Conferences and Reviews

The Pelvic Floor in Health and Disease

ANDREW A. SHELTON, MD *and* MARK L. WELTON, MD, *San Francisco, California*

Normal pelvic floor function involves a set of learned and reflex responses that are essential for the normal control and evacuation of stool. A variety of functional disturbances of the pelvic floor, including incontinence and constipation, are not life threatening, but can cause significant distress to affected patients. Understanding the normal anatomy and physiology of the pelvic floor is essential to understanding and treating these disorders of defecation. This article describes the normal function of the pelvic floor, the diagnostic tools available to investigate pelvic floor dysfunction, and the etiology, diagnosis, and management of the functional pelvic floor disorders that lead to incontinence and constipation.

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Normal Anatomy and Physiology

Pelvic Floor Anatomy

The pelvic floor is a consortium of funnel-shaped muscles that separates the pelvis and the perineum. It is composed of the *levator ani* and *puborectalis* muscles. The levator ani consists of two broad, thin, symmetric muscular sheets that originate around the pelvic sidewall and in the sacrospinous ligament that forms the principal support of the pelvic viscera. The puborectalis muscle originates in the posterior aspect of the pubis, forms a sling around the rectum, and returns to the posterior aspect of the pubis. The fibers of the puborectalis are situated immediately adjacent to and below the innermost component of the levator ani muscle, where they are intimately associated with the upper posterolateral fibers of the deep external anal sphincter. Thus, the puborectalis serves as a bridge between the broad sheetlike component of the funnel created by the levator and the narrow spout of the funnel created by the external anal sphincter (Figure 1). The puborectalis in the contracted state is responsible for the normal acute anorectal angle between the levators and the external sphincters. It is also responsible for the shelf that is normally palpable between the anal canal and the rectum on digital rectal exam.[1–3] The innervation of the muscles of the pelvic floor is from branches of ventral nerve roots of S2–S4. These originate proximal to the formation of the sacral plexus and run on the inner surface of the levator ani muscles.[4]

The anal canal is the closed slit-like lowermost portion of the digestive tract. It is encircled by the internal and external sphincter muscles which, in the resting state, keep the anus closed. From its junction with the rectum, it runs downward and backward. Two different definitions of the anal canal are used, the anatomic anal canal and the surgical anal canal. The anatomic anal canal is approximately 2 cm long and extends from the dentate line to the anal verge. The surgical anal canal is approximately 2 to 5 cm long and extends from the anorectal ring created by the puborectalis at the apex of the pelvic floor to the anal verge. The surgical anal canal is important in normal anorectal function and is considered the physiologic anal canal.[3] The anal canal high-pressure zone is generated by the internal and external anal sphincters at rest and constitutes the physiologic sphincter.

The anal canal is maintained in a closed slit-like conformation by the internal and external anal sphincter. The internal anal sphincter is a condensation of the inner circular muscle of the rectum. It is about 2.5 to 4 cm long and 0.5 cm thick. It ends about 1 to 1.5 cm distal to the dentate line, proximal to the end of the external anal sphincter.[5] The internal anal sphincter is composed of smooth muscle and is innervated by the autonomic nervous system. It receives excitatory sympathetic innervation via the hypogastric nerves (L5) and inhibitory parasympathetic innervation by pelvic splanchnic nerves (S2–S4).[6,7]

The external anal sphincter forms a circular cuff around the internal anal sphincter. It is slightly longer than the internal anal sphincter. It extends from the puborectalis (from which its most cranial fibers are indistinguishable) to the anal verge, where it becomes subcutaneous. The external anal sphincter is 6 to 10 mm thick.[8] The exact anatomy of the external anal sphincter is a matter of some debate. It has been viewed as a triple loop system with deep, superficial, and subcutaneous loops. The deep loop represents the puborectalis muscle in close association with the most cranial fibers of the external sphincter. The superficial component encircles

From the Department of Surgery, University of California San Francisco School of Medicine, San Francisco, California.
Address reprint requests to Mark L. Welton, MD, 505 Parnassus Ave, Box 0144, M-887, San Francisco, CA 94143-0144.

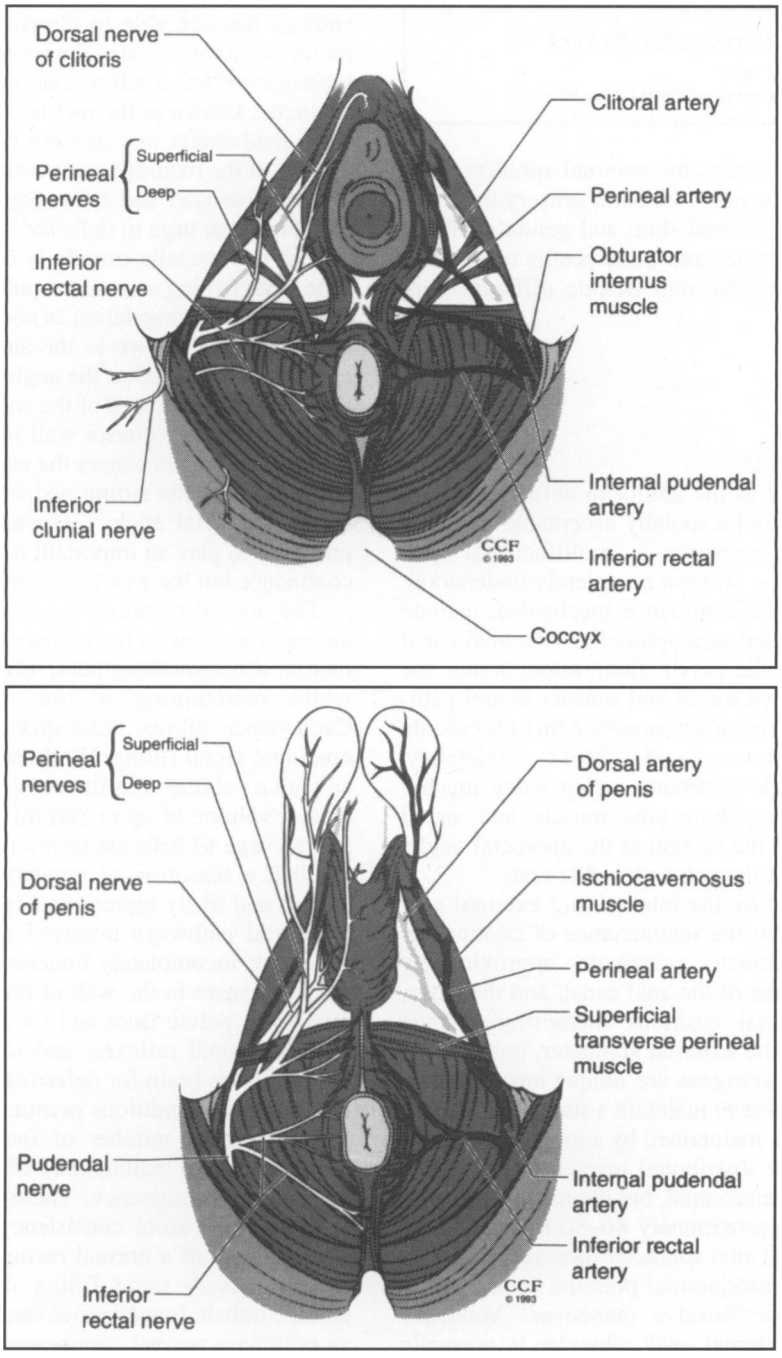

Figure 1.—Perineum superficial deep of a woman (upper panel) and a man (lower panel). Branches of the pudendal nerve are depicted on the left (white), and the pudendal artery and its branches are depicted on the right. Reprinted, with permission, from *Surgery of the Colon, Rectum, and Anus* (Mazier WP, et al., Eds; Philadelphia: Saunders, 1995, p 31).

the midportion of the anal canal and inserts posteriorly at the anococcygeal ligament and coccyx. The subcutaneous loop encircles the most distal anal canal and inserts anteriorly at the perianal skin.[9] Some experts believe that the external sphincter cannot be divided into three different loops and represents a single homologous muscle[10,11] or a dual system with a superficial and deep component only.[12] The external anal sphincter is composed of striated skeletal muscle and is innervated by the pudendal nerve (S2–S4).[4] The inferior rectal branch of

ABBREVIATIONS USED IN TEXT
EMG = electromyography
PNTML = pudendal nerve terminal motor latency

the pudendal nerve supplies the external sphincter, and afferent branches of the nerve transmit sensory impulses from the anal canal, perianal skin, and genitalia. Thus, the external anal sphincter and puborectalis muscle act as a functional, indivisible unit despite differences in their innervation.

Continence

Pelvic Floor Physiology

Continence is defined as the ability to defer defecation or passage of flatus until a socially acceptable time and place. Control of continence is multifactorial and, despite intense study, is still not completely understood. The components of the continence mechanism include the internal and external anal sphincters (the anal canal high-pressure zone), the pelvic floor musculature, the rectum, and interrelated motor and sensory neural pathways. Other factors affecting continence include colonic motility, stool volume, and stool consistency. Additionally, although in debate, a flap valve mechanism created by the puborectalis muscle and sacral attachments close off the rectum at the anorectal angle. This angulation keeps the valve closed at rest.

Pressure generated by the internal and external anal sphincters is critical to the maintenance of continence. The internal anal sphincter contributes approximately 85% of the resting tone of the anal canal, and the external sphincter and anal cushions (hemorrhoids), the remaining 15%.[13-15] The external sphincter, pelvic floor muscles, and cricopharyngeus are unique among skeletal muscles in being able to maintain a state of tonic contraction.[16] This tone is maintained by a low sacral reflex. Resting pressures are distributed unevenly around the circumference of the anal canal, but the mean anal canal resting pressure is approximately 40–80 mmHg. Tonic activity of the external anal sphincter increases with factors that increase intraabdominal pressure such as erect posture, coughing, or Valsalva maneuver.[3] Voluntary contraction of the external anal sphincter is normally able to approximately double the resting pressure in the anal canal.[17] The external sphincter fatigues after 3 minutes of sustained voluntary contraction.[18]

Distension of the rectum with gas or stool stimulates receptors located in the pelvic floor, causing a reflex receptive relaxation of the internal anal sphincter. This reflex is known as the rectoanal inhibitory reflex. It has been shown to occur up to seven times a day.[20] Relaxation of the internal anal sphincter lowers anal canal pressure, allowing rectal contents to come in contact with the mucosa of the upper anal canal in the anal transition zone. The mucosa of the anal transition zone is richly innervated with both free and organized nerve endings that are able to discriminate the nature of the rectal contents.[21] Simultaneous with the rectoanal inhibitory reflex, a reflex contraction of the external anal sphincter, known as the rectoanal contractile reflex, prevents inadvertent loss of stool or gas.[22] Progressive distension of the rectum causes continuous inhibition of the internal sphincter and relaxation of the external sphincter causing an urge to defecate.[18]

The puborectalis muscle is contracted in the resting state, maintaining an anterior pull on the intestine, resulting in an acute angulation of the junction of the rectum and anal canal known as the anorectal angle. It is radiographically defined as the angle formed by a line drawn along the posterior wall of the anal canal and a line drawn along the distal posterior wall of the rectum. Squeezing the anal sphincters causes the anorectal angle to become more acute, while sitting and squatting leads to a more obtuse anorectal angle.[23] The anorectal angle has been proposed to play an important role in the maintenance of continence but the exact mechanism is disputed.[24,25]

The normal rectum is a capacitance organ and plays an important role in the maintenance of continence. The rectum accommodates passively to increases in volume while maintaining a low intraluminal pressure. Capacitance allows defecation to be deferred during continual rectal filling. While rectal filling is appreciated with a volume of as little as 10 ml, the rectum can tolerate a volume of up to 300 ml until a sense of fullness and an urge to defecate develops. The volume required to elicit a sensation of urgency varies from patient to patient and likely represents a learned response.

Neural pathways involved in the control of continence are incompletely understood. They include local nerve plexuses in the wall of the rectum, sensory receptors in the pelvic floor and the lining of the anal canal, regional spinal reflexes, and ultimately higher control centers in the brain for deferring defecation until socially acceptable conditions permit.

There are a number of factors that, although not important in the maintenance of continence, can significantly affect continence. These include such factors as stool volume, stool consistency, and colonic motility. The capacity of a normal rectum may be overwhelmed by excessively rapid filling due to excessively fast colonic transit. Incontinence can result in the presence of an otherwise normal continence mechanism in the presence of, and excessive volume of, liquid stool.

Defecation

Normal defecation occurs when peristalsis propels a fecal bolus from the sigmoid colon into the rectum. Distension of the rectum initiates the rectoanal inhibitory reflex causing relaxation of the internal anal sphincter, allowing the rectal contents to come in contact with the sensory receptor–rich anal transition zone, which in turn allows discrimination between solid stool, liquid stool, and gas (sampling reflex). At the same time, the rectoanal excitatory reflex contracts the external anal

sphincter, preventing loss of stool. The integrated neural pathways activate higher cortical centers, and a determination is made about whether it is a socially acceptable time and place to defecate. If it is, the person assumes a sitting position on the toilet which straightens the anorectal angle. Straining is associated with a reflex relaxation of the puborectalis muscle and external anal sphincter. This allows descent of the pelvic floor, further straightening the anorectal angle. Thus, the anal canal shortens and becomes more funnel-shaped as it begins to open. Rectal evacuation is then primarily accomplished by increasing intraabdominal pressure due to contraction of the abdominal muscles, the diaphragm, and performance of a Valsalva maneuver. After complete evacuation, sphincter pressures return to normal, the puborectalis muscle returns to its normal state, and the anorectal junction resumes its normal acute angulation. Although lacking normal sensation, patients with spinal cord injuries are able to initiate the reflex mechanisms involved in normal defecation by digital stimulation, and thus initiate a bowel movement through the integrated neural pathways mentioned above.

Investigative Tools

Patients can present with a variety of complaints referable to abnormal pelvic floor function. In addition to a accurate history and physical examination, there are a number of objective tests available to evaluate the complaints and come to an appropriate diagnosis.

Anorectal Manometry

Anorectal manometry measures intraluminal pressures generated by the internal and external anal sphincters. Use of a thin, flexible, hydraulically perfused polyvinyl catheter with radially located channels is the most common method. The catheter is connected to a pressure transducer which is linked to a polygraph to provide a continuous tracing. The examination is typically performed in the left lateral decubitus position. The catheter is inserted approximately 8 cm into the anal canal, and the rectal pressure is recorded. The catheter is then slowly withdrawn until a rise is noted. This is the beginning of the anal canal high pressure zone. The distance from the anal verge is recorded, allowing determination of the length of the anal canal. The maximum resting pressure is the maximum pressure generated in the anal canal with the patient at rest. The internal anal sphincter is responsible for 80–85% of the resting tone, with the remainder due to tonic action of the external anal sphincter. Maximum voluntary contraction is determined by asking the patient to squeeze and represents action of the external anal sphincter. In patients who are unable to squeeze in a laboratory setting, the pressure generated during a cough usually accurately reflects squeeze pressure. The presence of the rectoanal inhibitory reflex is determined by inflating a balloon in the rectum while simultaneously measuring intraluminal pressure in the anal canal. The expected response is a decrease in the resting tone of at least 50%.

Absolute manometric values overlap considerably between "normal" patients and those with disorders of defecation. The values are useful, however, when taken in the clinical context. They are also useful in predicting and measuring responses to therapy.

Rectal Sensation

Rectal sensation can be determined by inflating an intrarectal balloon with increasing amounts of air. The balloon is serially distended with 10-ml volumes of air. The initial sensation is usually felt at about 20 ml. Recorded values include volume necessary to cause first sensation, volume necessary to cause urge to defecate, and maximum tolerable volume.[26] Rectal compliance can be measured by measuring the pressure in the distending balloon.

Abnormalities of rectal sensation are seen in both constipated and incontinent patients. Patients may have severe constipation and lack the urge to defecate due to an inability to sense stool in the rectal vault. Conversely, other patients with abnormal rectal sensation present with incontinence secondary to inability to sense stool in the rectum.

Defecography

Rectal evacuation requires coordinated activity or inactivity of the abdominal muscles, internal anal sphincter, external anal sphincter, and pelvic floor. The dynamics of defecation can be assessed radiographically using cinedefecography.[27] The patient is placed on a radiolucent commode and the rectum filled with a thick barium paste. The patient is then asked to squeeze, cough, strain, and then empty the rectum while lateral fluoroscopic views of the pelvis are obtained. Static images are useful for determining length of the anal canal; the anorectal angle at rest, during squeezing, and during evacuation; and degree of perineal descent.

Defecography qualitatively assesses puborectalis function and adequacy of rectal emptying. It also permits real-time visualization of rectocele, enterocele, and internal rectal intussusception. A paradoxical contraction of the puborectalis muscle, leading to obstructive defecation, is identified as *anismus*.

Electromyography

Electromyography (EMG) of the external anal sphincter and pelvic floor can be performed using either needle or surface electrodes.[28] Needle EMG can be either concentric needle EMG or single-fiber EMG. Concentric needle EMG records the summed electrical activity of approximately thirty motor units. It has been used primarily to map sphincter defects, such as after obstetrical injury. Single-fiber EMG uses a fine, 25-μm electrode. It is able to record the action potential from a single muscle fiber and is able to quantify fiber density. An increase in fiber density occurs with denervation due to sprouting from neighboring nerve fibers. Single-fiber

EMG can also be used to measure neuromuscular "jitter," which is an electromyographic indication of denervation and reinnervation.[29] EMG has the disadvantage of being uncomfortable for the patients, and it requires considerable technical expertise on the part of the examiner.

Ultrasound has largely replaced EMG for the mapping of sphincter defects, although EMG is still useful in the diagnosis of neurogenic fecal incontinence and in the demonstration of the abnormal recruitment of muscle fibers in anismus or the nonrelaxing puborectalis syndrome.

Pudendal Nerve Terminal Motor Latency

Pudendal neuropathy plays a role in the pathogenesis of idiopathic fecal incontinence. It is possible to diagnose injury to the pudendal nerve by measuring nerve conduction velocity. This is accomplished by digital rectal examination using a special glove-mounted electrode. The ischial spine is palpated, and the pudendal nerve is stimulated in Alcock's canal. The evoked electrical response in the external anal sphincter is recorded in an electrode at the base of the finger. The time from the stimulus of the pudendal nerve at the ischial spine to the recording of electrical activity of the external anal sphincter represents the pudendal nerve terminal motor latency (PNTML). The normal PNTML is 1.9 ± 0.2 seconds.[30] Any nerve injury will give a prolonged PNTML.

Measurement of PNTML is primarily used to diagnose pudendal neuropathy and neurogenic fecal incontinence. It is useful in predicting which patients will benefit from biofeedback and surgical intervention.

Ultrasonography

An ultrasound examination of the anal canal using a rigid, rotating 7.5 or 10.0 MHz transducer is able to define accurately the anatomy of the anal canal. It is able to measure the thickness of and define defects in the internal anal sphincter. It is also able to identify defects in the external anal sphincter and is much more comfortable than EMG.[31]

Ultrasound has broad applications in the diagnosis of many anorectal conditions. Incontinent patients, especially those who have had prior anorectal surgery or those in whom an obstetric injury is suspected, should have an ultrasound to rule out a sphincter defect that may be amenable to surgical correction.

Pelvic Floor Dysfunction

Neurogenic Incontinence

Fecal incontinence is defined as the inability to defer defecation to an appropriate time or place. It has been shown to occur in more than 2% of the general population. Many people are reluctant to admit the problem because of embarrassment, and thus the true incidence may be higher.[32] Fecal incontinence can be classified as traumatic (obstetric injury, anorectal surgery), neurologic, congenital, or idiopathic. Idiopathic fecal incontinence is generally believed to be due to denervation of the pelvic floor and external anal sphincter due to stretch injury to the pudendal nerve.

It is theorized that the terminal portion of the pudendal nerve is susceptible to stretch injury due to descent of the pelvic floor. This is seen in conditions such as chronic straining, descending perineum syndrome, intussusception, or prolapse, or after difficult vaginal delivery. Stretch injury to a peripheral nerve occurs when it is stretched more than 12% beyond its original length. It has been calculated that the terminal portion of the pudendal nerve is stretched 20–30% with an average descent of only 2 to 3 cm (normal descent up to 4 cm).[31] The fast conducting myelinated fibers of the pudendal nerve are those susceptible to stretch injury. Some stretch injury is reversible, but the degree or amount of stretch necessary to cause irreversible damage is not known.

A study of the effect of vaginal deliveries on pelvic floor musculature found that 80% of primagravida sustained reversible pudendal nerve damage.[33] Nerve injury may be unilateral or bilateral.[30] The end result is a denervation and weakening of the pelvic floor and external anal sphincter. As the pelvic floor weakens, it is less able to withstand increased intraabdominal pressure, resulting in further perineal descent. The neuropathy and sphincter dysfunction progress with time.[34]

The descending perineum syndrome is a relatively common disorder, primarily in women. The most pronounced feature is difficulty with defecation with repeated efforts of unsuccessful straining and a sense of incomplete evacuation. Straining leads to descent of the pelvic floor and straightening of the anorectal angle. This leads to prolapse of the anterior rectal wall into the rectal lumen which can obstruct defecation, setting up a vicious cycle with further straining. The end result is often further pelvic descent, stretching of the pudendal nerve, denervation of the pelvic floor and external anal sphincter, and finally, neurogenic fecal incontinence, sometimes accompanied by internal rectal prolapse (intussusception).[54]

Although stretch injury to the pudendal nerve is a common cause of neurogenic fecal incontinence, any neurologic disease may affect bowel control. Diabetes is a common condition that may cause diarrhea or produce a peripheral neuropathy, both of which may lead to incontinence.

Fecal incontinence is an underdiagnosed condition because patients are reluctant to discuss it with their physicians. Except in the most minor cases, patients with fecal incontinence should be referred to a colorectal surgeon or gastroenterologist for full evaluation. The diagnosis of incontinence due to pudendal nerve injury begins with a careful history detailing the patient's bowel habits, generalized pelvic floor function, and obstetrical history (Figure 2). In particular, the character (gas, liquid, or solid stool) and frequency of the incontinent episodes must be elicited, as well as a preceding history of chronic straining at stool. Associated uterine prolapse or stress urinary incontinence suggests generalized pelvic floor

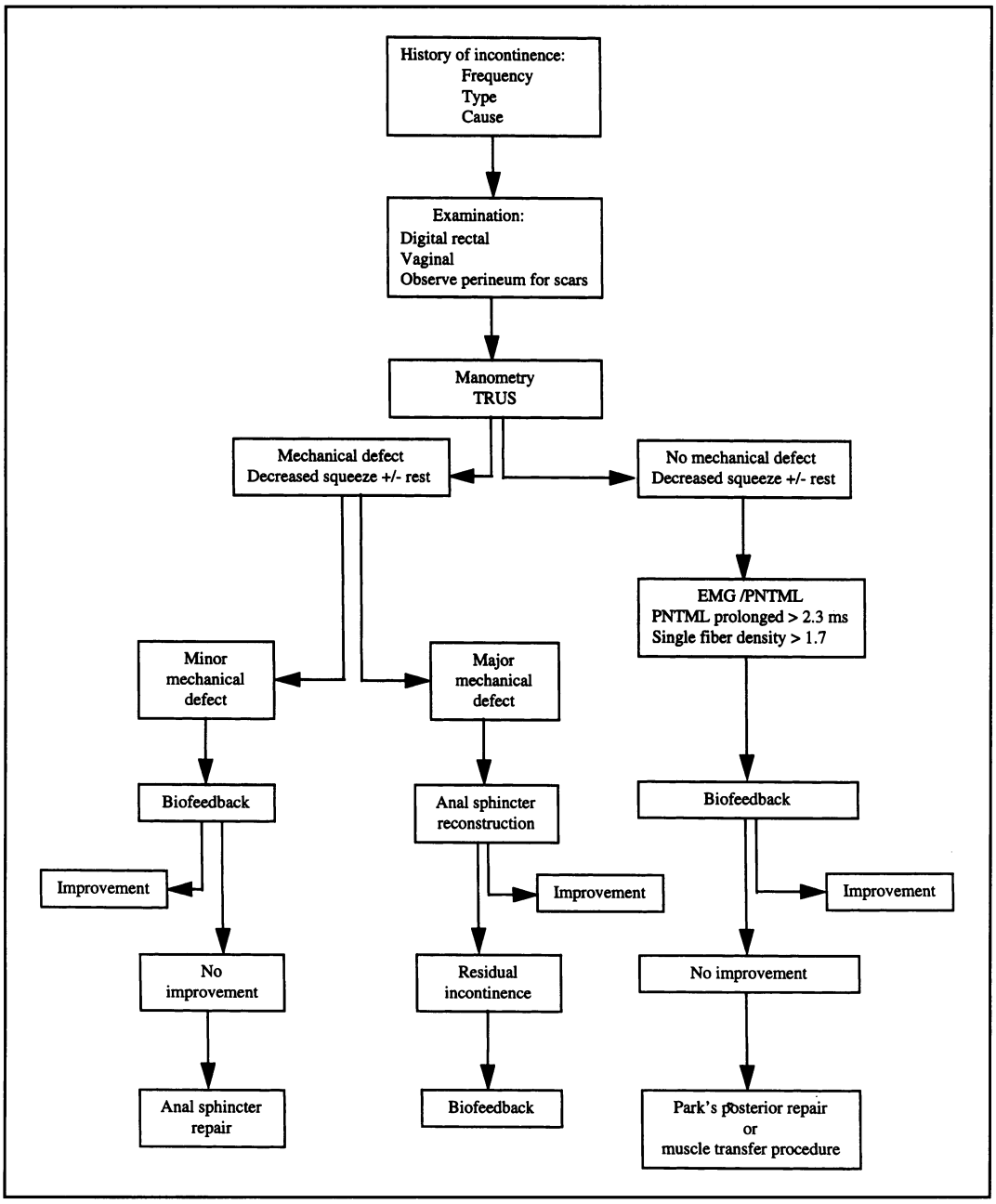

Figure 2.—Algorithm for work-up and treatment of fecal incontinence.

weakness. Finally, history of a prolonged and difficult second stage of labor, forceps delivery, or high birthweight also suggests nerve injury. Physical examination typically reveals a patulous anus and flattening of the perineum. Straining generally causes abnormal perineal descent. There may be altered perianal sensation and absence of the anocutaneous reflex. Digital rectal examination reveals weakened resting tone and decreased ability to augment pressure with squeeze. Manometry may be normal or reveal decreased resting and squeeze pressures.[35] Defecography may show a widened (more obtuse) anorectal angle and abnormal perineal descent on straining. However, the diagnosis of incontinence due to pudendal neuropathy rests on showing prolonged pudendal nerve terminal motor latency or evidence of denervation and reinnervation on EMG. A PNTML of greater than 2.2 seconds indicates pudendal nerve injury. This can be confirmed by finding a single fiber density greater than 1.66 using single-fiber EMG.[36] Ultrasonography may reveal an unsuspected sphincter defect.[28]

Management of patients with neurogenic fecal incontinence due to pudendal neuropathy often begins with

long trials of conservative medical measures. Changing stool consistency from liquid to solid may significantly decrease the number of incontinent episodes in patients with marginal sphincter function. Lomotil, loperamide, and codeine achieve this change in stool consistency by delaying colonic transit allowing increased absorption of water. Bulking agents such as psyllium bind water and thicken the stool. Immodium also tends to increase the resting tone of the anus.

Biofeedback is a technique that translates a physiologic function into an auditory or visual stimulus, thus allowing the patient to learn to control the physiologic function. During training, intraanal pressure or external sphincter myoelectric activity is transformed into an acoustic or visual signal which is fed back to the patient. The patient uses these signals to develop improved control of the sphincter function. Sensory function may also be improved by inflating a balloon in the rectum and training the patient to detect decreasing volumes in coordination with contraction of the external sphincter. Studies have shown a reduction of incontinent episodes in up to two-thirds of patients,[17] but objective improvement, such as increase in resting or squeeze pressure, is lacking.[37]

For patients with more severe neurogenic incontinence, or those who have failed nonoperative measures, there are several surgical options available, some of which are still experimental. One option is the postanal repair. The posterior aspect of the external sphincter, puborectalis, and levator ani are plicated in an attempt to restore the normal anorectal angle and lengthen the anal canal.[38] Although improvement was shown in 80% of patients in early studies, more recent studies have been less promising. Therefore, this operation is rarely performed in the US today.[39,40] Postanal repair generally is a reefing of external sphincter and does not involve an anterior approach, which may be confined to reef the external sphincter anteriorly and plicate the puborectal anterior. This aids in increasing passive resistance and active contraction. This repair may also be of benefit in restoring normal anal distance from the vagina when the anus is ectopically displaced anteriorly. The remaining restorative options attempt to create a controllable anal sphincter. A neosphincter using skeletal muscles based on its own neurovascular pedicle is one such option. Muscle, either the gracilis or gluteus maximus, is mobilized and wrapped snugly around the anus, and the tendon is sutured to the contralateral ischial tuberosity. Chronic electrical stimulation of the muscle graft with an implantable pulse generator converts the muscle fibers from fatigable, fast-twitch to fatigue-resistant slow-twitch muscle fibers that are capable of maintaining the prolonged contractions necessary for continence. Although this is a technically difficult operation, results using this technique have been promising.[41] An experimental, implantable, artificial anal sphincter composed of three silastic components (an inflatable cuff, a pressure-regulating balloon, and a control pump) has been developed. The inflatable cuff is implanted around the anus and is connected to the control pump situated in the scrotum or labia. The control pump is connected to the pressure-regulating balloon and is placed in the preperitoneal space behind the pubic bone. In its activated state, the cuff is inflated with fluid, the anus is occluded, and continence is maintained. When the patient experiences an urge to defecate, the cuff is deflated by pumping the control pump. In two recent studies, a successful outcome was achieved in 75–90% of patients.[42,43] Implantable neuroprostheses to directly stimulate the sacral nerve roots, pudendal nerve, or external sphincter itself to achieve continence are techniques currently under investigation. Sacral nerve root stimulation has shown promise in the treatment of urinary incontinence.[44] A diverting colostomy is the final surgical option for patients with ongoing significant incontinence despite medical treatment, biofeedback, or surgical intervention.

Nonrelaxing Puborectalis Syndrome

As discussed previously, normal defecation requires a series of coordinated actions of the pelvic floor and sphincter muscles. Obstructed defecation is a form of chronic functional constipation due to functional abnormalities of the pelvic floor and anal canal musculature leading to pelvic outlet obstruction. Obstructed defecation may be due to a variety of causes including rectocele, rectal prolapse, and aganglionosis of the anorectal junction (adult Hirschprung's disease). The most common cause, however, is anismus, or the nonrelaxing puborectalis syndrome.[45–48] The cause of the syndrome is unknown, but the main physiological abnormality is thought to be due to failure of the pelvic floor muscles to relax during defecation. This prevents straightening of the anorectal angle.[49] Thus, the patient strains against an acute anorectal angle which obstructs normal defecation. Anismus is thought to be an abnormal learned response. In some patients, it represents a reaction to sexual assault. Ironically, the end result of long-term anismus is often neurogenic incontinence as a consequence of chronic straining with resultant perineal descent and resultant stretch injury to the pudendal nerve.

Constipation is a very common condition, and most patients with a complaint of infrequent or difficult bowel movements do not require extensive work-up. A thorough history will often uncover a change in diet, medication or physical activity that is easily corrected. The physical examination should include a complete visual, digital, anoscopic, and sigmoidoscopic evaluation to rule out common and potentially serious causes of constipation such as anal stenosis, anal fissure, distal colorectal malignancies, extrinsic pelvic massess, and ischemic or inflammatory strictures of the rectosigmoid. When these causes of constipation are ruled out, conservative management consists of a high-fiber diet and increased fluid intake. If this fails, referral to rule out colonic inertia or anismus is indicated. Patients with anismus typically present with complaints of months to years of difficult evacuation with many unsuccessful calls to stool (Figure 3). They often spend minutes to

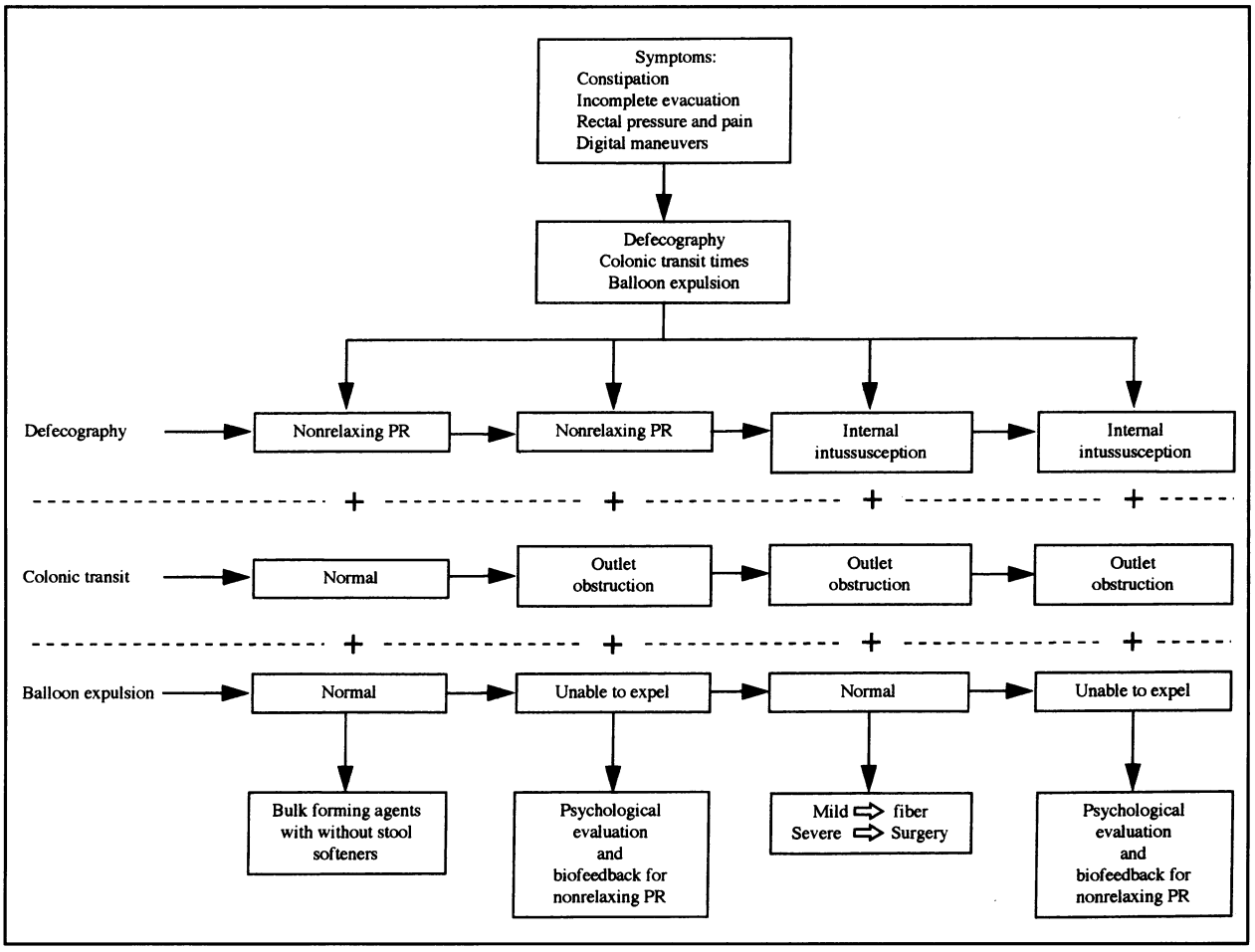

Figure 3.—Algorithm for work-up and treatment of obstructed defecation.

hours on the toilet without results. They complain of an inability to initiate defecation, a sense of incomplete evacuation, and pelvic or perianal pain. Physical examination is often normal, but may reveal abnormal perineal descent or a persistent, palpable puborectalis muscle posteriorly on straining. There is no consistent characteristic abnormality of the internal or external sphincters on anorectal manometry.[50] Defecography and EMG demonstrate absence of relaxation, or paradoxical contraction, of the puborectalis muscle during attempts to defecate. Defecography may also reveal a poorly emptying rectocele or internal intussusception, both of which lead to a sense of obstructed defecation. Needle electromyography of the pelvic floor can be painful, but surface electromyography using an anal plug can also demonstrate increased activity in the puborectalis muscle and external sphincter during straining to defecate. A useful comfirmatory test for the diagnosis of anismus or nonrelaxing puborectalis is the ability to expel an air-filled 60-ml balloon catheter. Failure of the patient to expel the balloon is highly suggestive of anismus.

Early surgical attempts to treat anismus by partial division of the puborectalis muscle have proven unsatisfactory.[51] Partial paralysis of the puborectalis muscle by injection of botulinum toxin has also proven unsuccessful.[52] Treatment, therefore, is primarily nonoperative. Patients are placed on a high-fiber diet coupled with adequate hydration. Patients with documented nonrelaxing puborectalis demonstrated by either defecography or balloon expulsion test should be offered biofeedback. Using the same biofeedback techniques described for the management of incontinence, the patient is taught to strain without contracting the puborectalis muscle. In our experience in patients without other ongoing psychological issues, biofeedback has been 80% effective in treating anismus, while others have reported that up to 90% of the patients improve.[53]

Conclusions

The normal coordinated actions of the pelvic floor are often taken for granted. However, functional disturbances of the pelvic floor leading to disturbances in defecation can be quite distressing. Fecal incontinence is an

embarrassing condition that often leads to self-imposed social isolation and is a major health care problem in that it is a leading cause for nursing home admissions. Severe constipation may lead to frustrating hours straining on the toilet, debilitating abdominal pain, and multiple visits to primary caretakers, gastroenterologists, and surgeons. These functional disorders are readily diagnosed by history and a variety of diagnostic tests available in a well-equipped anorectal physiology laboratory. Neurogenic fecal incontinence and obstructed defecation due to a nonrelaxing puborectalis are usually readily managed with medical means and biofeedback. A number of surgical options are available for the incontinent patient who fails these less invasive treatments.

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