Pelvic floor failure is a common disorder that can seriously jeopardize a woman’s quality of life by causing urinary and fecal incontinence, difficult defecation, and pelvic pain. Multiple congenital and acquired risk factors are associated with pelvic floor failure, including altered collagen metabolism, female sex, vaginal delivery, menopause, and advanced age. A complex variety of fascial and muscular lesions that range from stretching, insertion detachment, denervation atrophy, and combinations of pelvic floor relaxation to pelvic organ prolapse may manifest in a single patient. Thorough preoperative assessment of pelvic floor failure is necessary to reduce the rate of relapse, which is reported to be as high as 30%. Magnetic resonance (MR) imaging of the pelvic floor is a two-step process that includes analysis of anatomic damage on axial fast spin-echo (FSE) T2-weighted images and functional evaluation using sagittal dynamic single-shot T2-weighted sequences during straining and defecation. This article presents high-resolution FSE T2-weighted MR images that permit detailed assessment of anatomic lesions and briefly describes pelvic floor pathophysiology, associated clinical symptoms, and patterns of dysfunction seen with dynamic MR imaging sequences. MR imaging is a powerful tool that enables radiologists to comprehensively evaluate pelvic anatomic and functional abnormalities, thus helping surgeons provide appropriate treatment and avoid repeat operations.

Introduction

Pelvic floor failure is a common disorder that affects 23.7% of women in the United States, with a prevalence of 9.7%–49.7% that increases with age (1). One in nine women will undergo an invasive procedure for treatment of urinary incontinence or pelvic organ prolapse, with 30% requiring additional surgery for symptom recurrence by 80 years of age (2). Furthermore, the prevalence of pelvic floor disorders is predicted to increase substantially given the changing demographics of Western countries (3). Pelvic floor dysfunction may manifest with urinary tract symptoms (stress and urge incontinence, straining to void, and positional voiding maneuvers), sexual symptoms (difficult intercourse and dyspareunia), and bowel symptoms (fecal incontinence, difficult defecation, and rectal prolapse). General symptoms include pelvic pain and discomfort (4).
The mechanisms involved in pelvic floor failure are only partially understood. Multiple congenital and acquired factors may exist in a single patient (Fig 1). Urinary incontinence and pelvic organ prolapse show a genetic predisposition to abnormal extracellular matrix remodeling due to altered collagen and elastin metabolism (5). Female sex, menopause, and advanced age are the main risk factors; obesity, pregnancy, and parity cause increased abdominal pressure and reduced muscle tone. Vaginal delivery can be associated with various muscular, ligamentous, and osseous lesions, including muscle tearing and edema, bone marrow edema, and pubic bone fracture (6). Chronic straining due to constipation or chronic obstructive pulmonary disease may cause repetitive trauma to the pudendal nerve and lead to pudendal neuropathy (7). Previous pelvic surgery, especially hysterectomy, may result in anatomic damage with disruption of the pubovesical and rectovaginal fasciae. A recent study has identified an oversized pelvic outlet as an additional risk factor (8).

Evaluation of women with pelvic floor failure requires a comprehensive approach that includes clinical assessment, physiologic testing, and counseling about conservative versus surgical treatment. Clinical evaluation based on detailed physical, neurologic, and digital rectal examination is the cornerstone of diagnosis. However, clinical examination is limited in several ways: (a) it can lead to underestimating or misdiagnosing the site of prolapse; (b) it does not permit assessment of evacuation disorders; and (c) it cannot detect a peritoneocele, a finding that indicates the need for abdominal rather than vaginal surgery. Complementary physiologic testing (eg, urodynamic evaluation, anorectal manometry, and electromyography) (9) is invaluable for assessing pathophysiology (smooth and striate muscle function and sensitivity impairment) but is inadequate for planning a surgical strategy.

Conventional proctography has been used since 1984 (10) and is still extensively used (11). It requires opacification of the rectum with a dense barium preparation, urinary bladder catheterization for contrast medium instillation, and small bowel opacification with an orally administered barium solution the night before the test. The advantages of proctography are its reduced cost, the wide availability of conventional radiography and fluoroscopy, and the physiologic assessment provided because the patient is in a sitting position. The disadvantages are the radiation dose and the fact that anatomic assessment of the pelvic floor is not provided.

Magnetic resonance (MR) imaging proctography was introduced in 1993 (12) and is increasingly used. High-resolution fast spin-echo (FSE) T2-weighted imaging provides optimal natural contrast of soft tissues; muscular and fascial injuries as well as pelvic organ abnormalities can be assessed. However, imaging of the rectum and optionally the vagina requires contrast agent administration. Dynamic MR proctography with interactive T2-weighted single-shot FSE (SSFSE) sequences of the pelvic floor at rest and during straining and defecation enables real-time evaluation of patterns of dysfunction (eg, descending perineum syndrome, rectal prolapse, an enterocele or cystocele, and genital prolapse). Examination is

Figure 1. Diagram shows the pathophysiology of pelvic floor failure.
usually performed in a closed-magnet MR imaging unit by using a supine patient position. The results of one study showed no major disadvantage of examinations performed in a closed-magnet MR imaging unit compared with those performed in an open-magnet MR imaging unit with the patient in a sitting position (13). However, because imaging performed during defecation is necessary for adequate evaluation of functional abnormalities (14), the nonphysiologic supine patient position is considered the main drawback of using a closed-magnet MR imaging unit.

**MR Imaging Examination**

Patients are thoroughly instructed about the MR imaging examination, reassured about privacy, and encouraged to comply with the procedure. Ultrasound gel (100 mL) warmed to body temperature is instilled in the rectum, and 5 mL may be introduced in the vagina. The patient is loosely wrapped in a waterproof incontinence pad and is instructed to strain and defecate when asked. A 4- to 8-element coil is used to acquire high-resolution axial FSE T2-weighted MR images (22-cm field of view [FOV], 3-mm thickness, 320 × 320 matrix, echo time [TE] = 120 msec, repetition time [TR] = 2360 msec, number of signals acquired [NSA] = 4) from the superior border of the pubic symphysis to the lower end of the anal canal. Axial, coronal, and sagittal SSFSE images (32-cm FOV, 6-mm thickness, 384 × 256 matrix, TR = 750 msec, TE = 120 msec, NSA = 1) are then obtained of the entire pelvis.

Functional imaging is performed by acquiring serial single-section midsagittal SSFSE MR images centered on the anorectum at rest, during straining, and during defecation. The use of SSFSE MR imaging sequences allows real-time imaging (15). Optionally, imaging is also performed during a squeezing maneuver to evaluate puborectalis muscle contraction. Usually one image is obtained every 2 seconds. Because performance of the defecation phase is crucial to obtain complete information, it is especially important to encourage the patient and interact with her continuously during the functional part of the study. Having the patient slightly bend her knees and abduct her hips may be helpful.

**Normal and Pathologic Anatomy at MR Imaging**

The pelvic floor has traditionally been described as consisting of three compartments that are referred to as the anterior, middle, and posterior compartments. This distinction artificially reflects the fact that different medical specialists (ie, urologists, gynecologists, and general surgeons) are involved in the treatment of each compartment. However, this approach ignores the complexity of pelvic floor failure, which is usually generalized; therefore, the three compartments should be assessed simultaneously. Optimally, patients should be evaluated by a team of specialists that includes a radiologist.

Because anatomic descriptions of the pelvic floor in the radiologic literature are at times confusing, we use descriptions of pelvic anatomy cited in the most recent authoritative anatomic literature (16–18). The pelvic floor is composed of three layers, listed from cranial to caudal: (a) the endopelvic fascia, which wraps and gives support to the pelvic organs but which is too thin to be recognized at MR imaging except for its condensations (the cardinal, uterosacral, and urethral ligaments), although its integrity can be inferred by the efficiency of the support it provides; (b) the levator ani, which is well recognized at MR imaging by its main components, the pubococcygeus muscles; and (c) the perineal membrane, which is recognized at imaging by its connective tissue condensation, the perineal body, which is the insertion site of the perineal muscles and external anal sphincter. The pelvic floor and levator hiatus have a warped structure (Fig 2), and volume-rendering techniques are being used as a research tool to improve understanding of their complex anatomy and function (19–22).

**Levator Ani.**—The main components of the levator ani are the iliococcygeus and pubococcygeus muscles (Figs 2, 3). A third muscle, the pubococcygeus, is located between the iliococcygeus and pubococcygeus muscles and is inconsistently distinguished at MR imaging.

The iliococcygeus muscle is a thin, sagittally oriented, fan-shaped structure that takes a curved shape with inferior concavity when observed in the coronal plane. It has lateral insertions to the pelvic sidewalls on the ATLA and medial visceral insertions on the pelvic organs (Fig 2). Posteriorly it forms a raphe that blends with the attachment of the external anal sphincter to form the anococcygeal ligament (17), a fascial condensation formerly known as the levator plate, that inserts on the coccyx (Figs 2, 3). The ATLA runs on the pelvic sidewall obliquely, cranial to caudal, from the inferior pubic symphysis to the ischial spine (Fig 2). It is roughly paralleled by...
Figure 2. Pelvic floor overview. (a) Coronal FSE T2-weighted MR image shows the endopelvic fascia (white outline) wrapping the uterus (U) and vagina (V), the curved iliococcygeus muscle (IIC; dotted line) inserting on the arcus tendineus musculi levatoris ani (ATLA), the puborectalis muscle (PR), and the external anal sphincter (EAS; dashed line). * = anal canal and internal anal sphincter, arrowheads = perineal membrane, R = rectum. (b) Wax anatomic model of the male pelvic floor seen from the left side shows the pelvic floor muscles. The iliococcygeus muscle (IIC; dashed line) inserts on the ATLA (球星) superolaterally, on the pelvic organs inferiorly, and on the anococcygeal ligament (AC) posteriorly. The puborectalis (PR) has been drawn (dotted line) encircling the anorectal junction and inserting on the pubic bone. The insertion of the endopelvic fascia (arcus tendineus fasciae pelvis [ATFP]) on the pelvic sidewalls has been drawn (solid double line) running obliquely from the inferior symphysis (S) upward to join the ATLA shortly before joint insertion on the ischial spine (IS). The ischiococcygeus muscle (IsC; not part of the pelvic floor) is shown running from the ischial spine to the coccyx (C) and anococcygeal ligament. O = obturator muscle, P = piriformis muscle, R = rectum, U = urethra. (Fig 2b courtesy of the C. Susini Collection, University of Cagliari, Cagliari, Italy.)

The ATFP, which is the insertion site of the endopelvic fascia on the pelvic sidewalls.

The puborectalis muscle is a U-shaped sling inserted on the inner pubis that can be recognized in the axial plane as it passes around the anorectal junction (Fig 4). It encloses the urethra, vagina, and anal canal in a space called the urogenital hiatus that normally is not larger than 4.5 cm on the transverse axis and not wider than 5.5 cm in the sagittal plane (Fig 4a). The puborectalis is responsible for the angle between the anal canal and rectum (Fig 3a) and is crucial for gross fecal continence.

The external anal sphincter is an independent muscle, although some of its fibers merge with the puborectalis muscle (16). It has a cylindrical portion, the deep external anal sphincter, which surrounds the internal sphincter (Fig 2a), and a subcutaneous portion, the superficial external anal sphincter, which has a winglike configuration in the sagittal plane (Fig 3a) and a “forked-tail” arrangement in the axial plane (Fig 4b). Its posterior fibers decussate to join the anococcygeal ligament (23,24).

Various patterns of levator ani lesions can be observed at MR imaging. The iliococcygeus muscle can be disrupted at its visceral insertion, an injury that permits the rectum to herniate into the ischiorectal fossa (Fig 5). Loss or reversion of its normal downward concavity at rest or during straining indicates abnormal function and is associated with pelvic organ prolapse and descending perineum syndrome. The puborectalis muscle can be damaged during vaginal delivery and can even be torn off from its pubic insertion (Fig 6) and may appear at imaging as thinned, bowed, or irregular as a consequence of atrophy due to pudendal neuropathy (25). Weakness of the levator ani may cause sagging of the anococcygeal ligament. This sagging opens the urogenital hiatus and constitutes a predisposition to pelvic organ prolapse (26). The external sphincter can also be damaged during vaginal delivery and may appear at imaging as disrupted or thinned (Fig 7).

Endopelvic Fascia and Vaginal Support.—The endopelvic fascia is a network of connective tissue lying deep to the peritoneum that supports the pelvic organs. The endopelvic fascia wraps the bladder, vagina, and uterus and suspends them through the parametria and paracolpium to the ATFP (Fig 2), which is inserted on the
Figure 4. Urogenital hiatus. (a) Axial FSE T2-weighted MR image shows the puborectalis sling (dotted line) as it inserts on the pubic bones and encloses the vagina (V), urethra (U), and anorectum (R) in the urogenital hiatus. Note the typical \( H \) shape of the vagina at the middle third. (b) At a lower level, the internal anal sphincter (IAS) and external anal sphincter (EAS) with its characteristic posterior “forked-tail” are seen. The vagina (V) at this level has increased anterior concavity and supports the urethra (U) like a hammock.

Figure 3. Pelvic floor muscles. (a) Sagittal FSE T2-weighted MR image shows the bladder (B), uterus (U), and rectum (R). The puborectalis muscle (PR; dashed-dotted lines), which encircles the urogenital hiatus, is responsible for the angle between the rectum and the anal canal (angled solid line). The superficial external anal sphincter (EAS; dashed line) is indicated. The perineal body (*) is seen as a rounded hypointense structure interposed between the lower vagina and external anal sphincter. The anococcygeal ligament (AC; dotted line) is also indicated. The urethra (arrows) is entirely retropubic. \( S = \) symphysis. (b) Axial FSE T2-weighted MR image shows the ischial spine (IS), which is the insertion site of the ischiococcygeus muscle (IsC) and ATLA. The visceral insertion of the iliococcygeus muscle (IIC) is seen, as are the obturator muscle (O) and anococcygeal ligament (AC). B = bladder, C = coccyx, R = rectum, V = vagina.

pubic bone and pelvic sidewalls. Posterior support comes from the uterosacral ligaments, which are paired fascial condensations inserted on the sacrum (Fig 8).

Centrally, the endopelvic fascia forms the pubovesical ligaments (16,17) between the pubis, bladder, and anterior vaginal wall, whereas posteriorly it forms the rectovaginal fascia between the posterior vaginal wall and rectum (Fig 8). Three levels of fascial support have been described: level I, the upper third of the vagina and cervix; level II, the middle third of the vagina; and level III, the lower third of the vagina (Fig 8) (27).

The level I endopelvic fascia suspends the vaginal apex to the uterosacral ligaments and ischiococcygeal ligaments (Fig 8). In the axial plane, the upper
Figure 5. Iliococcygeus muscle lesions. (a) Axial FSE T2-weighted MR image in a 71-year-old woman with severe pelvic organ prolapse shows disruption of the iliococcygeus at its visceral insertion (arrows), a condition that allows the rectum (R) to protrude into the right ischiorectal fossa. (b) Coronal FSE T2-weighted MR image in the same patient shows thinning of the iliococcygeus on the right side (arrowheads). Compensatory thickening is noted on the opposite side (arrow), and inferior concavity is reduced.

Figure 6. Puborectalis muscle lesions. Gel was instilled in the rectum (R) of both patients. (a) Axial T2-weighted MR image of the lower vagina (V) in a 57-year-old woman (para 4) with genital prolapse shows complete detachment of the puborectalis on the right side (arrow). The left side is normal (arrowhead). (b) Axial FSE T2-weighted MR image in a 56-year-old woman who presented with a descending perineum shows bowing of the puborectalis on both sides (arrows).

The vagina may appear flat or curved, depending on the amount of rectal content (Fig 9). Vaginal delivery, chronic straining, and altered collagen metabolism are the main causes of fascial tearing. The posterior attachment of the ATFP to the ischial spine has been found to be avulsed in 96% of parous women (28). When the upper vagina is detached from the uterosacral ligaments (as may occur after hysterectomy), a level I fascial lesion exists, and bilateral sagging of the vagina (the “chevron” sign) is seen at MR imaging (Fig 9).

The level II endopelvic fascia supports the vagina at its middle third (Fig 8), with direct insertion to the ATFP; it is responsible for the typical H shape observed on axial images (Fig 4a). The level II endopelvic fascia also supports the posterior wall of the urinary bladder and is responsible for its flat appearance on axial images (Fig 8).

The level II fascia may be torn laterally (a paravaginal tear) or centrally in the pubocervical or rectovaginal portion. Lateral disruption of the level II endopelvic fascia causes the posterior bladder wall to fall posteriorly on one or both sides, an imaging finding described as the “saddlebag” sign (Fig 10a). When a lateral level II endopelvic fascial lesion is combined with a lesion
of the puborectalis muscle, it causes architectural distortion of the lateral vaginal wall (22) that appears at imaging as a posterior or lateral spilling on one or both sides (Fig 10b). This combined lesion has been shown to increase the risk of vaginal prolapse (29).

A posterior central (rectovesical fascia) defect may produce a gap between the rectum and vagina where peritoneal bowel or fat can herniate, causing a peritoneocele or an enterocele (Fig 11). During straining, a rectovaginal fascial tear may also produce a bulging of the anterior rectal wall on the posterior vagina (a rectocele), while a pubocervical fascial tear may cause the bladder wall to bulge on the anterior vagina (a cystocele).

The level III fascia merges with the lower third of the vaginal wall and the perineal membrane. The urethral suspension ligaments are also part of the level III endopelvic fascia (Fig 12a). Level III lesions are seen at imaging as a disruption or complete absence of the urethral suspension ligaments and enlargement of the retropubic (Retzius) space between the pubic bone and urethra, a finding called the “drooping mustache” sign (Fig 12b) (30).

**Urethra and Urinary Continence.**—The urethra has a targetlike appearance on axial images (Fig 12a), with a hypointense, striated-muscle, outer sphincter surrounding the smooth-muscle inner sphincter and innermost mucosa-submucosa. The average thickness of the urethral sphincters is 4 mm. On midsagittal T2-weighted images, the urethra appears as a hypointense, retropubic, tubular structure (Fig 3a). The urethra is normally located entirely behind the pubic symphysis at rest and during straining. The anatomic structures that guarantee urinary continence are divided into the sphincteric and supportive systems. The urethral mucosa, erectile tissue of corpora spongiosa clitoridis, and connective tissues in the urethral wall also aid in coaptation.

The striated urethral sphincter, or urogenital sphincter, is divided into three components (cranial to caudal): (a) the sphincter surrounding the proximal and mid urethra (Fig 12a), (b) the urethrovaginal sphincter with fibers surrounding the urethra and vagina (Fig 13), and (c) the compressor urethrae muscle (Fig 14). Functionally, the compressor urethrae muscle can both compress and elongate the urethra, thus improving continence (31).
Figure 9. Normal fascial support and level I fascial lesion. (a) Axial FSE T2-weighted MR image obtained with rectal gel shows normal level I fascial support to the upper vagina (V), which is gently curved around the rectum (R), and normal level II fascial support to the posterior bladder (B) wall (curved dotted line). The relationship of level I to level II endopelvic fasciae changes with urinary bladder distention and uterine involution. (b) Axial FSE T2-weighted MR image in a 59-year-old woman with descending perineum syndrome after a hysterectomy shows the typical distortion of an unsuspended upper vagina, known as the “chevron” sign (angled dotted line). B = bladder, R = rectum.

Figure 10. Level II paravaginal defect. Ultrasound gel was instilled in the rectum (R) and vagina (V in a; arrowhead in b) of both patients. (a) Axial FSE T2-weighted MR image in a 49-year-old multiparous woman referred for urinary incontinence shows bilateral loss of support of the posterior bladder wall (dotted line), a finding known as the “saddlebag” sign. B = bladder. (b) Axial FSE T2-weighted MR image in a 33-year-old woman (para 2) with mixed urinary incontinence and genital prolapse shows distortion of the vagina (arrowhead) (the H shape is lost on the right side) due to fascial disruption associated with a muscular lesion. The puborectalis muscle is detached from the pubic bone on the right side (black arrow). The white arrow indicates normal pubic insertion on the left side. U = urethra.

This supportive system has been likened to a hammock; in fact, the urethra lies on a supportive layer composed of the endopelvic fascia and anterior vaginal wall (Figs 4, 12). This layer gains structural stability through its anterior insertion on the pubic bone and lateral attachment to the ATFP and levator ani muscle (32). Abdominal pressure pushes the urethra against this hammock with a mechanism similar to that of a foot compressing a garden hose against the ground (33). Laxity of the urethral support ligaments causes dissipation of the compression strength on the urethra and may lead to incontinence.

With postmenopausal atrophy, the volume of striated muscle and vascular tissue decreases and the relative volume of connective tissue increases. Flattening of the posterior urethral wall (Ω-shaped urethra) (Fig 15a) or increased signal intensity of the thin external urethral sphincter
Figure 11. Level II central defect. Axial T2-weighted MR image in a 71-year-old woman with obstructed defecation syndrome shows disruption of the rectovaginal fascia with resultant peritoneal fat herniation (※) below the upper third of the vagina (V). Ultrasound gel is seen in the rectum (R). P = peritoneocele, U = urethra.

Figure 12. Normal and disrupted level III fascia. R = rectum. (a) Axial FSE T2-weighted MR image shows (from anterior to posterior) the pubourethral, periurethral, and paraurethral supporting ligaments (arrowheads). The ATFP anchors the lower vagina to the pubic bone, thus supporting the urethra (U). The urethra displays a typical targetlike appearance, with a thin hypointense external sphincter, thick internal sphincter, and hyperintense innermost mucosa. V = vagina. (b) Axial FSE T2-weighted MR image in a 50-year-old woman with stress urinary incontinence shows absence of the urethral supporting ligaments (due to complete disruption) and enlargement of the retropubic space (Rp; ※), findings known as the “drooping mustache” sign. Note that no fascial defect can occur between the urethra (U) and distal vagina (V) because at this level the two muscular layers are fused.

Figure 13. Urethrovaginal sphincter. Axial FSE T2-weighted MR image shows the distal vagina (V) and distal urethra (U) flattened anteroposteriorly and enclosed by the urethrovaginal sphincter (dashed contour line). IAS = internal anal sphincter.

Muscle may be seen on MR images. Repeated episodes of traction, prior surgery, trauma, and gradual loss of pudendal nerve function may also cause sphincteric damage. In severe cases, the urethral lumen becomes patent during straining or even at rest, a condition known as funneling (34) (Fig 15b). Although funneling and urethral hypermobility are easily recognized at MR imaging, evaluation of the urethral sphincter may be challenging when only an external coil is used.

Perineal Membrane.—The perineal membrane is the most caudal layer of the pelvic floor, bordering
Figure 14. Perineal membrane. Axial FSE T2-weighted MR image (a) and wax anatomic model (b) show the urethra (1) and vaginal vestibule (V) enclosed between the paired corpora spongiosa clitoridis (3). The perineal membrane (7) spans the anterior triangle between the pubis and ischial bones. The perineal body (5) is interposed between the vaginal vestibule and anal canal; the external anal sphincter (6) is seen in the midline. The compressor urethrae (2; arrows in a) runs from the perineal body to the ischial bone; it has been cut away in the wax model in b. The corpora cavernosa clitoridis and the ischiocavernous muscle (4) are seen at the same level. (Fig 14b courtesy of the C. Susini Collection, University of Cagliari, Cagliari, Italy.)

Figure 15. Torn urethral sphincter. (a) Axial FSE T2-weighted MR image in a 64-year-old woman with mixed urinary incontinence (urgency and stress incontinence) shows distortion of the normal appearance of the urethra (U), with flattening of the posterior wall. The arrowheads indicate an Ω-shaped dehiscence. The vagina (V) is unsupported because of a fascial lesion and appears flattened anteroposteriorly. The puborectalis muscle is detached from the pubic bone on the left side (arrow). The retropubic space (5) is enlarged. Ultrasound gel is seen in the rectum (R). (b) Sagittal T2-weighted MR image in a 47-year-old patient with obstructed defecation syndrome and stress urinary incontinence. During straining, the bladder neck and proximal urethra are pushed low and appear widely patent (arrows), a condition referred to as funnelling, because of sphincteric disruption. Ultrasound gel is seen in the vagina (V) and rectum (R). B = bladder.
Vaginal delivery can stretch and cause extensive damage to perineal tissues; this damage is recognized at MR imaging as scarring of the perineal body (Fig 16) and thinning or distortion of the compressor urethrae muscle. The perineal tissues are small and easily overlooked; however, when damaged, their role in maintaining normal motion is impaired. Contraction of the levator ani muscle should pull the pelvic floor superior and anterior, thereby narrowing the urogenital hiatus, but in the absence of the perineal body’s anchoring position, the muscle force may produce asymmetric or dysfunctional motion (36).

**Figure 16.** Scarring of the perineal body. Axial FSE T2-weighted MR image shows scarring of the perineal body (arrow) in a 68-year-old woman (para 5) who presented with fecal incontinence and a history of severe dystocia. Ultrasound gel is seen between the labia majora.

**Figure 17.** Drawings of a ship illustrate the interactions of the levator ani and endopelvic fascia. Support to the uterus and vagina (the ship) is provided mainly by the pelvic floor muscles (the sea), while the fascia (the ropes) holds the organs in place. On the left, the ship is supported by the sea and stabilized by ropes. The middle image shows low tide (the pelvic floor muscles are disrupted or atrophic). The ropes may still to some extent hold the boat, but eventually (right) the ropes may tear (increasing the risk of pelvic organ prolapse).

**Levator Ani and Pelvic Fascia Interactions.**—The levator ani muscle keeps the pelvic floor closed, and the fascia stabilizes the pelvic organs above it. The uterus has been compared to a ship floating on the water, attached by ropes on either side to a dock (Fig 17) (37). The ropes (fascia) hold the ship (uterus) as it floats on the water (muscles). If the muscles disrupt or atrophy, the fascia may be able to hold the uterus and vagina in the correct position (as at low tide) (Fig 6a). However, eventually the ropes will tear, increasing the risk for separation of the ship from the dock (Fig 10b). Accordingly, pelvic organ prolapse is primarily related to levator ani defects; associated fascial defects further increase the risk of pelvic floor dysfunction (22,29).

**Functional MR Imaging**

Analysis of pelvic floor functional abnormalities requires evaluation with dynamic MR imaging sequences. Several lines can be drawn on sagittal MR images to assess and measure the extent of pelvic floor dysfunction at rest and during straining. The pubococcygeal line (PCL) joins the inferior border of the symphysis pubis to the last coccygeal joint in the midsagittal plane (Fig 18) (38) and is the reference line for most measurements. A second line, drawn from the inferior border of the symphysis pubis to the posterior border of the anorectal junction, is called the H line. It is used to measure the anteroposterior width of the pelvic hiatus, which normally should not be more than 5 cm in its sagittal direction at rest or during defecation. Line M is traced between the anorectal junction and the PCL and should not exceed 2 cm (39).

The positions of the bladder, vaginal vault, and anorectal junction (the junction of the rectum and anal canal) are measured at a 90° angle to the PCL (Fig 18). The PCL is used as the reference line to determine the extent of a cystocele or enterocele, rectal descent, and vaginal vault descent. Prolapse is graded as mild (<3 cm below the PCL), moderate (3–6 cm), or severe (>6 cm) (40).
Figure 19. Abnormal pelvic floor measurements at MR imaging. Sagittal T2-weighted MR images were obtained at rest and during straining in a 62-year-old patient with positional voiding, pelvic organ prolapse, and occasional fecal incontinence. \( B = \text{bladder}, R = \text{rectum}. \) (a) At rest, a wide anorectal angle (ARA; curved black line) is seen between the central axis of the anal canal and the tangent to the posterior rectal wall. The anococcygeal ligament (AC; dashed white line) has an increased caudal angulation and points downward and away from the PCL (solid white line). Compare this with Figure 3a, where the AC ligament points anteriorly. \( U = \text{uterus}. \) (b) During straining, abnormal distances (dashed white lines) of the bladder base (B1), uterus (U), and anorectal junction (white arrow) from the PCL (solid white line) are seen. A cystocele, genital prolapse, descending perineum, and bowing of the anterior abdominal wall during straining (black arrow) are also seen.

The anorectal angle is drawn between the longitudinal axis of the anal canal and the posterior rectal wall (Fig 3a). It is normally 90°–110° and opens during defecation, but paradoxical reduction may be seen with normal anatomy. Correct performance of straining maneuvers is seen at imaging as a bowing of the abdominal wall and a whirl of flow in the urinary bladder.

The angle of the anococcygeal ligament with the PCL is also observed. In healthy women, the anococcygeal ligament roughly parallels the PCL at rest and during pelvic straining; an increased caudal inclination indicates loss of posterior muscular support (Fig 19) (41,42).

Pathophysiology and Functional Disorders
Pelvic floor functional disorders include pelvic organ prolapse, which may involve the urethra, urinary bladder, vaginal vault, uterus, and rectum, and pelvic floor relaxation. Pelvic floor relaxation, or descending perineum syndrome (Fig 20), is an excessive caudal movement of the pelvic floor during evacuation that may result from obstetric trauma, chronic straining, or pudendal neuropathy. It has two components: hiatal enlargement (the \( H \) line is elongated) and pelvic floor descent (the \( M \) line is increased). It can result in urinary stress incontinence, genital prolapse, and impaired
defecation. Different combinations of pelvic floor relaxation and pelvic organ prolapse may occur in a single patient, and similar symptoms can derive from different defects or dysfunctions.

**Urinary Incontinence**

Lower urinary tract symptoms associated with pelvic floor dysfunction include stress urinary incontinence, overactive bladder, and bladder outlet obstruction. Stress urinary incontinence is urethral incompetence that causes involuntary loss of urine during physical activity such as coughing, sneezing, laughing, or exercise. Overactive bladder (also called urge incontinence) is due to sudden contraction of the detrusor muscle and often is related to inflammation, infection, and nervous system diseases. It also is frequently reported by patients with pelvic organ prolapse and is likely induced by bladder outlet obstruction, as it may also occur in men with benign prostatic hyperplasia. In women with pelvic floor dysfunction, overactive bladder often is relieved by surgical treatment of pelvic organ prolapse (43).

**Stress urinary incontinence is caused by urethral hypermobility (80%–90% of patients)** (44) or intrinsic sphincter dysfunction (10%–20% of patients) with or without funneling (Fig 15). Urethral hypermobility results from laxity of the
urethral supporting structures (due to vaginal delivery, hysterectomy, or menopause) that leads to rotation of the urethral axis from vertical to horizontal, to a position more than 30° from its resting axis, during straining (termed rotational descent) (Fig 20). Hypermobility and sphincter damage may also be associated.

**Cystocele**
A cystocele is due to stretching or tearing of the endopelvic fascia that causes herniation of the bladder on the anterior vaginal wall. It is easily recognized at MR imaging as a descent of the bladder base below the PCL (Figs 19–24). It may be associated with damage to the urethral suspension ligaments and urinary incontinence. Cystocele-induced vaginal bulging may cause difficult intercourse or dyspareunia.

**Bladder Outlet Obstruction**
Bladder outlet obstruction may manifest as voiding hesitancy, required positional voiding, required manual reduction of a prolapse for voiding, and frank urinary retention that occasionally requires catheterization. The most commonly reported cause of bladder outlet obstruction is surgical repair for stress urinary incontinence (45).

Other causes of bladder outlet obstruction include urethral hypermobility, bladder outlet compression by a prolapsing uterus or rectocele (46–48), and kinking of the urethra or bladder outlet in patients with a cystocele as the bladder base shifts posteriorly and below the bladder outlet (Fig 21). Voiding dysfunction can be asymptomatic in cases of complex prolapse because kinking of the bladder neck may conceal incontinence before eliciting symptoms of bladder outlet obstruction. If this condition is not recognized, repair of a cystocele or genital prolapse will engender the postoperative onset of urinary incontinence and will require repeat surgery.

In some patients, bladder outlet obstruction and urge incontinence may coexist (Fig 22). Particularly in older women with prolonged bladder outlet obstruction, bladder trabeculation can be observed, a finding that also is commonly seen in male patients with benign prostatic hyperplasia (49).

**Bowel Symptoms**
Bowel symptoms caused by pelvic organ prolapse include difficult defecation, fecal incontinence, required digital manipulation to complete defecation, a feeling of incomplete evacuation, and rectal
Figure 24. Rectal prolapse. \( R = \text{rectum}, \) solid line = PCL. (a) Sagittal SSFSE T2-weighted MR image obtained during straining in an obese 20-year-old woman (para 1) with obstructed defecation syndrome shows a full-thickness rectoanal prolapse (intussusception). The rectal wall is circumferentially infolded (arrow). Note the cystocele \( (B) \), urethral hypermobility (dashed line), and uterine prolapse \( (C) \). (b) Sagittal SSFSE T2-weighted MR image obtained during straining in a 50-year-old woman with severely obstructed defecation, rectal bleeding that required a blood transfusion, and positional voiding shows external rectal prolapse (arrow). Urethral hypermobility (dashed line), a cystocele \( (B) \), and genital prolapse \( (C) \) are also seen.

protrusion during or after defecation. Defecation disturbances may be due to impairment of the puborectalis or anal sphincter, because voluntary defecation requires relaxation of the anal sphincters and puborectalis muscle, which straightens the rectoanl angle, and simultaneous contraction of the rectal smooth muscle.

Obstructed Defecation Syndrome
Obstructed defecation syndrome is a sensation of incomplete defecation or anorectal obstruction for 25% or more of defecations. Its prevalence is about 7% in the United States and Europe, it is more common in women, and it usually starts in the 4th or 5th decade of life (50). The pathophysiology of obstructed defecation syndrome is usually multifactorial and may include reduced rectal sensation, a nonrelaxing pelvic floor or paradoxical contraction of the puborectalis muscle, pelvic floor laxity, rectal prolapse, and rectocele or enterocoe formation (51). Slow colonic transit must be ruled out before a diagnosis of obstructive defecation syndrome is made. Pelvic floor descent is the most common finding in patients with obstructed defecation syndrome and is associated with a feeling of incomplete evacuation followed by more straining, which leads to increasing pudendal nerve impairment and, over time, to fecal incontinence (7).

Fecal Incontinence
Fecal incontinence is the continuous or recurrent passage of fecal material (>10 mL) for at least 1 month in a person older than 3–4 years (52). Its prevalence is unknown, but fecal incontinence is known to increase with age, with a prevalence of up to 11% in elderly women (25). Most cases of fecal incontinence are acquired. Vaginal delivery may cause sphincter disruption, which is especially difficult to detect at MR imaging with external coils because disruption typically is located in the anterior part of the sphincter, which is normally folded.

Pudendal neuropathy (denervation) induced by chronic straining, advanced age, or heavy smoking (53) may cause atrophy of the puborectalis muscle and sphincter. Symptomatic fecal incontinence occurs in 21% of women who present with urinary incontinence, pelvic organ prolapse, or both (25). Although the role of dynamic MR proctography in the evaluation of fecal incontinence is somewhat limited (unless an endorectal coil is used to better depict the internal sphincter), MR proctography has been shown to be useful in patients who are candidates for surgical treatment of incontinence (54) because it may demonstrate associated pelvic floor dysfunction. In one series, 94% of patients with fecal incontinence who were evaluated with MR defecography had a rectal descent of more than 6 cm (54). An imaging finding of a wide anorectal angle at rest is also an important factor in evaluation (Fig 19).

Rectocele
A rectocele is an outpouching of the rectal wall that protrudes onto the posterior aspect of the vagina and, similar to a cystocele, can bulge below the introitus (Figs 22, 23). A rectocele may
cause obstructed defecation because it causes dissipation of the vector force during straining. Rectal content may become trapped in rectoceles, which may impair emptying. A need for digital assistance for defecation is often reported. An anterior rectocele is due to a defect in the rectovaginal fascia, while a less-common posterior rectocele is due to a defect in the anococcygeal ligament (Fig 23). On MR images, a rectocele is measured as the distance of the anterior or posterior rectal wall from the anal canal axis. A rectal bulge of less than 2 cm is within normal limits (55); a bulge of more than 3.5 cm is considered large (56).

Rectal Prolapse
Rectal prolapse is an infolding of the rectal wall that is induced by chronic straining and fascial disruption (Fig 24). It can involve only the mucosa or the full wall thickness (a true intussusception) and may be internal (intrarectal or intranal) or external. It is usually circumferential, but mucosal prolapse limited to the anterior rectal wall may be observed (Fig 25). Rectal prolapse causes obstructed defecation that may subsequently progress to pudendal neuropathy and fecal incontinence. External anal sphincter atrophy is an associated finding.

Rectal prolapse can be difficult to detect at MR imaging, and fluoroscopy has been shown to be a superior imaging modality for detection. The sensitivity of MR imaging for rectal intussusception has been reported to be 70% relative to evacuation proctography (57). However, the soft-tissue resolution of MR imaging may permit better differentiation of mucosa-only intussusception from full-wall-thickness intussusception (57) if the evacuation phase is adequate (14).

Enterocoele
An enterocoele, or cul-de-sac hernia, is a herniation of peritoneal membrane that protrudes between the uterosacral ligaments at the apex of the vagina and extends distally into the rectovaginal septum, separating the rectum from the vagina (Fig 25). It may contain fat (a peritoneocele), small bowel, or sigmoid colon (a sigmoidocele). Patients who have undergone hysterectomy are primarily at risk for developing an enterocoele because of interruption to the continuity of the pubocervical and rectovaginal parts of the endopelvic fascia. An enterocoele often manifests only at the end of the evacuation phase after rectal and bladder emptying (Fig 25b). It may cause symptoms of bowel obstruction, vaginal pressure, dyspareunia, and low back pain. An enterocoele is an important MR imaging finding because it will require a peritoneal approach to surgical repair.

Genital Prolapse
Uterine or vaginal vault prolapse is due to muscle damage and stretching or tearing of the uterosacral ligaments that allows descent of the vaginal fornix and uterus below the PCL (Fig 26). This mechanism has been described as similar to the eversion of an invaginated finger in a glove. Uterine or
vaginal vault prolapse may manifest with a vaginal mass, dyspareunia, urinary retention, or back pain. Severe genital prolapse may be associated with ureteral obstruction (Fig 27). The pelvic organs (uterus, rectum, and bladder) may compete for room in the pelvic outlet (Fig 21a) so that a prolapsing organ may preclude the dislocation of other organs and conceal symptoms that may become overt after surgical repair of a single defect or after the emptying of a rectocele or cystocele.

**Spastic Pelvic Floor Syndrome**
Also known as anismus or solitary rectal ulcer syndrome, spastic pelvic floor syndrome is a paradoxical contraction of the puborectalis muscle during straining (Fig 28). Prolonged and incomplete evacuation is the main sign of spastic pelvic floor syndrome, because some degree of puborectalis contraction during defecation may be seen in healthy subjects.
Figure 28. Spastic pelvic floor syndrome. (a) Sagittal FSE T2-weighted MR image obtained at rest in a 61-year-old woman with urinary incontinence and occasional difficult defecation after hysterectomy shows the anorectal angle ($ARA$) (angled line) in the normal range ($90^\circ -110^\circ$). Dotted line = $H$ line. (b) Sagittal FSE T2-weighted MR image obtained during straining in the same patient shows that the anterior abdominal wall is bowed and the bladder neck is pushed down ($*$). The puborectalis muscle is paradoxically contracted, causing shortening of the $H$ line (dotted line) and narrowing of the anorectal angle ($ARA$) (angled line).

Conclusion
Multiple anatomic and functional lesions usually coexist in a patient with pelvic floor failure. Even in patients who present with symptoms in a single compartment, the pelvic floor as a whole is usually damaged, and relapses may occur if only the symptomatic compartment is surgically repaired. Radiologists can use MR imaging to evaluate pelvic floor functional abnormalities (eg, descending pelvic floor syndrome and pelvic organ prolapse) and accurately assess associated muscular and fascial defects, thus providing the surgeon with a road map for tailored treatment.

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References


Pelvic Floor Failure: MR Imaging Evaluation of Anatomic and Functional Abnormalities

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Page 432
The puborectalis muscle can be damaged during vaginal delivery and can even be torn off from its pubic insertion and may appear at imaging as thinned, bowed, or irregular as a consequence of atrophy due to pudendal neuropathy.

Page 434
The level II fascia may be torn laterally (a paravaginal tear) or centrally in the pubocervical or rectovaginal portion.

Page 439
The pubococcygeal line (PCL) joins the inferior border of the symphysis pubis to the last coccygeal joint in the midsagittal plane and is the reference line for most measurements.

Page 441
Stress urinary incontinence is caused by urethral hypermobility (80%–90% of patients) or intrinsic sphincter dysfunction (10%–20% of patients) with or without funneling.

Page 444
On MR images, a rectocele is measured as the distance of the anterior or posterior rectal wall from the anal canal axis. A rectal bulge of less than 2 cm is within normal limits; a bulge of more than 3.5 cm is considered large.