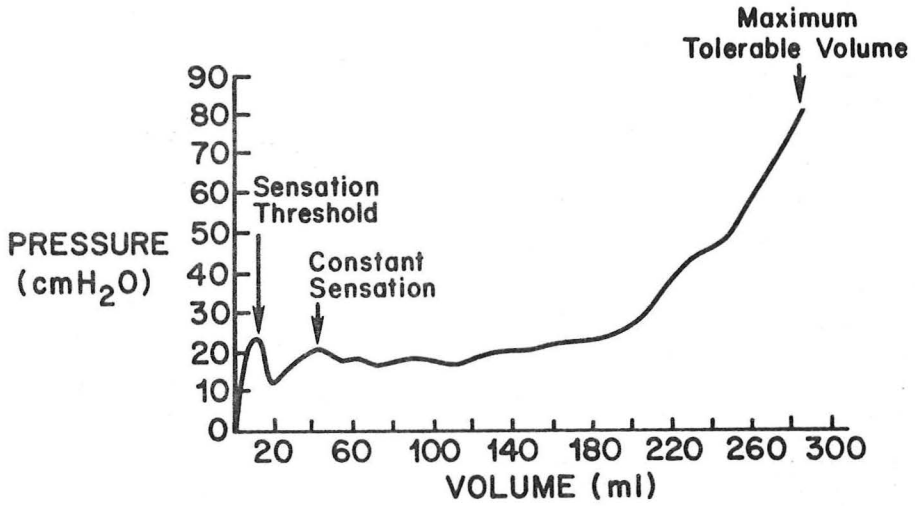

FIGURE 10. Elastic Properties of the Rectum. An intrarectal balloon was gradually inflated and pressures were monitored. The intraballoon pressure was maintained at a low level until a substantial volume had been introduced (After Devroede, 1982).

bolus and to reduce intraluminal pressure to a level that will not threaten continence. The elastic properties of the rectum allow it to maintain a low pressure while filling. These properties appear to depend on an intact intrinsic nervous system and viable muscle: rectal accomodation is impaired in both Hirschsprung's disease (Arhan et al, 1978), a disease in which the intrinsic neural plexus is abnormal, and chronic rectal ischemia (Devroede et al, 1982), a disease which produces a rigid rectal wall because of chronic fibrosis.

The internal anal sphincter maintains a basal tone which acts to keep the anal canal closed. This tonic contraction is probably due in part to intrinsic myogenic properties of the muscle (Ustach et al, 1970) and also to the action of intrinsic and extrinsic autonomic nerves (Meunier and Mollard, 1977; Frenckner and Ihre, 1976). When the rectum is distended (by the arrival of stool in nature or by inflation of a balloon in the laboratory), the internal sphincter transiently relaxes (Denny-Brown and Robertson, 1935; Schuster, 1968). An example of this is shown in Figure 11. In general, the degree of internal sphincter relaxation is proportional to the distending volume (Figure 12). This relaxation is mediated by the intrinsic neural plexus of the gut (Denny-Brown and Roberson, 1935) and is missing in Hirschsprung's disease (Schnaufer et al, 1967; Lawson and Nixon, 1967). Some cite this relaxation or the fact that transection of the internal sphincter (sphincterotomy, an operation sometimes done for the treatment of anal fissure) results in only a modest compromise of continence as evidence that the internal anal sphincter is of little or no importance to the preservation of continence (Phillips and Edwards, 1965). Others note that the internal anal sphincter regains its tone as the rectum accommodates to distention and suggest that the internal sphincter does contribute

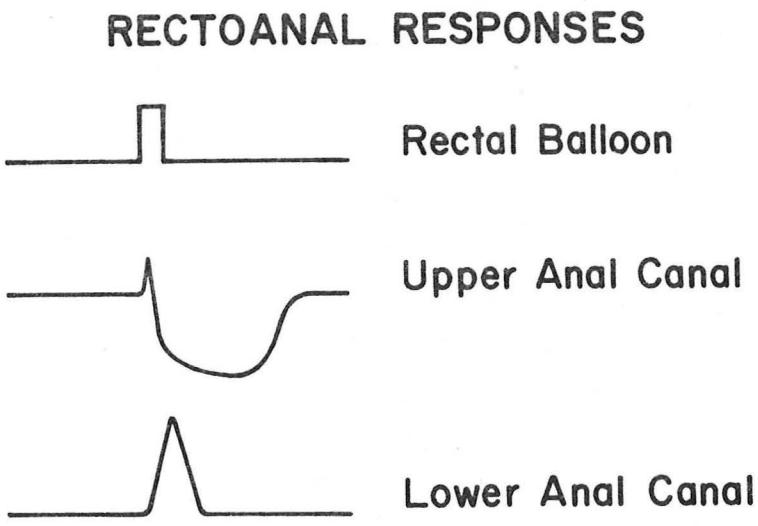

FIGURE 11. Responses of Anal Canal Pressure to Distention of a Balloon in the Rectum. The upper anal canal (reflecting largely the internal anal sphincter) relaxes and the lower anal canal (reflecting largely the external anal sphincter) contracts transiently.

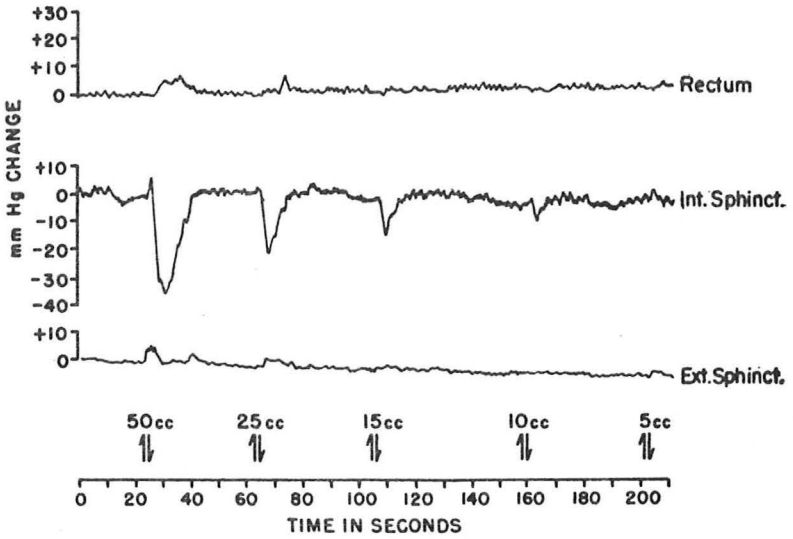

FIGURE 12. Responses of Anal Canal Pressure to Graded Rectal Distention (From Schuster, 1968).

anus. These investigators propose that internal sphincter relaxation at the time of rectal distention allows a small amount of fecal material to come into contact with the very sensitive lining of the anal canal and that this allows the individual to differentiate between solid and liquid stools and gas (Duthie, 1975). The individual could then make a conscious decision to allow the distending material (e.g., gas) to be released or to voluntarily contract the external anal sphincter to preserve continence if faced with liquid or solid stool. Whether this "sampling response" is important or not cannot be decided at present (Duthie, 1982). It does seem clear that the internal anal sphincter is the major source of resting pressure in the anal canal (Duthie and Watts, 1965; Frenckner and Euler, 1975; Schweiger, 1979) and therefore must make some contribution to the preservation of continence. Additional evidence for this contention is the relationship between basal pressure and continence for liquids under experimental and clinical circumstances (see below).

Rectal sensation is important for triggering both the autonomous smooth muscle changes and the voluntary skeletal muscle response that preserves continence (Schuster, 1968). Although we speak of rectal sensation as a unitary process, it is likely that sensation triggering smooth muscle effects and that triggering skeletal muscle effects involve different mechanisms and pathways and may involve different receptors altogether. This is probably true because the autonomic and somatic responses to rectal distention have different thresholds and are affected differently and separately by some disease processes. Thus some diseases selectively reduce conscious sensation and awareness of rectal distention while leaving the autonomic responses intact (Wald and Tunuguntla, 1984). Such conditions would be particularly apt to result in incontinence since rectal distention would result in smooth muscle (internal anal sphincter) relaxation without voluntary muscle responses to maintain continence.

The precise neuroanatomy mediating rectal sensation is uncertain (Duthie, 1982). It is possible that rectal distention activates the same intramural stretch receptors as elsewhere in the gut and that this produces internal anal sphincter relaxation by means of the intramural myenteric plexus. Evidence for this is that internal anal sphincter relaxation is lost in Hirschsprung's Disease, a condition in which the intrinsic, but not the extrinsic nerves are abnormal. It is also possible that the extrinsic autonomic nerves play a role in rectal sensation by transmitting information from the rectoanal area to the spinal cord. However, firm information on this point is not available in human beings. Whether information reaches conscious levels via the extrinsic autonomic nerves is unknown. Another mechanism for transmitting information from the anorectal area to conscious levels is via the somatic nerves innervating the pelvic muscles (Duthie, 1982). It is possible that stretching of these muscles produced by entry of feces into the rectum alters tension receptors in these skeletal muscles and sends impulses to the spinal cord and to consciousness. Some mechanism like this probably accounts for reflex activation of the pelvic diaphragm during acute increases in intra-abdominal pressure as with coughing (Dickinson, 1978).

Skeletal muscle responses are of great importance in the maintenence of continence (Schuster, 1968; Dickinson, 1978; Duthie, 1982; Wunderlich and Parks, 1982). As mentioned previously the puborectalis muscle plays a crucial role (Scharli and Kiesewetter, 1970). The tension in this muscle is under active control, increasing when increases in intra-abdominal pressure threaten continence. The puborectalis muscle is under both conscious and reflex control and is tonically active (Dickinson, 1978). The physiology underlying this tonic activity must be located at a spinal level but the details of this control are unknown as are the circuits modulating its inhibition during defecation.

The external anal sphincter is under similar control as the puborectalis muscle although the pathway of its innervation is somewhat different (pudendal nerve versus direct sacral branches for the puborectalis) (Schuster, 1975; Percy et al, 1980). The external anal sphincter response to rectal distention has been the most studied skeletal muscle response in this area. Rectal distention produced by inflation of a balloon in the rectal vault not only produces upper anal canal relaxation (due to internal anal sphincter relaxation) but also produces lower anal canal contraction (Figure 11). This has been attributed to reflex contraction of the external anal sphincter but may instead be a learned response (Whitehead et al, 1981). Evidence for this contention is that infants do not have this contractile response before toilet training and adults do not demonstrate it while sleeping. Attempts to use biofeedback training for fecal incontinence rely on the ability to learn to contract the external anal sphincter in response to rectal distention (see section on "Treatment" below).

Defecation. When sufficient material enters the rectum it normally evokes a "call to stool" (Duthie, 1982). This can be duplicated by distention of a balloon in the rectum with approximately 50-60 ml of air or water. The physiological equivalent of this is not well defined. If continence mechanisms are intact, defecation can be delayed until the person reaches a place appropriate for defecation (Figure 13). At that time the person sits or squats and the puborectalis muscle and external anal sphincter are inhibited, allowing the recto-anal angle to straighten out. The levator ani muscles contract and open the anal canal, splinting the anal canal and fixing the anus. Internal sphincter tone is inhibited, the person performs a valsalva maneuver and splints the anterior abdominal wall, thus increasing intra-abdominal pressure. This tends to force stool distally and may help to initiate a contraction in the sigmoid colon and proximal rectum, ejecting stool through the anus (Scharli and

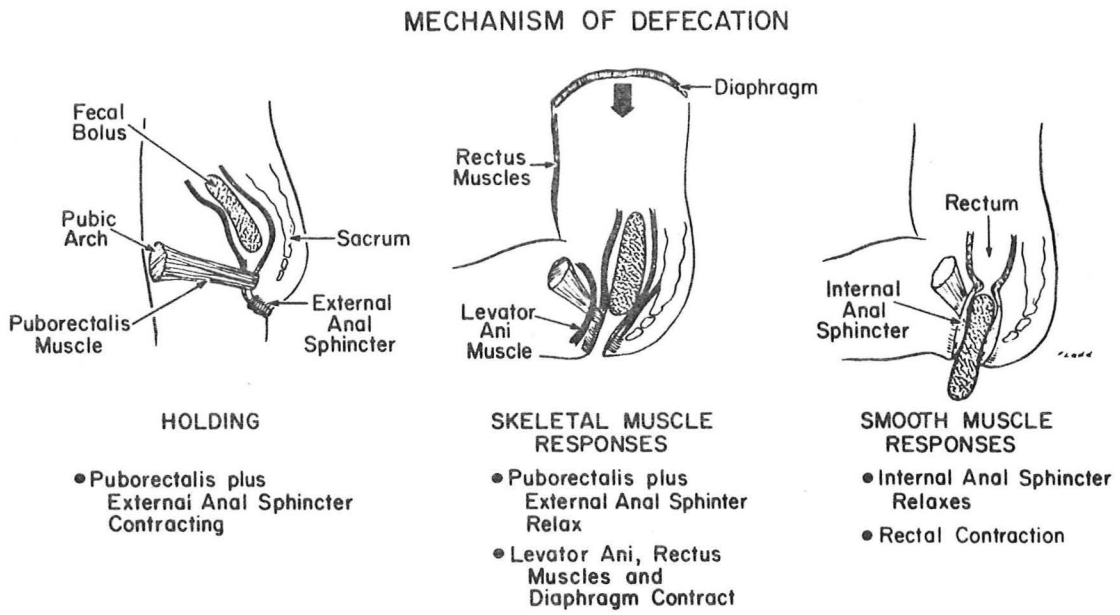

FIGURE 13. Mechanism of Defecation (See text).

Kiesewetter, 1970). It has been postulated that a defecation reflex organized in the spinal cord results in persistence of the colonic and rectal contraction as long as stool is in contact with the sensitive epithelium of the anal canal (Scharli and Kiesewetter, 1970), but the exact mechanism producing this effect is uncertain. Defecation can occur as a coordinated response even in paraplegics and thus can be organized by the lumbosacral cord without intact connections to the brain (Denny-Brown and Robertson, 1935).

PATHOPHYSIOLOGY OF INCONTINENCE

With such a complicated mechanism for the preservation of continence, it is no wonder that there can be multiple mechanisms for developing incontinence. In a conceptual way, the mechanisms causing incontinence can be divided up into anatomic problems and functional (dynamic) problems. Anatomic problems would include the effects of infection, trauma, surgery or congenital malformations on the muscles and structures regulating continence. Functional problems would include situations in which the continence structures are intact but dysfunctional.

Anatomic disruptions are easy to understand (if not treat). For instance, it is not difficult to appreciate why a patient is incontinent when he has massive deformation of the perineum due to perirectal inflammation in Crohn's disease or when he has had the puborectalis muscle cut at surgery. For this reason, I shall not discuss such problems further. Instead we shall concentrate on functional disturbances of the continence mechanisms.

Functional problems leading to incontinence can be classified by the abnormal function encountered, mirroring the normal functions contributing to continence (Table 5). Not every disease respects these functional divisions (some

have several mechanisms of producing incontinence), but it is a useful way of thinking about what has gone wrong.

TABLE 5. MECHANISMS OF INCONTINENCE

PROBLEM	EXAMPLE
1. Abnormal Delivery of Feces to Rectum	1. Voluminous Diarrhea?
2. Decreased Rectal Compliance	2. Rectal Ischemia
3. Impaired Rectal Sensation/ Perception	3. Diabetes Mellitus, Stroke
4. Impaired Internal Anal Sphincter (Smooth Muscle) Function	4. Diabetes Mellitus, Post Dilatation for Hemorrhoids
5. Impaired Skeletal Muscle Function	5. Perineal Descent, Peripheral Neuropathy

The extent to which abnormal delivery of feces to the rectum contributes to incontinence is uncertain. Patients with defective continence mechanisms clearly have more trouble with liquid stools than solid stools and yet it is unclear how much this is due to the rheologic properties of liquid stool and how much is due to voluminous amounts of stool overwhelming a basically normal continence mechanism.

To examine this issue we recently studied the ability of normal individuals to remain continent in the face of voluminous watery diarrhea. Diarrhea was induced by rapid intragastric infusion of Golytely (Braintree Laboratories, Braintree, Massachusetts), a non-absorbable solution (Davis et al, 1980) ordinarily used to prepare patients for colonoscopy (Figure 14). Golytely was

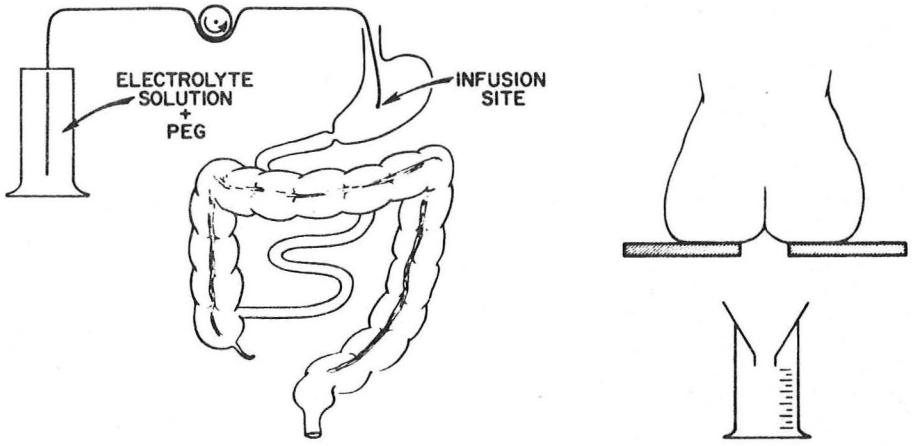

FIGURE 14. Experimental Diarrhea in the Pathophysiology of Incontinence. Golytely was infused intragastrically at a rate of 30 ml/min (450 ml/15 min) and traversed the entire gastrointestinal tract (left panel). Watery diarrhea and any leakage was collected in a graduated cylinder (right panel).

infused at a rate of 450 ml/15 min until subjects reached a steady-state in which an equal volume of watery stools was produced every 15 min (equivalent to more than 40 liters of stool in 24 hours, a flow rate seen with only the most severe cases of cholera). Subjects were then instructed to attempt to avoid defecation for 15 min while additional Golytely was infused. They were then allowed to defecate, and then asked to attempt to avoid defecation for another 15 min. This was repeated a total of 8 times. Continence was defined as the ability to avoid defecation for 15 minutes. Eight of 10 normal individuals were able to remain continent during the entire study. Two normal individuals had incontinence for 5 of their 16 experimental periods. In contrast, 4 patients with known incontinence leaked during almost every experimental period. Thus the normal continence mechanisms can withstand an enormous flow of liquid stool if presented at a relatively constant rate. This does not exclude the possibility that more rapid flows might overwhelm normal continence mechanisms, but does suggest that they must exceed a rate of 30 ml/min (450 ml/15 min) to do so. No measurements of the phasic rate of entry of feces into the rectum in either normal individuals or incontinent patients with diarrhea are available and so this mechanism for producing incontinence must remain hypothetical.

A mechanism with more impressive support is abnormal rectal compliance. If the normal pressure-volume relationship in the rectum is disturbed, accommodation of feces delivered from the colon will be disturbed and a smaller volume of feces will produce higher intrarectal pressures which could overwhelm the sphincter pressure barrier. Such a mechanism has been described in individuals with chronic rectal ischemia (Figure 15) by the group in Sherbrooke, Canada (Devroede, 1982). Whether it occurs in other diseases which infiltrate

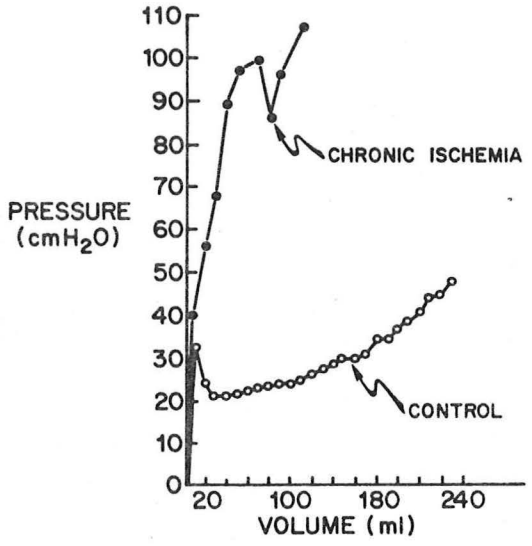

FIGURE 15. Rectal Compliance in a Normal Subject and in a Patient with Chronic Rectal Ischemia. Patients tolerated a much lower volume than normal individuals (After Devroede, 1982).

rectal muscle or change its contractile properties (e.g. inflammatory bowel disease) is uncertain and untested. It is quite likely that altered rectal compliance contributes to the symptom of urgency which often accompanies incontinence.

Abnormal rectal sensation or perception may affect continence. Individuals with altered mental status (encephalopathy, stroke, dementia) may not recognize or respond to threats to continence. Patients with neurological disease or sensory neuropathy may have diminished rectal sensation. This mechanism has been most convincingly shown for patients with incontinence and diabetes mellitus (Wald and Tunuguntla, 1984) who have higher thresholds for conscious perception of rectal distention than normal but no change in the threshold for internal anal sphincter relaxation (Figure 16). This suggests that these patients have a sensory neuropathy affecting conscious perception but that the autonomic pathways mediating the recto-anal inhibitory reflex are intact.

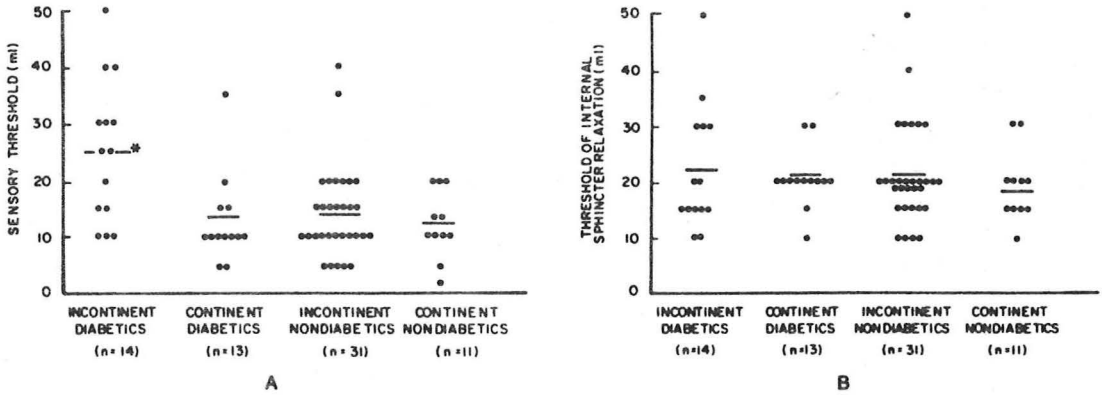

FIGURE 16. Sensory threshold for Conscious Perception (left panel) and Internal Sphincter Relaxation (right panel) in Diabetics and Controls (From Wald and Tunuguntla, 1984).

Incontinent diabetics also have problems with internal anal sphincter function (Schiller et al, 1982). As a group, incontinent diabetics have lower basal pressures (a measure of mainly internal sphincter function) than continent diabetic or normal subjects (Figure 17). This may predispose these patients to develop incontinence if they also develop diarrhea, a common gastrointestinal complication of diabetes. Indeed, diabetics often develop incontinence simultaneously or within a few years of the start of diarrhea.

Another group of incontinent patients with internal anal sphincter dysfunction includes those patients treated for hemorrhoids by forceful anal dilatation (Lord's procedure). While patients with hemorrhoids tend to have higher than normal basal anal sphincter pressures which are reduced by this procedure (Hancock, 1981), some go on to develop incontinence associated with low basal pressures (Snooks et al, 1984).

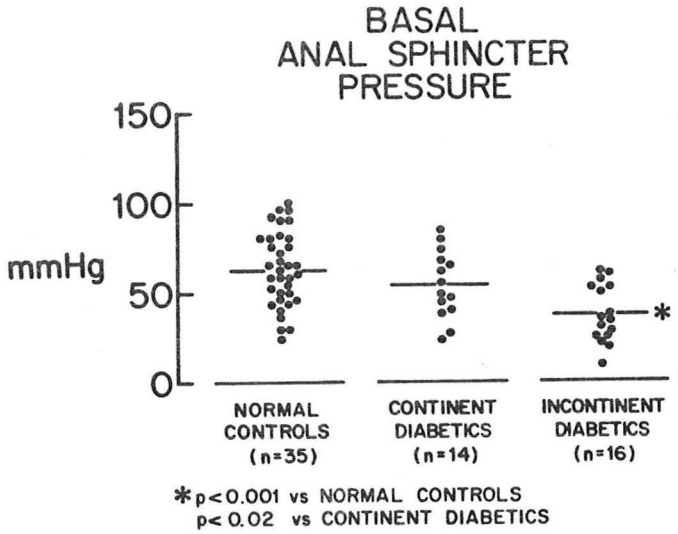

FIGURE 17. Anal Sphincter Pressures in Diabetics and Control Groups (From Schiller et al, 1982).

Abnormal skeletal muscle function appears to be a common thread in many patients with "idiopathic" fecal incontinence. Studies in England indicate that many patients with this condition have evidence of denervation of the puborectalis and external anal sphincter muscles by histopathology (Beersiek, Parks and Swash, 1979; Henry, Parks and Swash, 1982), and electromyography (Neill and Swash, 1980; Henry, Parks and Swash, 1980; Neill, Parks and Swash, 1981; Bartolo et al, 1983a; Kiff and Swash, 1984a; Kiff and Swash, 1984b). This situation is often part of the "perineal descent syndrome" in which patients have the feeling of obstructed defecation and strain at stool. This results in stretching of the pelvic floor and perineal muscles and abnormal inferior movement of the recto-anal angle. Since the motor nerves to the pelvic floor muscles (including the puborectalis) and to the external sphincter run on the surface of these muscles, stretching the pelvic floor stretches the motor nerves and damages them. This results in greater weakness of the muscles, more stretching and more weakness. Eventually the skeletal muscle can be effectively denervated and incontinence develops. Usually both the puborectalis and external anal sphincter must be compromised for incontinence to complicate the course of descending perineum syndrome (Bartolo et al, 1983b). Individuals with more severe impairment of external sphincter function tend to be incontinent for solid as well as liquid stools (Read, Bartolo and Read, 1984).

A similar mechanism may damage the continence mechanism during childbirth. Prolonged labor may cause neuropathic damage to the sphincter mechanism and this, rather than perineal tears, may result in sphincter dysfunction in multiparous women (Snooks, Setchell et al, 1984).

EVALUATION OF PATIENTS WITH INCONTINENCE

History. A careful history can go a long way toward defining the presence and cause of incontinence in a given patient. Of course, the most important point is to ask whether or not a patient has episodes of fecal incontinence. Few patients will volunteer this information and it is usually necessary to ask about it directly. This is especially important in patients complaining of chronic diarrhea, urgency, other defecation problems, urinary incontinence, neurological diseases, muscle diseases, and diabetes mellitus. Synonyms such as "leakage", "soiling" or "accidents" should be used if necessary.

Once a history of fecal incontinence is elicited, questioning should include whether or not loose stools are also present, how long the incontinence has been present and if there are any physical problems or barriers to defecating in an appropriate place (such as the need for assistance to get to the toilet or a flight of stairs that needs to be climbed to get to a toilet). The physician should determine whether incontinence is occurring only during sleep or some other alteration of consciousness, such as after taking sedatives, or whether it is occurring while the patient is fully awake. It is also important to explore the adaptations that a patient may have made to limit the possibility of an incontinent episode occurring. It is only against such a framework that the severity of incontinence can be judged.

Questioning should then be directed at possible causes of incontinence: the patient should be asked about anorectal trauma, infections and operations; about straining at stool and difficult childbirth; about problems with skeletal muscle function elsewhere in the body; about autonomic and peripheral neuropathy, spinal cord injury and stroke; and about the presence of diabetes mellitus. The temporal relationship between any of these conditions and incontinence should be developed. The clues gained from the history can be used to direct the rest of the evaluation.

Physical Examination. The rectal examination is usually one of the more perfunctorily performed parts of the physical examination. Often it is aimed solely at feeling the prostate gland or obtaining a stool specimen to test for occult blood. It can be and should be much more in the incontinent patient.

The perianal skin should be inspected for evidence of dermatitis or infection, local hygiene, and the presence of scars. The anus should be examined both while the patient is relaxed (for observation of hemorrhoids and skin tags) and while the patient is bearing down (for observation of rectal prolapse or leakage). The perianal skin should be lightly stroked with a pin to induce the cutaneo-anal contractile reflex (Wunderlich and Parks, 1982). During this maneuver, the subcutaneous external anal sphincter should contract and produce a puckering at the anal margin.

The gloved examining finger should be lightly lubricated and then inserted into the anal canal. Although it is impossible to estimate anal sphincter pressures accurately by digital assessment, a frankly patulous anal canal can be readily distinguished by most examiners (Figure 18). The patient should be asked to squeeze the anal sphincter as if trying to prevent defecation and the ability to generate a squeeze pressure can be noted. The cutaneo-anal reflex can again be elicited by stroking the perianal skin with a pin and the reflex contraction of the external sphincter can be appreciated by the examining finger. The puborectalis can be palpated in the posterior midline as an extrinsic transverse bar. This muscle should be stretched by gently pulling the examining finger posteriorly and the anal canal should be observed for gaping

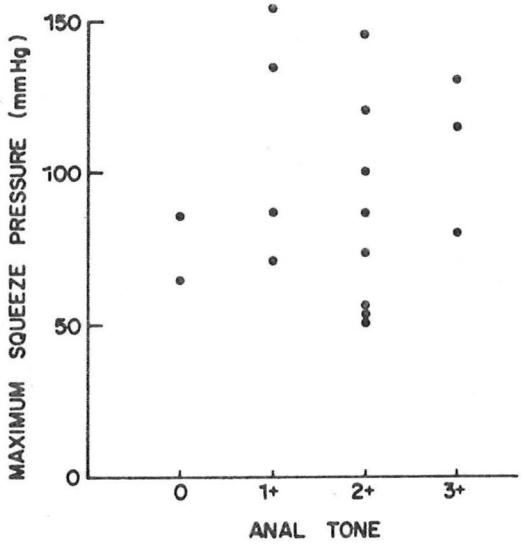

FIGURE 18. Physician Estimates of Anal "Tone" Versus Measured Pressures.

(Devroede, 1982). Such a response indicates weakness of the skeletal muscles in this area. The patient should then be asked to try to prevent the passage of stool again and a firm anterior tug produced by the puborectalis should be felt. The examining finger should then palpate each quadrant of the anal canal for the presence of fluctuation or scar. The presence or absence of a fecal impaction should also be noted, since incontinence of liquid feces can sometimes occur around an impaction. Lastly, the prostate and rectal wall should be palpated for tumor and a fleck of stool should be retrieved for occult blood testing.

These observations can allow one to draw conclusions about the presence or absence of local pathology and can give the examiner several clues as to the functional status of the nerves and muscles in the rectoanal area.

Objective Tests. A series of physiological tests can be applied to obtain objective information about rectoanal function and capabilities. These are listed in Table 6 and discussed below.

TABLE 6. OBJECTIVE TESTS OF RECTOANAL FUNCTION

TEST	PURPOSE
Perfusion Manometry	Measurement of basal and squeeze pressures in anal canal
Balloon Manometry	Measurement of rectal sensation, rectal compliance, recto-anal reflex relaxation and recto-anal contractile response
Test of Continence for Solid Sphere	Assessment of active and passive resistance to passage of a solid bolus
Test of Continence for Rectally-Infused Saline	Quantitative assessment of ability to maintain continence against a reproducible stress
Electromyography of Puborectalis and External Anal Sphincter	Assessment of motor nerve supply and skeletal muscle responses
Radiographic Measurement of Recto-Anal Angle	Assessment of perineal descent and puborectalis function

Perfusion manometry can be used to provide a direct measure of the pressures generated in the anal canal under basal conditions and with squeezing. We use a triple lumen catheter with recording ports located at the same level 120° apart (Figure 19) and a station-pull-through technique which allows measurements of basal and squeeze pressures during withdrawal of the catheter through the anal canal (Read et al, 1979). Others have used different pressure recording devices such as intraluminal transducers and balloon devices (Schuster, 1968; Devroede, 1982). Important artifacts in pressure recordings may result from radial and longitudinal variations in pressure in the anal canal (Taylor, Beart and Phillips, 1984). There are also important differences in pressures measured in men and women and in young and old patients which need to be taken into consideration when interpreting the results of these studies (Table 7) (Read et al, 1979). Physicians are unable to accurately estimate sphincter pressure from digital examination alone and thus measurements of pressure are of fundamental importance in evaluating these patients.

TABLE 7. AGE AND SEX DIFFERENCES IN ANAL PRESSURES IN NORMAL SUBJECTS

	Sphincter Pressure (mmHg)	
	Basal	Squeeze
Men (N = 15)	66±6	218±18
Women (N = 22)	58±3	135±15
Ages 23-49 (N=19)	69±5	214±20
Ages 50-83 (N=18)	54±4	126±11

FROM READ ET AL, 1979

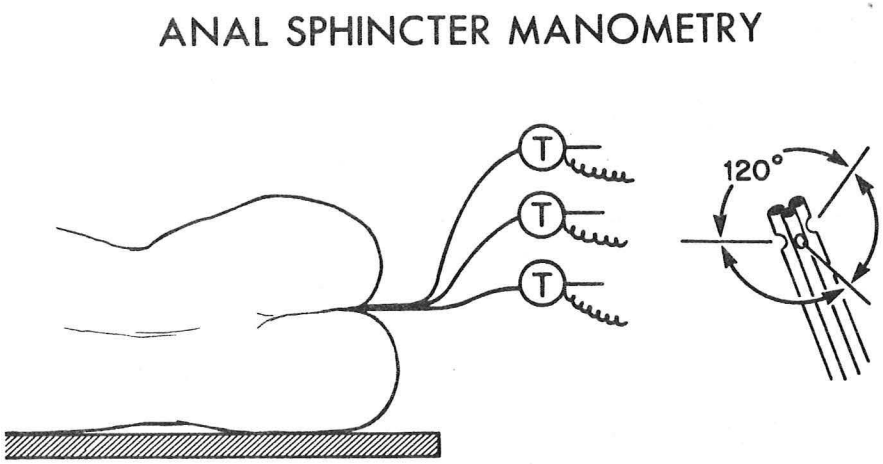

FIGURE 19. Technique of Anal Sphincter Manometry.

Studies done here at Soutwestern Medical School show that, as a group, patients with incontinence have significantly lower basal and squeeze pressures than age and sex matched control patients or continence patients with diarrhea (Read et al, 1979). These difference are shown in Figure 20. However, as you can see there is considerable overlap among groups.

Basal sphincter pressure is thought to be largely due to tonic contraction of the internal anal sphincter because it is well maintained even with nerve blocks that paralyze the external anal sphincter (Duthie and Watts, 1965; Frenkkner and Euler, 1975; Schweiger, 1979). The component of squeeze pressure that exceeds basal pressure is due to active contraction of the external anal sphincter and is abolished by similar nerve blocks. Since the sphincters are coaxial over most of their length, one usually does not find segments representing only the internal or only the external anal sphincters except at the anal verge where the subcutaneous external anal sphincter may sometimes be recorded by itself.

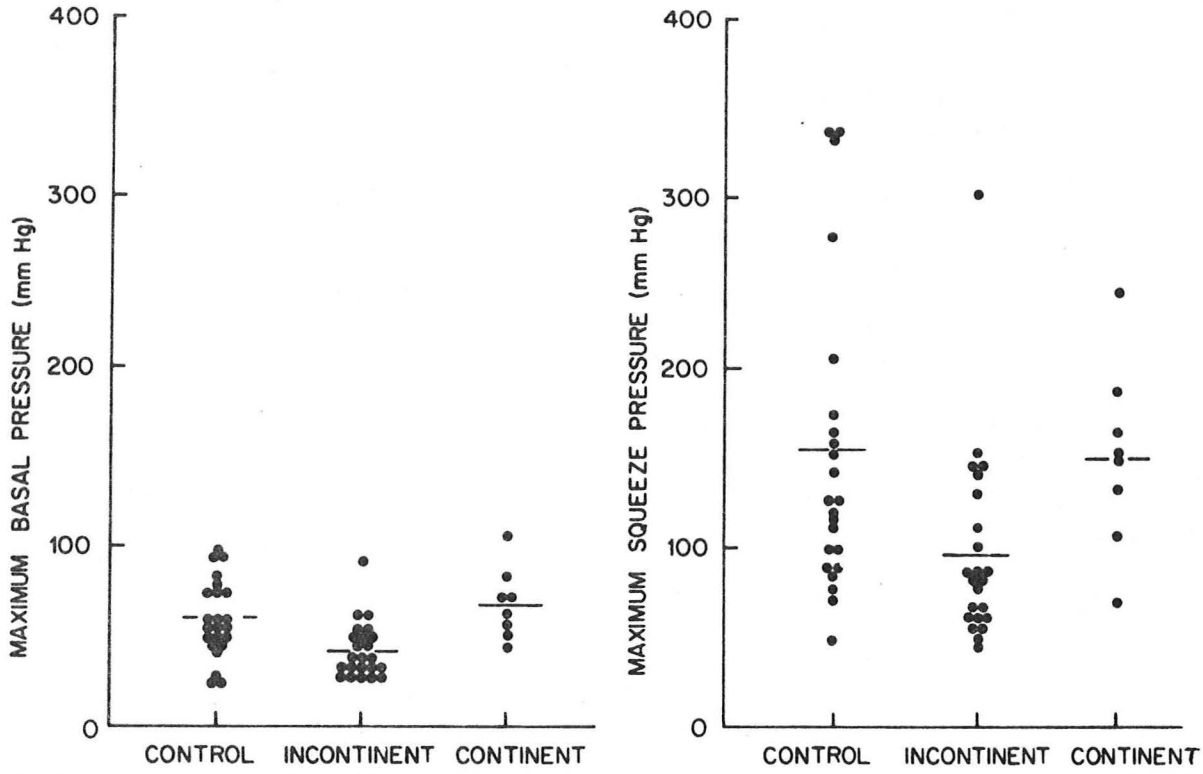

FIGURE 20. Basal (left) and Squeeze (right) Pressures Measured in Continent Control Subjects, incontinent patients with chronic diarrhea and continent patients with diarrhea (From Read et al, 1979).

Balloon manometry, in which a three-balloon assembly ("Schuster Probe") is inserted into the rectum and anal canal (Figure 21), can provide information about several physiological functions (Schuster, 1968). By inflating the rectal balloon with increasing volumes of air, the threshold for conscious sensation of rectal distention can be determined. Most normal individuals can sense as little as 10 ml of air in the rectal balloon. Some incontinent patients (most notably diabetics) have higher sensory thresholds that might contribute to the occurrence of incontinence by allowing greater volumes of stool to enter the rectum unknowingly.

By monitoring the pressure in a balloon inflated in the upper anal canal, the recto-anal inhibitory reflex can be appreciated (Figure 22) and the threshold for inducing this reflex can be measured. This threshold is usually

higher than that for conscious sensation and averages about 20 ml in normal subjects. The response of the upper anal canal to rectal distention can be quantitated as to its amplitude and duration which, in general, are related to the volume with which the rectum is distended. However, since these measurements are usually made with an air- or fluid-filled balloon the actual pressure recorded may have only a distant relationship to the anal canal pressures measured by perfusion manometry (Schuster, 1968).

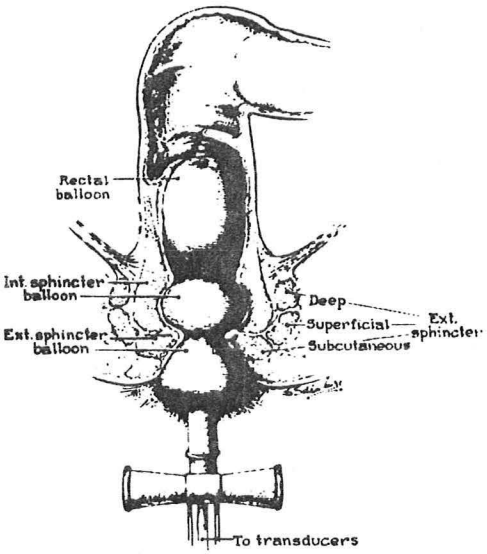

FIGURE 21. Balloon Manometry Device ("Schuster Probe").

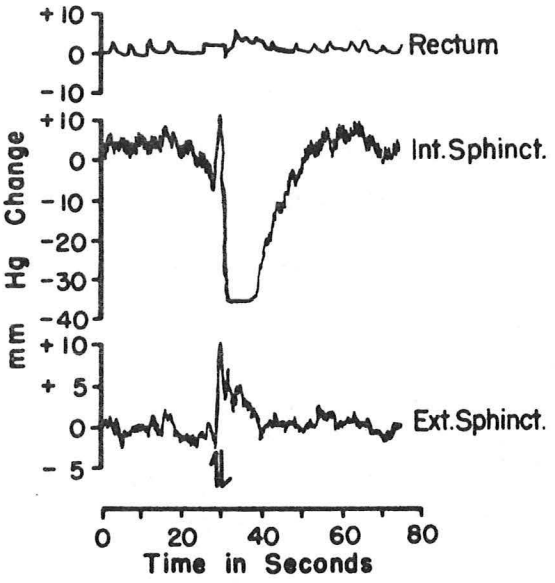

FIGURE 22. Pressure Responses Recorded by Balloon Manometry

The pressure in the balloon placed in the lower anal canal reflects the contractile activity in the external anal sphincter because it is usually wedged at the anal verge and can feel the contraction generated by the subcutaneous portion of the external anal sphincter, the recto-anal contractile response. The usual manometric record (Figure 22) shows a sharp inflection simultaneous with internal anal sphincter relaxation but usually of shorter duration than internal sphincter relaxation. The amplitude and duration of this contractile response can be measured, but again is only indirectly related to pressures measured with a perfused catheter. Many incontinent patients have lost this learned response (Engel, Nikoomanesh, and Schuster, 1974). This aspect of balloon manometry is important in the technique of biofeedback training for fecal incontinence (see below).

Balloon manometry can also be used to estimate rectal compliance. By gradually inflating a balloon placed in the rectum and simultaneously recording pressure within the balloon, a compliance curve can be generated (Devroede, 1982). This should be corrected for the elastic properties of the balloon itself by subtracting the pressure-volume curve obtained outside the body.

Another objective test of the continence mechanisms is the test of continence for a solid sphere (Read et al, 1979). In this test, a small sphere attached to a string (actually, a fishing bob) is inserted into the rectum. The patient is seated on a chair with a central aperture and the string is attached to a bucket (Figure 23). Steel shot (BB's, 3 mm in diabeter) are then poured into the bucket until the sphere is pulled from the rectum. The weight of the filled bucket is recorded. The test is done once with the patient making no special effort to prevent passage of the sphere and once with the patient trying her hardest to prevent passage of the sphere.

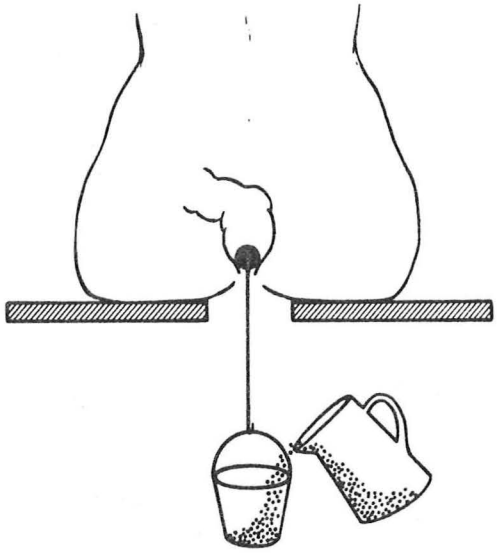

FIGURE 23. Test of Continence For Solid Sphere.

Results of this test are shown in Figure 24. Incontinent patients required significantly less weight to pull the sphere from the rectum than control subjects both with their sphincters relaxed and contracted (Read et al, 1979). The weight required to pull the sphere through the contracted sphincter correlates well with maximum squeeze pressure and probably is related to the strength of the external anal sphincter.

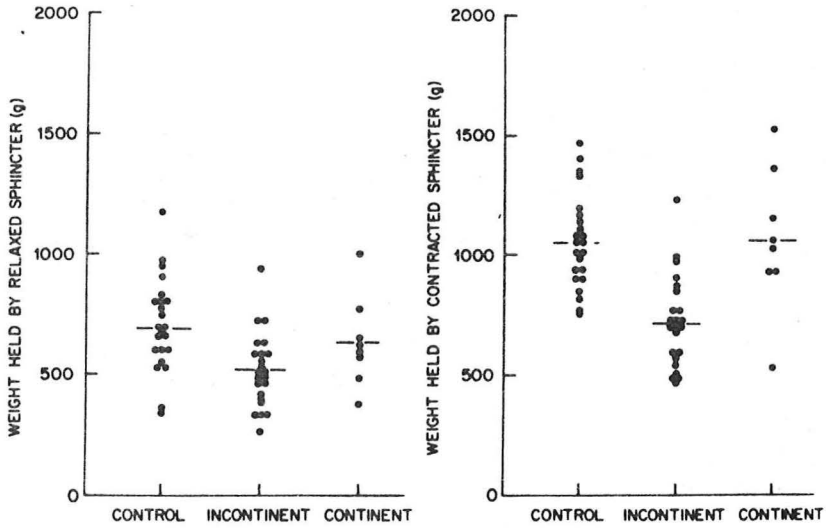

FIGURE 24. Results of Test of Continence for Solid Sphere (From Read et al, 1979).

The test of continence for rectally infused saline evaluates the interaction of all mechanisms preserving continence in an individual and is especially relevant to incontinent patients with diarrhea (Read et al, 1979). In this test, the patient is again seated on a special chair with a central aperture (Figure 25). A fine catheter is inserted into the rectum and 1500 ml of saline is pumped into the rectum over 25 minutes. (This is the approximate volume used to fill the colon for a barium enema.) The patient is instructed to try to hold on to all the infused fluid. A funnel and graduated cylinder are placed under the chair to catch any leakage. The cumulative volume retained can be plotted versus time (Figure 26).

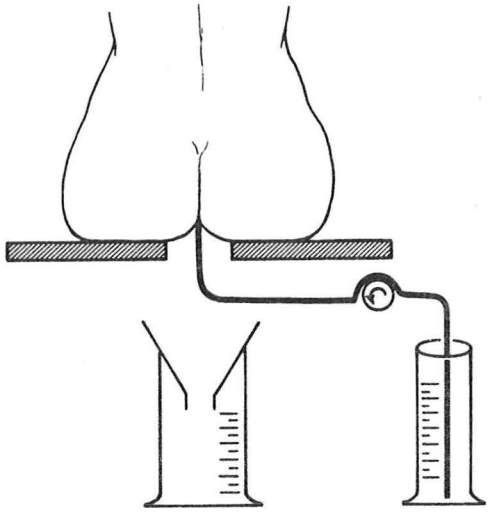

FIGURE 25. Test of Continence for Rectally-Infused Saline.

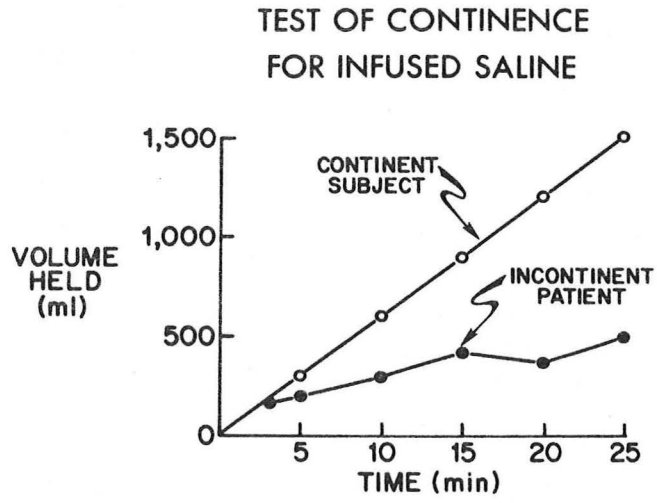

FIGURE 26. Examples of two possible results of Test of Continence for Rectally-Infused Saline.

Studies in which a radioisotope was added to the saline show that the rectum fills to an average maximum of 525 ml over approximately 10 minutes, the sigmoid colon fills to a maximum of less than 200 ml, the descending colon begins to fill after 5-7 minutes and the transverse and ascending colon after 10 minutes (Haynes and Read, 1982). During infusion there is a pattern of regular rectal pressure peaks and internal sphincter relaxations frequently (but not always) associated with transient external sphincter contractions. However, at no time in continent individuals does the highest rectal pressure exceed the lowest pressures on the anal canal indicating the presence of a persistent pressure barrier in the anal canal (Haynes and Read, 1982).

Most normal individuals do not leak at all during this test (Figure 27). In contrast, most incontinent patients leak a fair amount of fluid during the period of infusion. In one study, leaks coincided with peaks in rectal pressure in incontinent patients (Read et al, 1983). These incontinent patients belonged to one of two groups. In one group of patients, basal pressure was low and easily overcame by even modest increases in rectal pressure. In another group of incontinent patients, abnormally high peak rectal pressures overcame normal basal pressures (Read et al, 1983). The genesis of this overpressure is unknown but may have to do with colon or rectal compliance since patients in the latter group tended to have greater intrarectal pressures for a given volume of rectal distention.

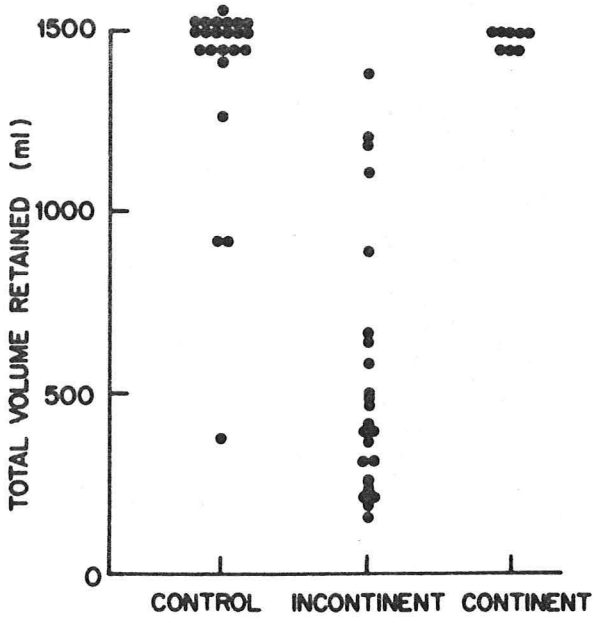

FIGURE 27. Results of Test of Continence for Rectally-Infused Saline (From Read et al, 1979).

Results in this test are not dependent upon mucosal sensation since continence is maintained during rectal infusion even after topical anesthesics have been applied to the anal canal (Read and Read, 1982). In normal subjects there is a logarithmic relationship between basal pressure and volume held (Figure 28), suggesting that internal anal sphincter contraction is important in the maintenance of continence during this test. As expected from the results cited previously, this relationship is disturbed in patients with incontinence.

The value of the test of continence for saline is that it places a fixed and reproduceable stress on the continence mechanisms. The determination of volume leaked correlates in a crude way with severity of incontinence under natural conditions (Read et al, 1979), but eliminates the variation in whether or not incontinence will occur due to stool consistency or the availability and nearness of toilit facilities. It is therefore an excellent way to reproduceably challenge the continence mechanisms and thus judge the effect of therapy for incontinence. It can also be used to establish the presence of a defect in con-

tinence in persons with an unclear history or with a latent problem with continence.

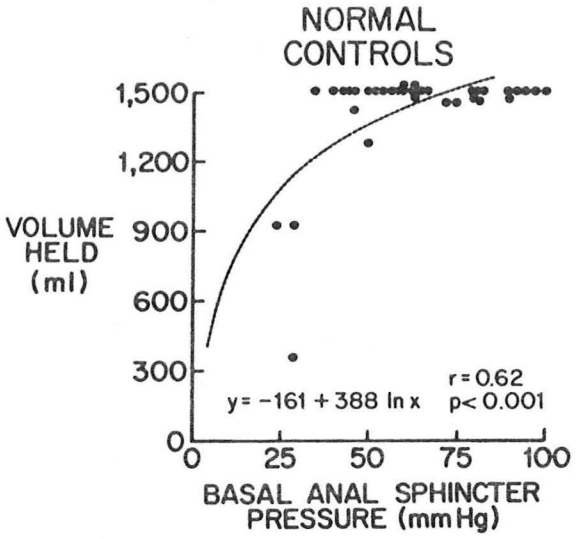

FIGURE 28. Relationship between Basal Anal Sphincter Pressure and the Volume Retained in Test of Continence for Rectally-Infused Saline.

Two problems with the test of continence for rectally-infused saline are: 1) it depends in a critical way on the patient's motivation to hold the saline, and 2) the fluid is infused in a retrograde fashion into the gut which does not mimic the normal flow of contents through the colon. The extent to which these problems limit its usefulness depends on the patient being studied and the question being asked.

Electromyography, both with standard concentric needles or with more sophisticated, single fiber EMG techniques, can provide information about the innervation and responses of the skeletal muscles involved with continence (Henry and Swash, 1978; Swash, 1982; Pedersen et al, 1982; Bartolo, Jarratt and Read, 1983 a and b). EMG techniques rely on recording the motor unit potential duration, latency between either cutaneous or electrical stimulation and the onset of motor activity, or the integrated amplitude of the EMG signal which correlates with the strength of contraction of skeletal muscle. These measurements provide information about the presence of denervation and reinnervation of these muscles and the soundness of reflex arcs affecting either the external anal sphincter or puborectalis muscles. EMG is useful in establishing a neuropathic origin of incontinence in patients without an obvious cause for their condition.

Radiographic measurement of the rectoanal angle and of the extent of perineal descent is also useful in selected patients. By injecting a small amount of barium into the rectum, placing a metal chain through the anal canal and making a well penetrated lateral view of the pelvis, the angle made by the axis of the rectum and the anal canal can be measured as can the relationship of this angle to a line drawn between the public arch and tip of the coccyx (Bartolo et al, 1983b). Normally the angle is maintained at approximately 90^{o} at rest and when straining and the angle itself is located within 1 cm of the pubococcygeal line (Figure 29). Patients with pelvic floor (perineal) descent have more obtuse rectoanal angles and angles located distinctly below the pubococcygeal line, if not at rest, when straining (Figure 30). Since this syndrome is thought to produce ongoing damage to the innervation of the puborectalis and external anal sphincter, identification of these individuals might allow for modification of their pattern of defecation and the prevention of further disability.

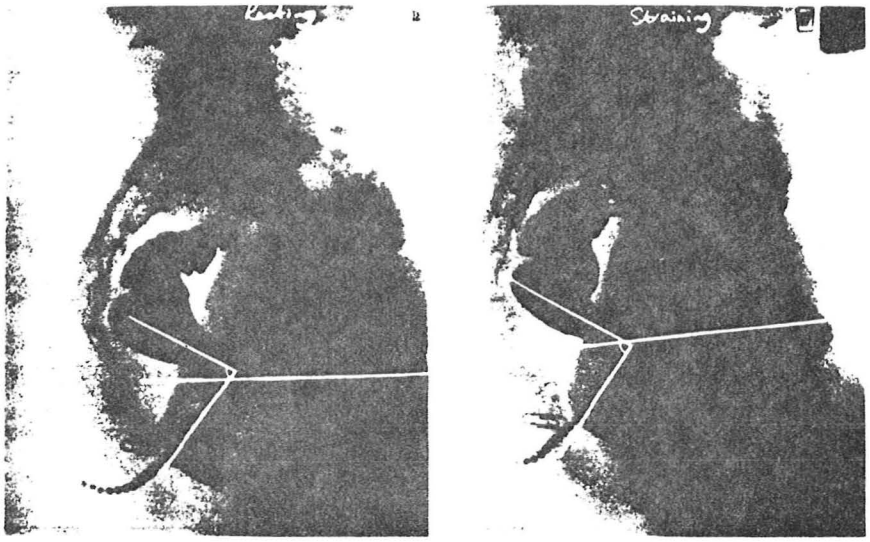

FIGURE 29. Lateral Radiographs of the Pelvis in a Normal Individual at rest (left) and while straining (right). The rectoanal angle and pubococcygeal line are indicated (From Bartolo et al, 1983b).

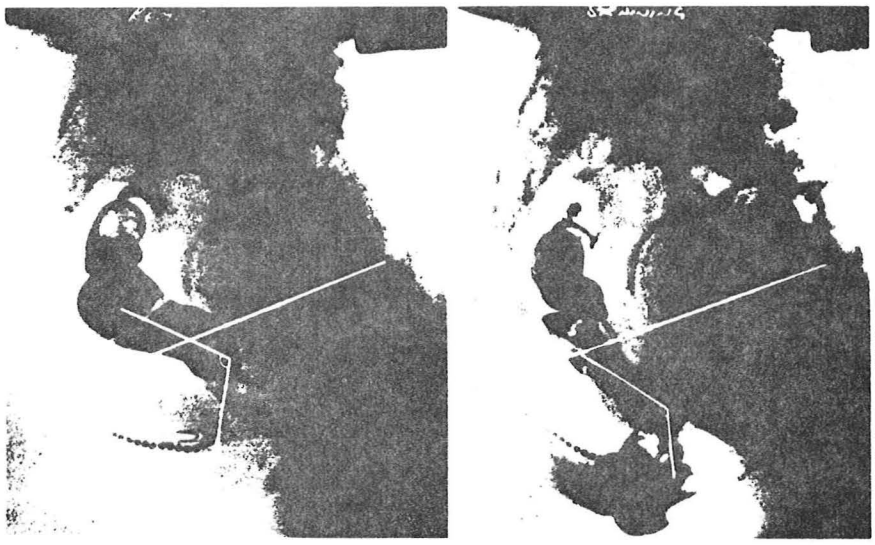

FIGURE 30. Lateral Radiographs of the Pelvis in a Patient with Perineal Descent at rest (left) and while straining (right). Note the obtuse rectoanal angle and its relation to the pubococcygeal line (From Bartolo et al, 1983b).

Other radiographic techniques have also been described for evaluating the anorectum and act of defecation (Preston, Lennard-Jones and Thomas, 1984; Kuipers and Strijk, 1984).

TREATMENT OF FECAL INCONTINENCE

General Principles. If a specific cause for incontinence can be discovered, therapy directed against that problem may help to alleviate incontinence. Thus, a diabetic patient with incontinence should have careful therapy for her diabetes in the hope that the autonomic neuropathy which probably is responsible for incontinence will resolve (Feldman and Schiller, 1983). Other etiologies such as perineal descent syndrome may not progress further if the patient can be helped to modify her defecation habits to prevent straining at stool.

Stimulating defecation at regular intervals to keep the colon and rectum empty of feces has also been recommended by some, particularly in patients with fecal impaction and "overflow incontinence" (Brocklehurst, 1975).

If diarrhea is a coexisting problem, vigorous attempts to diagnose and treat the patient may result in a solid stool that can more easily be controlled by the continence mechanisms. Bulking agents or nonspecific antidiarrheal drugs may have a place in this regard (see below).

Patients with incontinence often need psychological help in dealing with their problems. Often specific recommendations as to the use of "training pants" or other continence aids can provide the patient with enough confidence to resume more normal social activities (Beber, 1980; Morton, 1981).

Of course, the best basis for reassurance is to do something that will prevent, or at least reduce the chance of incontinence. Three such approaches are available: drug therapy, biofeedback training and surgery.

Drug Therapy. Only two drugs have been scientifically evaluated for an effect on incontinence: Lomotil and loperamide, both opioid antidiarrheals. The study of Lomotil was done here at Southwestern Medical School (Harford et al, 1980) and compared the acute effect of Lomotil and placebo on stool weight, frequency, anal sphincter pressure and tests of continence for a solid sphere and for rectally-infused saline in patients with chronic diarrhea and fecal incontinence. Although this antidiarrheal had its expected effects on stool weight and frequency, it had no effect on any of the other tests. Incontinent episodes occurred too infrequently while these patients were under observation in the hospital to draw any conclusions as to the effect of this drug on the frequency of incontinent episodes.

The study of loperamide used a similar format (M Read et al, 1982) but found that loperamide 4 mg TID significantly reduced the frequency of incontinent episodes and urgency, increased basal anal sphincter pressure and improved the results of the test of continence to rectally-infused saline. While these results seem impressive, only 11 of the 26 patients had episodes of incontinence during the course of the trial and only 17 of the 26 had abnormal retention of saline before treatment with loperamide. Still, when faced with a patient with chronic diarrhea and incontinence, loperamide seems to be the better nonspecific antidiarrheal to use.

The pharmacology of the rectal and internal sphincter smooth muscle is just starting to be explored (Parks et al, 1969; Burleigh, D'Mello and Parks, 1979). The regulation of these muscles appears to be quite complex and the muscles

themselves have striking regional differences when studied in vitro. For instance, smooth muscle strips taken from the upper anal canal respond differently to the same agents than muscle strips taken from the lower anal canal. Traditional neurotransmitters like acetylcholine and noradrenaline as well as more recently recognized neurotransmitters such as prostaglandins, serotonin, dopamine, adenosome triphosphate, vasoactive intestinal polypeptide and neurotensin appear to be involved with the mechanisms regulating smooth muscle. This presents the possibility that pharmacological intervention may modify sphincter function, but various agonists and antagonists have not been studied systematically in this regard. Perhaps some day, therapy for incontinence will include such agents but that day is not here yet.

Biofeedback Training. Operant conditioning in the term applied by psychologists to the process of learning in which a particular response is elicited by a stimulus because that response produces consequences desired by the organism. As it pertains to fecal incontinence it involves techniques designed to reinforce physiological mechanisms preserving continence.

As designed by Schuster and colleagues (Engel, Nikoomanesh and Schuster, 1974), operant conditioning is utilized to improve the conscious threshold for sensation of rectal distention and to coordinate external anal sphincter contraction with rectal distention. These investigators noted that incontinent patients often had unusually high sensory thresholds and often lacked the lower anal canal contractile response to rectal distention. They reasoned that patients might be able to improve their sensory threshold and contract the external sphincter in a timely fashion if they could practice these tasks in a setting where they could receive conscious information about the occurrence of rectal distention.

To do this they utilized the three-balloon manometry device described earlier (Figure 21). The device and the proper responses on the physiograph are then described to the patient and the patient is shown how he can produce external anal sphincter contraction and alter the physiograph tracing. He is then instructed to do this everytime the rectal balloon is distended. The rectal balloon is rapidly inflated to the sensory threshold and deflated, a maneuver that produces a characteristic spike on the pressure record from the rectal balloon which the patient can see. The appropriate response (external anal sphincter contraction) is rewarded verbally and then the process is repeated again and again. The volume in the rectal balloon is gradually reduced as the patient is able to sense rectal distention consistently. This process is called biofeedback training because the physiograph record provides information to the patient about a biological process which would otherwise be unconscious. This information can be used to modify his response which gradually becomes automatic. After a while the physiograph display is removed from the sight of the trainee and the extent to which he has learned to perceive rectal distention and to coordinate the external sphincter response can be judged. A successful session is illustrated in Figure 31.

Reports from centers where biofeedback training is regularly done suggest that approximately 70% of patients in whom training can be done (i.e., patients with some degree of perception of rectal distention and the ability to generate a squeeze pressure) have either a disappearance or substantial reduction of spontaneous episodes of incontinence (Cerulli, Nikoomanesh and Schuster, 1979; Goldenberg et al, 1980; Wald, 1981). Most of these patients responded after a single session, and in most the results were long-lasting.

In one study (Cerulli, Nikoomanesh and Schuster, 1979), patients with a good

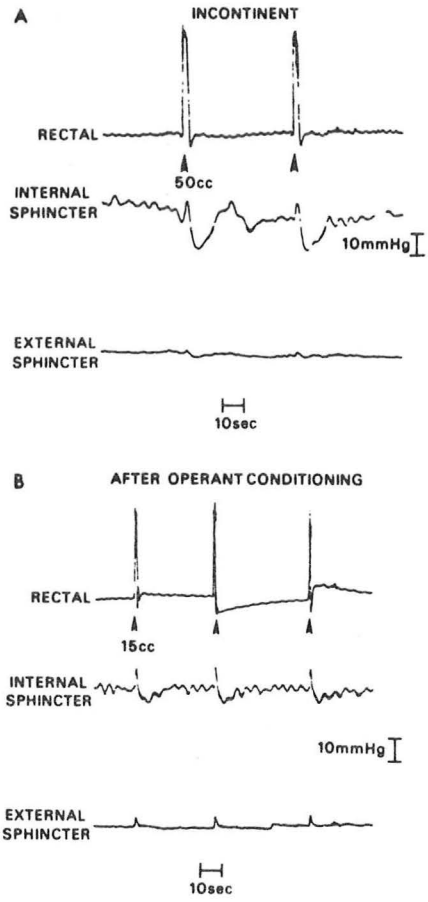

FIGURE 31. Successful response to biofeedback therapy shows rectoanal responses before (upper panel) and after (lower panel) biofeedback training (From Cerulli, Nikoomanesh and Schuster, 1979).

response were different than poor responders in that their threshold for conscious sensation improved from approximately 40 ml to 15 ml whereas the nonresponders had no significant improvement in sensory perception. Whether this improvement alone or the combination of improvement in sensation and appropriate external anal sphincter contraction (or just exercise of the pelvic floor) is necessary for improvement with this technique is unresolved. No blinded trials of biofeedback or its components have been tried and there have been no attempts to show changes in any objective tests of sphincter function or continence mechanisms. It is also unclear how sensory improvement is mediated if the sensing structures or pathways are damaged. Thus we cannot say how biofeedback works to improve continence or even that it does work better than placebo treatment would.

Still, the overwhelming suggestion of collected series is that biofeedback does work. However, it is clear that patients must be carefully selected if response rates are to be good. To be reasonable candidates for biofeedback therapy, patients must have some ability to sense rectal sensation (albeit at a higher than normal threshold), good motivation, the ability to understand instructions and the ability to contract the external anal sphincter voluntarily. Patients with dementia, major central nervous system disease, absence of rectal sensation or inability to contract the skeletal muscles of the area cannot be expected to respond to biofeedback therapy.

Other techniques than the triple balloon probe have also been used for biofeedback training for fecal incontinence in small groups of patients. These have included a cheap pneumatic device for home use (Constantinides and Cywes, 1983), an intranal plug and electromyometer (MacLeod, 1983) and intrarectal

infusion of saline (Schiller et al, 1979). This last technique involved repeated use of the test of continence for rectally-infused saline with the biofeedback consisting of knowledge of the leaked volume. One patient subjected to this technique was able to improve the volume of saline held and was able to become continent for natural stools even though chronic diarrhea persisted (Figure 32).

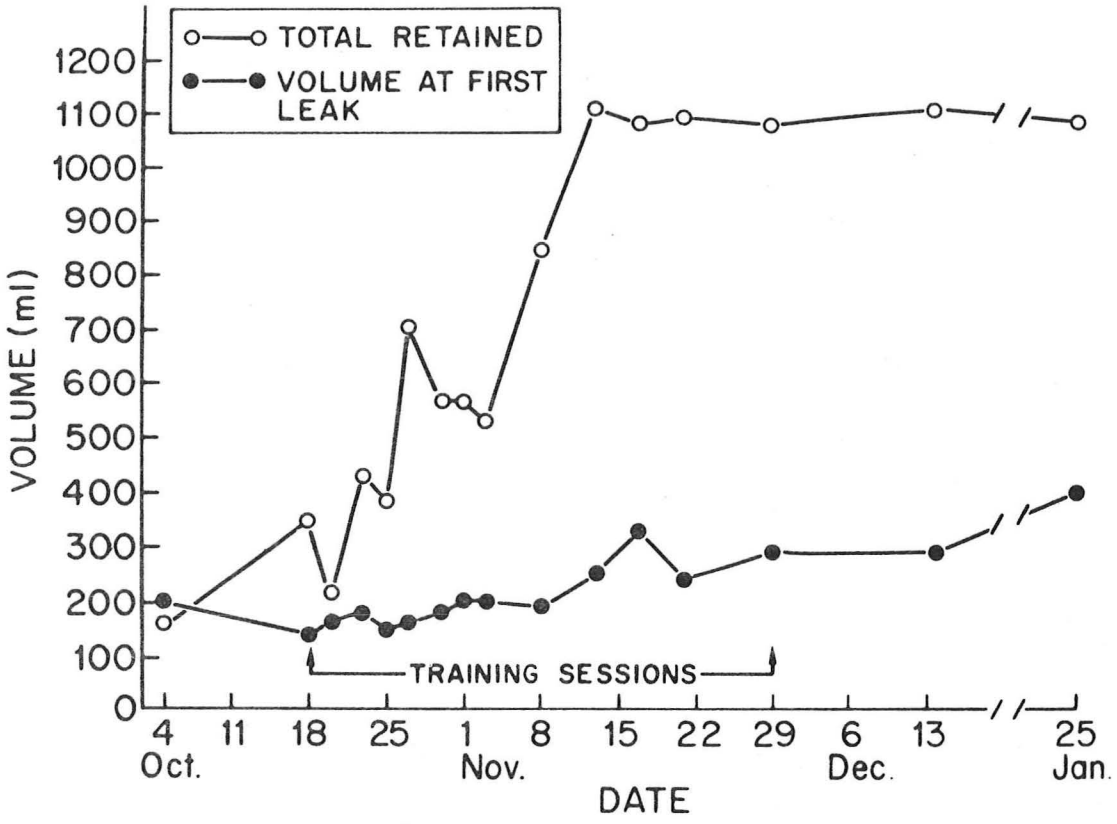

FIGURE 32. Response of Patient to Biofeedback Training with Saline (From Schiller et al, 1979).

Surgery. The large number of surgical procedures advocated for control of fecal incontinence gives indication of the lack of any single procedure that is effective in most patients (Corman, 1983). Methods used have ranged from electrical stimulation of sphincter muscle to implantation of an elastic band around the anal canal, from muscle transplantation to reconstruction of the pelvic floor. None of these procedures is easy or free of complications and they should always be done by colon and rectal surgeons expert in their application. A botched operation in this area can easily make a bad situation worse.

Electrical stimulation of the external anal sphincter by either implanted (Caldwell, 1963) electrodes or by electrodes attached to an intranal plug (Collins, Brown and Duthie, 1969; Glen, 1971) has been attempted for over 20 years with mixed results. Some patients treated with electrical stimulation appeared to regain anal tone and were able to stop using the equipment. Whether this was due to the electrical stimulation or represented spontaneous improvement is unknown.

More traditional operative approaches to fecal incontinence are listed in Table 8.

TABLE 8. SURGICAL TREATMENT OF FECAL INCONTINENCE

1. USE OF PELVIC FLOOR MUSCLES
   - Classic Repair (Apposition of Transected Sphincter Muscles)
   - Modified Classic Repair (Overlapping of Transected Sphincter Muscles)
   - Reefing (Plication) of Sphincter Muscles
   - Mobilization of Scar Tissue to Narrow the Anus
   - Use of Non-sphincteric Perineal Muscles
   - Puborectalis Repair (Post Anal Perineorrhaphy)
   - Pubococcygeus Repair
2. USE OF FLAPS OF OTHER MUSCLES
   - Gluteus
   - Adductor Longus
   - Gracilis
3. CIRCUMANAL FASCIAL STRIPS CONNECTED TO GLUTEAL MUSCLES
4. THIERSCH PROCEDURES (CIRCUMANAL WIRE, TEFLON, MARLEX, OR ELASTIC BAND)

Each of these can be complicated by infection and some experts routinely construct diverting colostomies prior to reconstruction. A problem with many of these operations is that the transposed muscles must be viable and their nerve supply must be intact for the results to be good. A careful and meticulous evaluation is therefore necessary before deciding on the operative approach to each individual. A detailed review of these operative procedures is beyond the scope of these Grand Rounds but the interested reader is referred to several recent reviews (Corman, 1983; Browning and Parks, 1983; Keighley and Fielding, 1983).

Collected series of what must be highly selected patients reveal good to excellent results with surgical procedures with success related to the ability of the surgeon to improve anal sphincter pressures or anatomy (Browning and Parks, 1983). Few long-term studies and no randomized comparative studies are available, so that the best operation is still a matter of conjecture. Still, it should be noted that there are operative alternatives to permanent colostomy for treating patients with fecal incontinence and these, perhaps, ought to be used more often.

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REFERENCES

1. Arhan, P., Devroede, G., Danis, K., Dornic, C., Faverdin, C., Persoz, B. & Pellerin, D. (1978) Viscoelastic properties of the rectal wall in Hirschsprung's disease. Journal of Clinical Investigation, 62, 82-87.

2. Arhan, P., Faverdin, C., Persoz, B., Devroede, G., Dubois, F., Dornic, C. & Pellerin, D. (1976) Relationship between viscoelastic properties of the rectum and anal pressure in man. Journal of Applied Physiology, 41, 677-682.

3. Bartolo, D.C.C., Jarratt, J.A. & Read, N.W. (1983a) The use of conventional electromyography to assess external sphincter neuropathy in man. Journal of Neurology, Neurosurgery and Psychiatry, 46, 1115-1118.

4. Bartolo, D.C.C., Jarratt, J.A. & Read, N.W. (1983b) The cutaneo-anal reflex: a useful index of neuropathy? British Journal of Surgery, 70, 660-663.

5. Bartolo, D.C.C., Jarratt, J.A., Read, M.A., Donnelly, T.C. & Read, N.W. (1983a) The role of partial denervation of the puborectalis in idiopathic faecal incontinence. British Journal of Surgery, 70, 664-667.

6. Bartolo, D.C.C., Read, N.W., Jarratt, J.A., Read, M.G., Donnelly, T.C. & Johnson, A.G. (1983b) Differences in anal sphincter function and clinical presentation in patients with pelvic floor descent. Gastroenterology, 85, 68-75.

7. Beber, C.R. (1980) Freedom for the incontinent. American Journal of Nursing, 80, 482-484.

8. Beersiek, F., Parks, A.G., & Swash, M. (1979) Pathogenesis of ano-rectal incontinence: a histometric study of the anal sphincter musculature. Journal of the Neurological Sciences, 42, 111-127.

9. Brocklehurst, J.C. (1975) Management of anal incontinence. Clinics in Gastroenterology, 4, 479-487.

10. Browning, G.G.P. & Parks, A.G. (1983) Postanal repair for neuropathic faecal incontinence: correlation of clinical result and anal canal pressures. British Journal of Surgery, 70, 101-104.

11. Burleigh, D.E., D'Mello, A. & Parks, A.G. (1979) Responses of isolated human internal anal sphincter to drugs and electrical field stimulation. Gastroenterology, 77, 484-490.

12. Caldwell, K.P.S. (1963) The electrical control of sphincter incompetence. Lancet, 2, 174-175.

13. Cerulli, M.A., Nikoomanesh, P. & Schuster, M.M. (1979) Progress in biofeedback conditioning for fecal incontinence. Gastroenterology, 76, 742-746.

14. Christensen, J. (1981) Motility of the colon. Chapter 14 in Physiology of the Gastrointestinal Tract (Ed.) Johnson, L.R. pp.445-471. New York: Raven Press.

15. Collins, C.D., Brown, B.H. & Duthie, H.L. (1969) An assessment of intraluminal electrical stimulation for anal incontinence. British Journal of Surgery, 56, 542-546.

16. Constantinides, C.B. & Cywes, S. (1983) Fecal incontinence: a simple pneumatic device for home biofeedback training. Journal of Pediatric Surgery, 18, 276-277.

17. Corman, M.L. (1983) The management of anal incontinence. Surgical Clinics of North America, 63, 177-192.

18. Davis, G.R., Santa Ana, C.A., Morawski, S.G. & Fordtran, J.S. (1980) Development of a lavage solution associated with minimal water and electrolyte absorption or secretion. Gastroenterology, 78, 991-995.

19. Denis, Ph., Colin, R., Galmiche, J.P., Geffroy, Y., Hecketsweiler, Ph., LeFrancois, R. & Pasquis, P. (1979) Elastic properties of the rectal wall in normal adults and in patients with ulcerative colitis. Gastroenterology, 77, 45-48.

20. Denny-Brown, D. & Robertson, E.G. (1935) An investigation of the nervous control of defaecation. Brain, 58, 256-310.

21. Devroede, G. (1982) Anal incontinence. Diseases of the Colon and Rectum, 25, 90-95.

22. Devroede, G., Vobecky, S., Masse, S., Arhan, P., Leger, C., Duguay, C. & Hemond, M. (1982) Ischemic fecal incontinence and rectal angina. Gastroenterology, 83, 970-980.

23. Dickinson, V.A. (1978) Maintenance of anal continence: a review of pelvic floor physiology. Gut, 19, 1163-1174.

24. Drossman, D.A., Sandler, R.S., McKee, D.C. & Lovitz, A.J. (1982) Bowel patterns among subjects not seeking health care. Gastroenterology, 83, 529-534.

25. Duthie, H.L. (1975) Dynamics of the rectum and anus. Clinics in Gastroenterology, 4, 467-477.

26. Duthie, H.L. (1982) Defaecation and the anal sphincter. Clinics in Gastroenterology, 11, 621-631.

27. Duthie, H.L. & Watts, J.M. (1965) Contribution of the external anal sphincter to the pressure zone in the anal canal. Gut, 6, 64-68.

28. Ehrman, J.S. (1983) Use of biofeedback to treat incontinence. Journal of the American Geriatrics Society, 31, 182-184.

29. Engel, B.T., Nikoomanesh, P. & Schuster, M.M. (1974) Operant conditioning of rectosphincteric responses in the treatment of fecal incontinence. New England Journal of Medicine, 290, 646-649.

30. Fay, D.F., Jones, N.B. & Porter, N.H. (1976) Spectral analysis of the myoelectric activity of the pelvic floor during voluntary contractions. Electromyography and Clinical Neurophysiology, 16, 525-551.

31. Feldman, M. & Schiller, L.R. (1983) Disorders of gastrointestinal motility associated with diabetes mellitus. Annals of Internal Medicine, 98, 378-384.

32. Frenckner, B. & Euler, C.V. (1975) Influence of pudendal block on the function of the anal sphincters. Gut, 16, 482-489.

33. Frenckner, B. & Ihre, T. (1976) Influence of autonomic nerves on the internal anal sphincter in man. Gut, 17, 306-312.

34. Garry, R.C. (1933) The responses to stimulation of the caudal end of the large bowel in the cat. Journal of Physiology, 78, 208-224.

35. Glen, E.S. (1971) Effective and safe control of incontinence by the intra-anal plug electrode. British Journal of Surgery, 58, 249-252.

36. Goldenberg, D.A., Hodges, K., Hersh, T. & Jinich, H. (1980) Biofeedback therapy for fecal incontinence. American Journal of Gastroenterology, 74, 342-345.

37. Hancock, B.D. (1981) Lord's procedure for hemorrhoids: a prospective anal pressure study. British Journal of Surgery, 68, 729-730.

38. Harford, W.V., Krejs, G.J., Santa Ana, C.A. & Fordtran, J.S. (1980) Acute effect of diphenoxylate with atropine (Lomotil) in patients with chronic diarrhea and fecal incontinence. Gastroenterology, 78, 440-443.

39. Haynes, W.G. & Read, N.W. (1982) Ano-rectal activity in man during rectal infusion of saline: a dynamic assessment of the anal continence mechanism. Journal of Physiology, 330, 45-56.

40. Henry, M.M., Parks, A.G. & Swash, M. (1980) The anal reflex in idiopathic faecal incontinence: an electrophysiological study. British Journal of Surgery, 67, 781-783.

41. Henry, M.M., Parks, A.G. & Swash, M. (1982) The pelvic floor musculature in the descending perineum syndrome. British Journal of Surgery, 69, 470-472.

42. Henry, M.M. & Swash, M. (1978) Assessment of pelvic-floor disorders and incontinence by electrophysiological recording of the anal reflex. Lancet, 1, 1290-1291.

43. Ihre, T. (1974) Studies on anal function in continent and incontinent patients. Scandinavian Journal of Gastroenterology, 9 (Suppl 25), 1-80.

44. Keighley, M.R.B. & Fielding, J.W.L. (1983) Management of faecal incontinence and results of surgical treatment. British Journal of Surgery, 70, 463-468.

45. Kiff, E.S. & Swash, M. (1984a) Normal proximal and delayed distal conduction in the pudendal nerves of patients with idiopathic (neurogenic) faecal incontinence. Journal of Neurology, Neurosurgery and Psychiatry, 47, 820-823.

46. Kiff, E.S. & Swash, M. (1984b) Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinece. British Journal of Surgery, 71, 614-616.

47. Krejs, G. & Fordtran, J.S. (1983) Diarrhea. Chapter 16 in Gastrointestinal Disease: Pathophysiology, Diagnosis, Management (Ed.) Sleisenger, M.H. & Fordtran, J.S. pp. 257-280. Philadelphia: W.B. Saunders Company.

48. Kuijpers, H.C. & Strijk, S.P. (1984) Diagnosis of disturbances of continence and defecation. Diseases of the Colon and Rectum, 27, 658-662.

49. Lawson, J.O.N. & Nixon, H.H. (1967) Anal canal pressures in the diagnosis of Hirschsprung's disease. Journal of Pediatric Surgery, 2, 544-552.

50. Leigh, R.J. & Turnberg, L.A. (1982) Faecal incontinence: the unvoiced symptom. Lancet, 1, 1349-1351.

51. MacLeod, J.H. (1983) Biofeedback in the management of partial anal incontinence. Diseases of the Colon and Rectum, 26, 244-246.

52. McLaren, S.M., McPherson, F.M., Sinclair, F. & Ballinger, B.R. (1981) Prevalence and severity of incontinence among hospitalized, female psychogeriatric patients. Health Bulletin, 39, 157-161.

53. McNeil, N.I. & Rampton, D.S. (1981) Is the rectum usually empty? - a quantitative study in subjects with and without diarrhea. Diseases of the Colon and Rectum, 24, 596-599.

54. Meunier, P. & Mollard, P. (1977) Control of the internal anal sphincter (manometric study with human subjects). Pflugers Archiv, 370, 233-239.

55. Millard, P.H. (1981) The prevention of incontinence. The Practitioner, 225, 1739-1743.

56. Misiewicz, J.J. (1984) Human colonic motility. Scandinavian Journal of Gastroenterology, 19 (Suppl 93), 43-51.

57. Morton, G. (1981) Management of the incontinent patient: practical guidelines for home care. Australian Family Physician, 10, 40-43.

58. Neill, M.E., Parks, A.G. & Swash, M. (1981) Physiological studies of the anal sphincter musculature in faecal incontinence and rectal prolapse. British Journal of Surgery, 68, 531-536.

59. Neill, M.E. & Swash, M. (1980) Increased motor unit fibre density in the external anal sphincter muscle in ano-rectal incontinence: a single fibre EMG study. Journal of Neurology, Neurosurgery and Psychiatry, 43, 343-347.

60. Parks, A.G., Fishlock, D.J., Cameron, J.D.H. & May, H. (1969) Preliminary investigation of the pharmacology of the human internal anal sphincter. Gut, 10, 674-677.

61. Pedersen, E., Klemar, B., Schroder, H.D.A.A. & Torring, J. (1982) Anal sphincter responses after perianal electrical stimulation. Journal of Neurology, Neurosurgery and Psychiatry, 45, 770-773.

62. Percy, J.P., Neill, M.E., Swash, M. & Parks, A.G. (1980) Electrophysiological study of motor nerve supply of pelvic floor. Lancet, 1, 16-17.

63. Phillips, S.F. & Edwards, D.A.W. (1965) Some aspects of anal continence and defaecation. Gut, 6, 396-406.

64. Preston, D.M., Lennard-Jones, J.E. & Thomas, B.M. (1984) The balloon proctogram. British Journal of Surgery, 71, 29-32.

65. Read, M.G. & Read, N.W. (1982) Role of anorectal sensation in preserving continence. Gut, 23, 345-347.

66. Read, M., Read, N.W., Barber, D.C. & Duthie, H.L. (1982) Effects of loperamide on anal sphincter function in patients complaining of chronic diarrhea with fecal incontinence and urgency. Digestive Diseases and Sciences, 27, 807-814.

67. Read, N.W., Bartolo, D.C.C. & Read, M.G. (1984) Differences in anal function in patients with incontinence to solids and in patients with incontinence to liquids. British Journal of Surgery, 71, 39-42.

68. Read, N.W., Harford, W.V., Schmulen, A.C., Read, M.G., Santa Ana, C. & Fordtran, J.S. (1979) A clinical study of patients with fecal incontinence and diarrhea. Gastroenterology, 76, 747-756.

69. Read, N.W., Haynes, W.G., Bartolo, D.C.C., Hall, J., Read, M.G., Donnelly, T.C. & Johnson, A.G. (1983) Use of anorectal manometry during rectal infusion of saline to investigate sphincter function in incontinent patients. Gastroenterology, 85, 105-113.

70. Sarna, S., Latimer, P., Campbell, D. & Waterfall, W.E. (1982) Electrical and contractile activities of the human rectosigmoid. Gut, 23, 698-705.

71. Scharli, A.F. & Kiesewetter, W.B. (1970) Defecation and continence: some new concepts. Diseases of the Colon and Rectum, 13, 81-107.

72. Schiller, L.R., Santa Ana, C., Davis, G.R. & Fordtran, J.S. (1979) Fecal incontinence in chronic diarrhea: report of a case with improvement after training with rectally-infused saline. Gastroenterology, 77, 751-753.

73. Schiller, L.R., Santa Ana, C.A., Schmulen, A.C., Hendler, R.S., Harford, W.V. & Fordtran, J.S. (1982) Pathogenesis of fecal incontinence in diabetes mellitus: evidence for internal-anal-sphincter dysfunction. New England Journal of Medicine, 307, 1666-1671.

74. Schnaufer, L., Talbert, J.L., Haller, J.A., Reid, N.C., Tobon, F. & Schuster, M.M. (1967) Differential sphincteric studies in the diagnosis of anorectal disorders of childhood. Journal of Pediatric Surgery, 2, 538-543.

75. Schuster, M.M. (1968) Motor action of rectum and anal sphincters in continence and defecation. Chapter 103 in Handbook of Physiology, Section 6, Volume IV. Motility (Ed.) Code, C.F. pp. 2121-2146. Washington: American Physiology Society.

76. Schuster, M.M. (1975) The riddle of the sphincters. Gastroenterology, 69, 249-262.

77. Schweiger, M. (1979) Method for determining individual contributions of voluntary and involuntary anal sphincters to resting tone. Diseases of the Colon and Rectum, 22, 415-416.

78. Shafik, A. (1975) New concept of the anatomy of the anal sphincter mechanism and the physiology of defecation. II. Anatomy of the levator ani muscle with special reference to puborectalis. Investigative Urology, 13, 175-182.

79. Smith, R.G. (1983) Fecal incontinence. Journal of the American Geriatrics Society, 31, 694-697.

80. Snooks, S., Henry, M.M. & Swash, M. (1984) Faecal incontinence after anal dilatation. British Journal of Surgery, 71, 617-618.

81. Snooks, S.J., Setchell, M., Swash, M. & Henry, M.M. (1984) Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet, 2, 546-550.

82. Suzuki, H. & Fujioka, M. (1982) Rectal pressure and rectal compliance in ulcerative colitis. Japanese Journal of Surgery, 12, 79-81.

83. Swash, M. (1982) Early and late components in the human anal reflex. Journal of Neurology, Neurosurgery and Psychiatry, 45, 767-769.

84. Tagart, R.E.B. (1966) The anal canal and rectum: their varying relationship and its effect on anal continence. Diseases of the Colon and Rectum, 9, 449-452.

85. Taylor, B.M., Beart, R.W., Jr. & Phillips, S.F. (1984) Longitudinal and radial variations of pressure in the human anal sphincter. Gastroenterology, 86, 693-697.

86. Thomas, T.M., Egan, M., Walgrove, A. & Meade, T.W. (1984) The prevalence of faecal and double incontinence. Community Medicine, 6, 216-220.

87. Thompson, W.G. & Heaton, K.W. (1980) Functional bowel disorders in apparently healthy people. Gastroenterology, 79, 283-288.

88. Ustach, T.J., Tobon, F., Hambrecht, T., Bass, D.D. & Schuster, M.M. (1970) Electrophysiological aspects of human sphincter function. Journal of Clinical Investigation, 49, 41-48.

89. Vereecken, R.L., Puers, B. & Van Mulders, J. (1982) Spectral analysis of perineal muscles EMG. Electromyography and Clinical Neurophysiology, 22, 321-326.

90. Wald, A. (1981) Biofeedback therapy for fecal incontinence. Annals of Internal Medicine, 95, 146-149.

91. Wald, A. & Tunuguntla, A.K. (1984) Anorectal sensorimotor dysfunction in fecal incontinence and diabetes mellitus: modification with biofeedback therapy. New England Journal of Medicine, 310, 1282-1287.

92. Whitehead, W.E., Orr, W.C., Engel, B.T. & Schuster, M.M. (1981) External anal sphincter response to rectal distention: learned response or reflex. Psychophysiology, 19, 57-62.

93. Wunderlich, M. & Parks, A.G. (1982) Physiology and pathophysiology of the anal sphincters. International Surgery, 67, 291-298.