Structural anatomy of the posterior pelvic compartment as it relates to rectocele

John O.L. DeLancey, MD
Ann Arbor, Michigan

OBJECTIVE: This study was undertaken to define posterior compartment structural anatomy relevant to rectocele.

STUDY DESIGN: Dissection of 42 fresh and 22 fixed cadavers was supplemented by examination of histologic (n = 3) and macroscopic (n = 5) serial sections.

RESULTS: Distal posterior compartment support involves connection of the halves of the perineal membrane (urogenital diaphragm) through the perineal body, preventing downward protrusion of the lower rectum. Above this level the posterior vaginal wall is held in place by sheets of bilateral endopelvic fascia that attach each side of the posterior vaginal wall to the pelvic diaphragm. Most of these fascial fibers attach to the vaginal wall and a few fibers unite in the midline. Pelvic floor closure by the levator ani muscles relieves pressure-induced stress on the midvaginal fascial supports.

CONCLUSIONS: Midline perineal membrane union supports the distal posterior compartment and a fascial connection between the pelvic diaphragm and vagina supports the mid vagina. Muscular pelvic floor closure helps to relieve fascial stress. (Am J Obstet Gynecol 1999;180:815-23.)

Key words: Endopelvic fascia, levator ani muscles, pelvic organ prolapse, rectocele

One in every 9 American women requires surgery for problems related to defective pelvic organ support, and among these women 1 in every 4 needs a second operation. Among women with documented prolapse, 76% had defects found in support of the posterior compartment. Despite the common occurrence of rectocele, the structural defects responsible for its formation remain poorly understood.

The pelvic organ support system has an anterior compartment containing the urethra and bladder and a posterior compartment containing the anus and rectum. The vagina, the uterus, and the endopelvic fascia that attaches them to the pelvic walls separate these 2 compartments and prevent their contents from protruding downward through the urogenital hiatus in the levator ani muscles.

The study described here was carried out to define the detailed anatomy of the normal structural supports of the posterior compartment that prevent anterior protrusion of the rectal wall. This research was necessary so that the specific sites of anatomic defects could be identified to guide more precise scientific and clinical research.

Material and methods

The anatomic materials used in this study are summarized in Table I. Among the specimens 14 cadavers were nulliparous, 40 were parous, and parity could not reliably be established for 18, either from a review of hospital records when these were available or by physical examination. Six cadavers were African American and the remaining 66 were white. Twelve had undergone hysterectomy, 1 had undergone a supracervical hysterectomy, and 47 had intact uteri. Early in the study, during the first 12 dissections, hysterectomy status was not noted.

Anatomic findings reported are confined to those structures and relationships that could be documented on dissection of both fresh and embalmed cadavers and verified.
with macroscopic and histologic serial cross sections. The term vaginal wall is used to include the vaginal mucosa, submucosa, and muscularis. The term endopelvic fascia is used to denote those tissues between the vaginal muscularis and adjacent organs or the pelvic walls. Description of different levels of support corresponds to those in an earlier publication. Anatomic terms used conform to the Nomina Anatomica.

The overall arrangement of pelvic floor structures was studied in specimens fixed by injection embalming. Because these specimens are known to exhibit distorted spatial relationships, additional specimens were specially fixed by an immersion technique that produces specimens with topographic relationships that correspond to data available from living women. This process involves floating the specimen in formalin to avoid gravity-induced sagging caused by loss of muscle tone.

Resistance of posterior vaginal wall support was studied in fresh cadavers in a state not altered by fixation.
Results

The distal rectum abuts against the dense connective tissue of the perineal body (level III). The perineal body represents the central connection between the halves of the perineal membrane (urogenital diaphragm). When the distal rectum is subjected to increased force directed caudally, the fibers of the perineal membrane become tight and resist further displacement. These fibers derive their lateral support from their attachment to the pelvic bones at the ischiopubic rami (Fig 1, A, and Fig 2). This layer’s ability to resist downward displacement depends on the structural continuity between the right and left sides of the perineal membrane. Transection of these fibers in the cadaver leaves the rectum exposed and allows the distal rectum (Fig 1, B) to prolapse downward.

The connection between the halves of the perineal membrane extends cranially for a distance of approximately 2 to 3 cm above the hymenal ring (Fig 3). It is thickest and densest in the distal perineal body, becoming progressively thinner toward its cranial margin. This region (level III) of the posterior vagina is characterized by a U-shaped contour, in contrast to the W-shaped contour seen in level II (Fig 1, A). The upper extent of the perineal body becomes confluent with the supportive structures of level II, and these 2 levels are structurally interdependent.

During selected dissections (n = 6) in which rigor mortis (as assessed by resistance to forearm extension) maintained levator ani muscle tone, downward force was applied to the pelvic floor. This was done first with a finger in the rectum to localize applied force in a specific direction and then with an air-filled transparent bag to apply pressure to the pelvis as a whole. In addition, in 3 of the specimens fixed by immersion a string was threaded from the origin of the puborectalis muscle on the inner surface of the pubic bones, parallel to the muscle fibers passing dorsal to the anorectal junction and returning to the pubic bone on the opposite side. Tension on the string was adjusted to achieve normal urogenital hiatus size and perineal body position. This technique served as a surrogate for puborectalis muscle tone and allowed interactions of connective tissue and muscle to be simulated and observed.

The in situ relationships of the organs and their supportive structures were studied in serial cross sections to supplement the observations mentioned previously because dissection by its very nature distorts the specimen. Macroscopic whole-pelvis cross sections were cut from frozen immersion-fixed specimens at intervals ranging from 5 to 20 mm.

Serial histologic sections were made available to me by Dr Thomas Oelrich; their preparation has previously been described elsewhere. These were used to confirm the histologic nature of the tissues present, to determine the specific direction of various fibers within the pelvis, and to elucidate the nature of attachments between various structures.

**Fig 4.** A, Macroscopic section of 14-year-old nulliparous cadaver. B, Histologic slide of Mallory trichrome–stained section of left half of 1-year-old infant. Note that most fibers of endopelvic fascia (outlined by dots) attach to lateral sulcus of posterior vaginal wall (VAG WALL) with only a small proportion of fibers connecting with fibers of contralateral side (asterisk). In B note origin of endopelvic fascia from superior fascia of levator ani muscle (LAM). OI, Obturator internus muscle; URETH, urethra.
The lateral margin of the perineal body contains the termination of the bulbocavernous muscle. Caudally directed force does not put this muscle under tension, however, because of its sole anterior insertion onto the clitoris. A few wispy striated muscle fibers, named the superficial transverse muscles of perineum, lie along the posterior margin of the perineal membrane.

The middle portion of the posterior vaginal wall (level II) is attached on either side of the rectum to the inner surface of the pelvic diaphragm by a sheet of endopelvic fascia. These fascial sheets attach to the posterior lateral vaginal wall, where the dorsally directed tension results in a posterior vaginal sulcus on each side of the rectum (Fig 4). These endopelvic fascial sheets prevent the ventral movement of the posterior vaginal wall (Fig 5) and create the W shape of the posterior vaginal wall characteristic of level II.

Most of the endopelvic fascia fibers attach to the vaginal wall, with only a few fibers passing from one side to the other (Fig 4, B). Cutting the fibers of the endopelvic fascia between the vaginal sulcus and pelvic diaphragm results in significant destabilization of the posterior vagina in level II, whereas transection of midline fascial fibers results in a much less dramatic change in support.

The level II and level III supports are continuous with one another. Force applied to the anterior rectal wall in level II is resisted by the posterior vaginal wall and its attachments to the inner surface of the pelvic diaphragm. Pressure applied to the perineal body in a caudal direction in level III is resisted not only by the perineal membrane but also by the connection of the upper vaginal wall to the level II attachments that help hold the top of the perineal body (level III) in place.

The directions of the connective tissue fibers that provide support in level III and level II are different. The predominant direction of fiber flow in level III is from side to side, spanning the gap between the ischiopubic rami. In level II the fibers change to a dorsal and cranial direction, where they pull upward in a parasagittal plane.

The upper portion of the posterior vaginal wall in level I is attached to the pelvic wall by the sheetlike mesentery of the paracolpium (Fig 6). This anatomy has been described elsewhere and will not be recounted here.

When the dynamics of posterior compartment support are examined with the puborectalis portion of the levator ani muscle in a simulated state of contraction, the posterior vaginal wall was in contact with the anterior wall (Fig 7, A). In this situation, the pressures in the anterior and posterior compartments are balanced and there is little or no stress on Level II fascial supports. The force is carried by the levator ani muscles and perineal body. When the muscle is relaxed or damaged, the vaginal canal opens (Fig 7, B) and support is required from the connective tissues in level II (Fig 7, C), indicating an interaction between the muscular and fascial supports.

It is not possible to completely separate the effects of muscular action from those of connective tissue support. The levator ani muscles are directly connected to the upper surface of the perineal membrane in level III (Fig 2). Loss of connection between the left and right halves...
of the perineal membrane allows the levator ani muscles to move apart, and reuniting the separated ends of the perineal membrane restores the muscles to their normal positions. In addition the endopelvic fascia in level II arises from the superior fascia of the levator ani muscles (Fig 4, B). When muscle contraction is simulated the connective tissue is elevated, indicating that these tissues work together in the pelvic floor as they do in the rest of the body. It should be noted that the levator ani muscles are lateral to the vagina and rectum at all levels and do not come to the midline (Fig 4) and that the muscle fibers themselves do not form a layer that directly influences the rectovaginal support.

**Comment**

This study reveals that posterior compartment support is multifaceted. Rectal position is maintained by the interrelated actions of connective tissue (endopelvic fascia and perineal membrane) and striated muscle (levator ani muscle). Furthermore, connective tissue support differs at different levels. In the distal vagina the connective fibers of the perineal membrane lie in a transverse plane spanning the anterior triangle between the ischiopubic rami, whereas in the mid vagina support comes from parallel fascial sheets in the parasagittal plane connecting to the inner surface of the pelvic diaphragm. Contraction of the levator ani muscles, especially the puborectalis portions, closes the vagina, relieving connective tissue of constant load, whereas the connective tissue must support the structures when the levator ani muscles relax or lose power through neuromuscular damage.

The arrangement of structures in the posterior compartment reveals how the levator ani muscles contribute to posterior wall support. When the muscles have their normal resting tone pressures in the anterior and posterior compartment are balanced and no stress occurs on the level II supports. It is the distal vagina that does not benefit from levator ani closure, and this explains the dense connective tissue seen in the midline union of the perineal membranes through the perineal body. In this...
way the muscular and fascial supports are closely interrelated.

Experienced surgeons recognize different types of rectoceles and have emphasized the need for individually designed treatment. The anatomic findings of this study correlate with the different types of rectoceles encountered. Defects in midvaginal support can give rise to a rectocele, which may occur in the middle of the vagina despite normal levator function and an intact perineal body. On the other hand a perineal rectocele may occur below this region. This condition must be addressed not by plicating the rectovaginal fascia but by retrieving and reuniting the separated fibers of the perineal body. If the perineal body has separated from the level II supports, they must be reunited.

Scientific studies to determine how the type of fascial and muscular damage influences objective treatment outcome have not yet been carried out. Richardson has begun to make progress in this direction by calling attention to different sites of localized fascial disruption involving the rectovaginal septum. The findings of this study expand on these observations to describe the levator ani muscle’s role in posterior compartment abnormalities. This study also clarifies the perineal membrane’s role in distal posterior compartment support. This anatomic picture provides an expanded list of structures that must be assessed in determining the structural defects present when studying posterior compartment problems.

Classification of the numbers and types of anatomic defects present in individual women should help in studying surgical outcome. For example, the success rates of rectocele repair in women with normal and defective levator ani muscles can be compared. I believe that it will be possible to develop magnetic resonance imaging scans capable of displaying specific sites of damage on the basis of the anatomic observations in this study. This objective morphologic information, combined with functional measures of levator strength and connective tissue properties, should permit more systematic study of surgical strategies and outcomes. Such studies would allow the anatomic and physiologic factors associated with operative failure and success to be clarified.

This study adds to previous descriptions of the rectocele anatomy that have emphasized a layer of tissue separating the vagina and rectum (rectovaginal septum). These studies nicely describe the normal rectovaginal septum and its connections to the pelvic wall and perineal body. In addition they describe how detachment between the septum and perineal body results in specific types of rectocele. My findings highlight additional concepts important to posterior compartment support. First, the interaction between the levator ani muscles and connective tissue, wherein levator ani muscle tone relieves connective tissue stress, helps to explain the relationship between prolapse and levator ani muscle dysfunction. Second, level II insertion of the endopelvic fascia into the vaginal wall helps to clarify the nature of interaction between the vaginal wall, pelvic diaphragm and endopelvic fascia. Finally, the relationships among the perineal membrane, levator ani muscles, and perineal body help to define the nature of distal defects.

There has been a long-standing debate as to whether there is a “surgically useful fascia” separating the rectum from the vagina. This debate has existed because surgeons find that they can dissect a layer between the vagina and rectum but histologic observations disclose little if any tissue between the vaginal muscularis and the rectal wall. This analysis of serial sections reveals that most of the endopelvic fascial fibers attach to the vaginal wall and that only a relatively few cross the midline. I believe that the plane dissected surgically and labeled as fascia includes portions of the vaginal muscularis. Studies are ongoing in our unit to address this issue. Breaking strength tests, which measure the amount of force needed to overcome connective tissue supports, should be performed with and without separation of the midline fascial fibers to determine the relative contributions of the endopelvic fascia and the posterior vaginal wall to structural support.

Concepts of the perineal membrane have undergone significant revision in recent years. This membrane has been shown to be a primarily fibrous structure and the muscles previously presumed to be associated with it (deep transverse perineus) have been revised and renamed as the compressor urethrae and urethrovaginal sphincter. An excellent discussion of current concepts of this region can be found in Oelrich.

Anatomic examination of pelvic floor structure in cadavers has limitations. I sought to minimize these inherent problems. Examination of both fresh and embalmed material lessens the likelihood of making errors as a result of embalming artifact, and studying cross-sectional anatomy avoids distortions created by dissection. Loss of muscle tone after death has been addressed by studying some cadavers during the phase of rigor mortis and correcting for sagging caused by loss of muscle function through the use of flotation fixation. The greatest challenge comes in assessing the role of the levator ani muscles. The techniques discussed in the Methods section are not an exact recreation of normal muscular function. They are useful, however, in gaining general insights into the critical interaction between muscle and connective tissue. Now that some general idea about this anatomy has been established, further evaluations in living women with magnetic resonance imaging and measurements made with and without striated muscle paralysis can clarify the nature of interactions between muscle and connective tissue supports.

Great strides have been made in gynecologic surgery
by evaluating the scientific basis of such pelvic diseases as stress urinary incontinence and fecal incontinence. By applying these same principles to rectocele, progress can be made in understanding the pathologic anatomy responsible for the development of this common condition. Once a more clear understanding of the nature of individual defects has been achieved it will be possible to study the relationships among clinical rectocele appearance, symptoms, and the underlying anatomic defects present. Such knowledge will allow specific surgical strategies intended to correct anatomically localized defects to be carried out successfully.

REFERENCES

Discussion

Dr Paul B. Underwood, Jr, Charlottesville, Virginia. This study involved the anatomic dissection of the posterior lower pelvis of 64 fresh or fixed cadavers with macroscopic and microscopic sections to document the findings. In addition, Dr DeLancey evaluated the effect of intraluminal rectal pressure and simulated levator ani muscle contraction on the posterior pelvic support. The article describes anatomic findings only and does not discuss how this anatomy can be used in surgical management of the rectocele.

I must admit that throughout my professional career I believed—and taught students and residents—that a urogenital diaphragm existed that supported the anterior pelvis and was formed by deep transverse perineal muscle with its anterior and posterior fascia. Dr DeLancey has shown us that the deep transverse perineal muscle does not exist and that the previously recognized urogenital diaphragm is almost all connective tissue, with only a few stray muscle fibers. He goes even further and eliminates the term urogenital diaphragm and replaces it with a new name, the perineal diaphragm. He even further destroys my concept of perineal anatomy by stating that there is not any muscle in the perineal body between the vagina and rectum, only condensed connective tissue. He also states that the superior endopelvic fascia that I believed to exist between the vagina and rectum primarily attaches to the posterior lateral vaginal wall, with only a few fibers united between the vagina and rectum. His pictures nicely illustrate these findings. I have certainly revised my opinion of the pelvic support, and I wonder why the older anatomists were so far off in their descriptions.

By default I have become the pelvic reconstructive surgeon at the University of Virginia and perform several such repairs every month. Although the posterior repair is a common operation, it has not been my experience that 1 in 9 elderly women require this surgical procedure. Such an incidence would probably make it the most common operation in elderly women. I do agree that many women require a second repair, but I do not believe that this is because of tissue weakness or lack of a known proper surgical repair procedure. I believe that it is because gynecologists who have not been adequately trained in technique or rarely perform it are doing the procedure. I am extremely concerned that decreased surgical training in some residency training programs and the decreased financial incentive to refer patients because of managed health care plans will only aggravate this problem in the future. Without question there is an art to performing a lasting posterior repair. Long-term experience and grey hair helps. As a card-carrying gynecologic oncologist, I am proud to say that most of us are superb pelvic surgeons; however, I am embarrassed that most are not trained to perform reconstructive pelvic surgery. In my opinion this big void in our oncology training programs has resulted in a loss of a pool of superbly trained gynecologic surgeons performing this potentially technical difficult surgical procedure. I am fearful that as we older self-taught reconstructive pelvic surgeons fade away there will be hiatus before adequate numbers of young reconstructive pelvic surgeons are trained. I sincerely hope that I am wrong because there is an enormous need as the number of older women grows, their life expectancy increases, and their desire to feel normal and function normally is paramount.

I have 2 questions to ask Dr DeLancey. (1) In light of the fact that the anatomic findings illustrate that the su-
perior endopelvic fascia primarily attaches to the lateral vaginal wall, rather than running between the vagina and rectum, why does trimming away the mid vagina and sewing the lateral vaginal wall together in the midline not correct a rectocele? We know that this does not work. Would you please explain this discrepancy with your anatomic findings? (2) Because the perineal body does not contain any muscle but only condensed connective tissue, is the new obstetric trend not to perform episiotomies but rather to “stretch the perineum” going to result in more posterior pelvic relaxations in the future?

Dr Alfred I. Sherman, Bloomfield, Michigan. The causes of rectoceles, of course, are probably multifactorial, and there are many reasons for them to develop. Some women have a greater tendency than others. One area to which you are attending is that of the muscular factors and the collagen factors, the support from below. I think that you also touched on the pressure from above, that we encounter the uterosacral ligaments. What role do they play in the prevention of rectoceles?

Dr Frederick B. Stehman, Indianapolis, Indiana. I noticed that your cadaver specimens represented a broad range of ages. I wonder whether a range of parity was also represented. Can you comment on the differential impact of advancing age versus advancing parity in your anatomic specimens?

Dr Richard C. Bump, Durham, North Carolina. First, you did not mention the relationship between the perineal membrane and the posterior endopelvic fascia or fibromuscular wall of the vagina. That tends to be among the areas on which we concentrate most in repair; do you have any observations on that relationship?

Second, please discuss the rectal wall itself and its importance in the generation of rectoceles. We find a number of patients on evacuation proctograms who have undergone successful posterior repairs but still have rectoceles either posteriorly or laterally or have internal mucosal prolapse of the rectal wall.

Dr Gregorio Delgado, Pittsburgh, Pennsylvania. When we do operations posteriorly, once we open the perirectal space and the rectovaginal septum the first element that we confront is the rectal pillars, and right after that we encounter the uterosacral ligaments. What role do they play in the prevention of rectoceles?

Dr Delancey (Closing). Dr Underwood’s first question was why removal of the posterior vaginal wall is used in rectocele repairs if the tissues do not cross in the midline. The approach described compensates for abnormal anatomy rather than correcting it. Many of our operations do not actually recreate normal anatomy but are still effective. A familiar analogy is that stomach stapling does not address the cause of obesity; it does, nevertheless, correct overweight conditions. The operations that we use are often empirically derived, and the success of these operations can be determined by outcome evaluation. This is preferable to condemning them because they do not restore normal anatomy. I would agree that this operative approach is not a particularly logical way to do things on the basis of the normal anatomy, but there are certain things about the rectocele anatomy that we cannot correct. I believe that much of the cause of rectocele is muscular dysfunction, and that is uncorrectable at this time. So the strategy of compensating for defects is, I think, important in posterior wall support.

The second question concerned the fact that the perineal body does not contain muscle but rather connective tissue. Dr Underwood asked about what happens with the prolonged second stage of labor and attenuation of these tissues. The cervix goes from a diameter of 1 cm to 10 cm, and that connective tissue is able to reassemble to form a normal cervix afterward. I think that as long as there is no mechanical disruption of the connective tissue in the perineal body one could expect a similar kind of recovery.

If you look at the literature on connective tissue injury, injury arises from disruption of connective tissue. I think that in this instance major disruption of this connective tissue in a way that would prevent healing would be the mechanism of long-term obstetric damage. I think that more focused research in looking at the different mechanisms of connective tissue injury at the time of vaginal birth certainly would be fruitful.

Dr Underwood also talked about the term perineal membrane. This is, in fact, the current Nomina Anatomica term. It is not a term that I have coined. Its adoption reflects the improvements in anatomic understanding of this area.

It is interesting that in the 1840s both correct anatomy and incorrect anatomy were described at about the same time. Unfortunately, the incorrect anatomy was perpetuated for many years, but the Nomina Anatomica has now recognized the change in anatomy with the new term perineal membrane.

Dr Sherman pointed out the multifactorial nature of this mechanism that contains both muscle and connective tissue. I agree that this is critically important to understanding the structural unit and the way that muscle and connective tissue interact. He asked specifically about the pressures on the pelvic floor. Pressure, as you know, can be measured in pounds per square inch, so if you increase the number of square inches exposed to the same pressure you increase the force. I therefore believe that we will find that increases in pelvic size place greater forces on the pelvic floor even though the pressures on the pelvic floor may be the same. I think that this will prove an important factor in the genesis of pelvic organ prolapse.

Dr Stehman asked about the issues of differences in anatomy between the young cadavers and the old cadavers and between the nulliparous cadavers and the multiparous cadavers. It is interesting that the neonatal and young material is almost an ideal schema, a very clean anatomy and very little distortion. What you see as you get into the middle-aged and older cadavers, in which parity and age have intervened, is a series of different distortions of that basic schema. We are in the process of
starting to study those distortions to see which are normal age-related changes and which are exceptions to the normal age-related changes. I think that the normal variation is an important point into which we are just starting to get some insight.

Dr Bump made the important observation that there are some lateral and posterior rectoceles. I have been confining my remarks to the anterior rectocele at present. The lateral margins of the rectum abut against the levator ani muscles, as does the posterior part; changes in rectal shape therefore come from changes in the rectal wall, which have been studied by the colorectal surgeons to some extent, or from changes in the levator ani that supports these areas. I think that these will be productive areas of investigation in starting to define the many different types of rectocele. When you have one morphologic picture of a rectocele, you have to answer the question of the anatomic reason that has given rise to that change in shape. I think that magnetic resonance imaging will be the tool to provide that answer.

Dr Delgado called attention to the rectal pillars that are seen at the time of a radical vaginal hysterectomy. I think that these are the upper parts of vaginal support, which go off of the vaginal wall on either side. I also think that these are areas of connective tissue that, with appropriate traction, become visible in the operating room.

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