Site-specific fascial defects in the diagnosis and surgical management of enterocele

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OBJECTIVE: The aim of this study was to assess the surgical feasibility and clinical outcomes of a vaginal enterocele repair that was based on the theory of site-specific defects in the vaginal fascia.

STUDY DESIGN: Seventeen patients during a 2-year period with a diagnosis of enterocele and vaginal vault descensus with or without coexisting rectocele underwent surgical correction with a site-specific fascial defect repair. An enterocele was defined as vaginal wall prolapse seen during the operation in which the peritoneum was found to be in direct contact with the vaginal epithelium, with no intervening fascia. Patients were examined at 4 weeks after the operation and then at 6-month intervals, with site-specific analysis of pelvic prolapse at the vaginal apex and posterior vaginal segment.

RESULTS: Identification and site-specific fascial defect repair of the enterocele were successfully performed in all 17 cases. All patients also underwent a uterosacral ligament vaginal vault suspension, and 15 patients (88%) underwent concurrent posterior colporrhaphy. There were no intraoperative complications. At a mean follow-up of 6.3 months (range 1-17 months), 2 patients (12%) had mild, asymptomatic vaginal vault descensus but no patients (0/17) had evidence of a recurrent enterocele or rectocele.

CONCLUSION: Enterocele correction through a fascial defect repair is easily performed through the vaginal route and is associated with excellent surgical outcomes on short-term follow-up. (Am J Obstet Gynecol 1998;179:1418-23.)

Key words: Culdoplasty, enterocele, posterior vaginal hernia

> *Until more information is available about the cause of the problem (enterocele) and the specific defective anatomy involved, the planning of a rational surgical procedure to correct the situation will continue to be elusive.*
>
> Robert Zacharin[1]

Uterovaginal prolapse has posed a diagnostic and therapeutic challenge to physicians for centuries. As our knowledge of the anatomy and pathophysiology of pelvic prolapse has continued to evolve, a variety of new surgical techniques and procedures have been described, with expectations that they would improve surgical outcomes. Although the diagnosis and surgical correction of anterior vaginal segment prolapse have significantly changed during the last 20 years with greater understanding of midline, transverse, and paravaginal defects in the pubocervical fascia, the diagnosis and management of the enterocele have continued to challenge the gynecologic surgeon.

A variety of culdoplasty procedures for the surgical treatment of enterocele have been described previously. Vaginal techniques include the vaginal enterocele repair[2] and McCall culdoplasty,[3] whereas the abdominal approaches include the Moschcowitz culdoplasty,[4] the Halban procedure, and uterosacral ligament plication.[5] Most advocates of these surgical procedures have described the surgical repair of the enterocele without specifically defining its anatomy or pathophysiology. Previous definitions of the enterocele emphasized clinical features, including etiology, location, associated symptoms, and physical examination findings, with no mention of the specific anatomic defect.

Contrary to the traditional belief that uterovaginal prolapse results from a generalized stretching or attenuation of the pelvic fascial supports, current observations implicate site-specific defects in the origin of pelvic organ prolapse. Richardson et al[6] described the pathophysiology and anatomic basis for cystoceles and rectoceles as caused by specific defects or detachments of the pubocervical fascia or rectovaginal septum, respectively. Recently, Richardson[7] postulated that an apical enterocele results from a defect in the integrity of the endopelvic fascia at the vaginal apex. The vagina, a fibro-

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muscular tube lined with a superficial epithelial layer, is supported anteriorly by the pubocervical fascia and posteriorly by the rectovaginal fascia. In the patient with an intact uterus, the hiatus between the proximal edges these fascial layers is bridged by the cervix and the uterine fundus. In rare cases detachment of the rectovaginal fascia from the posterior surface of the uterus occurs, resulting in a posterior enterocele with an intact uterus. More commonly, in the case of a patient who has undergone previous hysterectomy, failure to reapproximate the pubocervical fascia to the rectovaginal fascia during vaginal cuff closure or subsequent detachment of these 2 fascial layers results in a fascial defect, generally at the posterior vaginal apex (Fig 1). This break in the integrity of the fibromuscular tube results in an area where the peritoneum comes into direct contact with the vaginal epithelium, eventually stretching and resulting in an enterocele noted on clinical examination.

On the basis of this concept of site-specific defects in the endopelvic fascia as the cause of enteroceles, a series of patients underwent intraoperative diagnosis and surgical correction of the enterocele by means of site-specific fascial repair. Our experience with the surgical feasibility and clinical outcomes of the site-specific vaginal enterocele repair is reviewed.

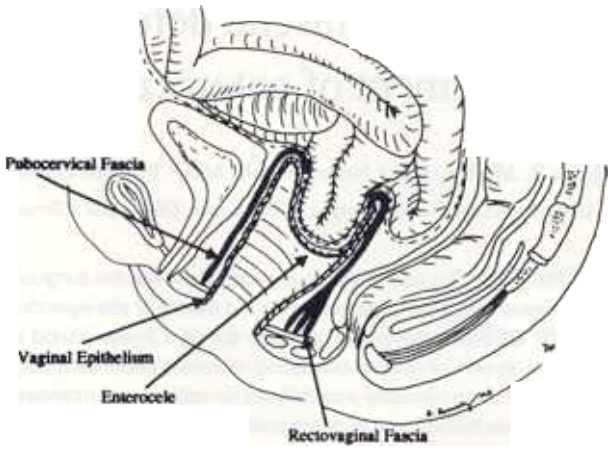

Fig 1. Apical enterocele. Note the separation of the pubocervical fascia of the anterior vaginal wall from the rectovaginal fascia of the posterior vaginal wall.

Material and methods

Seventeen patients with symptomatic enteroceles, with or without concurrent rectoceles, underwent surgical correction between February 1996 and January 1998 by site-specific fascial defect repair to reestablish normal anatomy. Patients were initially evaluated in a urogynecologic office practice, where they underwent a detailed history and physical examination. Vaginal examination with site-specific analysis for pelvic support defects involving the anterior vaginal segment, the cervix or vaginal cuff, and the posterior vaginal segment was performed before the operation. Pelvic support defects were graded with the patient straining in the supine position according to the "halfway system" proposed by Baden and Walker.[8] Patients were placed under general anesthesia and underwent surgical correction with site-specific fascial defect repair as described. Patients were reexamined at the 4-week postoperative visit and then longitudinally at 6-month intervals. At each follow-up visit a symptom diary was reviewed and vaginal examination with grading of the prolapse was performed.

Surgical technique. All patients underwent surgical correction while they were under general anesthesia. Intraoperative vaginal examination was performed with the patient under anesthesia. The apex and posterior vaginal segment were carefully examined for loss of lateral sulci, lack of epithelial rugation, and elongation of the vaginal apex. A rectovaginal examination was also performed to assess for a rectocele and find defects in the rectovaginal septum from its normal points of attachment.

A laparoscopic approach was used to locate and tag with sutures the uterosacral ligaments and a vaginal approach was used to repair the enterocele and rectocele in a site-specific manner. Open laparoscopy was performed in all cases, and accessory ports were placed under direct visualization. The pelvic cavity was examined with a sponge stick or end-to-end anastomosis sizer used to elevate the vaginal cuff. Each uterosacral ligament was found by placing the vaginal apex under tension to the contralateral side. The ureters were located bilaterally. Next, a permanent 2-0 suture was used to tag the uterosacral ligaments at the level of the ischial spine. The needle was cut, the suture was tied with an extracorporeal knot-tying technique, and the free end of the suture was dropped into the abdominal cavity for removal during the vaginal repairs.

Vaginal repair of the enterocele was performed next. A transverse incision was made through the vaginal epithelium at the posterior hymenal ring. The vaginal epithelium was incised in the midline and then dissected off the underlying rectovaginal fascia laterally and proximally with Metzenbaum scissors. As the dissection was carried toward the vaginal apex, a distinct loss of the rectovaginal fascia with a sudden protrusion of peritoneum (enterocele sac) was noted. The enterocele sac was entered and excess peritoneum was excised. Careful examination of the enterocele sac revealed it to be demarcated posteriorly by the edge of the rectovaginal septum and anteriorly by the pubocervical fascia. Dissection of the anterior vaginal mucosa from its underlying pubocervical fascia was performed, beginning at the vaginal apex and extending to anterior vaginal segment. The edge of the pubocervical fascia was located throughout its length at

Table I. Previous pelvic operations in all patients (N = 17)

Procedure	*%*
Abdominal hysterectomy	53
Paravaginal repair	53
Vaginal hysterectomy (includes LAVH)	47
Anterior-posterior repair	29
Retropubic urethropexy	29
Endoscopic needle suspension	29
Posterior repair	21
Culdoplasty	12
Suburethral sling	6

LAVH, Laparoscopically assisted vaginal hysterectomy.

the vaginal apex. The uterosacral ligaments were reattached to the vaginal apex to provide vaginal vault support by passing the previously placed uterosacral ligament sutures through the apical fascia on each side, with an end of the suture incorporating the anterior pubocervical fascia and the other end incorporating the posterior rectovaginal fascia. Next the enterocele was repaired with closure of the fascial defect by reapproximating the pubocervical fascia anteriorly to the rectovaginal fascia posteriorly with a series of 4 to 6 interrupted 2-0 permanent sutures. After closure of the enterocele defect, the uterosacral suspension sutures were tied down, resulting in suspension of the newly created vaginal apex. A laparoscopic approach was used to place an additional uterosacral ligament suspension suture on both sides. The patient was given intravenous indigo carmine, and transurethral cystoscopy was performed to document bilateral ureteral patency.

Posterior colpoperineorraphy was then performed in a site-specific manner, as previously described,[7] with a series of 2-0 permanent sutures. Finally, excess vaginal epithelium was excised and the epithelial edges were reapproximated in the midline with a continuous 3-0 absorbable suture. The vagina was packed with a sterile gauze dressing and an indwelling Foley catheter was inserted for postoperative bladder drainage.

Results

During the 2-year study period 17 women underwent site-specific fascial defect repair and uterosacral ligament vaginal vault suspension through a combined laparoscopic and vaginal approach. The average age of the study group was 64.2 years (range 41-80 years), with a mean parity of 3.2 (range 1-6). Fifteen of the 17 patients (88%) were postmenopausal. All patients had undergone a previous hysterectomy and additional pelvic operations (Table I). Before the operation, 2 patients had a grade 1 enterocele, 14 patients had a grade 2 enterocele, and 1 patient had a grade 3 enterocele. No patients had grade 4 enteroceles. All patients were noted to have grade 1 vaginal vault decensus. Fifteen patients (88%) had a coexisting rectocele. None of the patients were found on office examination to have clinically evident prolapse of the anterior vaginal segment or urethral hypermobility. The presenting primary complaint varied within the study group (Table II).

Table II. Primary reported symptoms of patients with prolapsed vaginal vault with enterocele (N = 17)

Reported symptom	*No.*
Pressure	17
Protrusion	16
Difficulty defecating	9
Impaired coitus	8
Urinary incontinence	3
Voiding difficulty	3

All patients underwent a vaginal enterocele repair with laparoscopically assisted uterosacral vaginal vault suspension. Fifteen patients underwent concurrent posterior colpoperineorraphy. The enterocele with associated defect in the endopelvic fascia was located during the operation in all 17 patients. Site-specific fascial defect repair of the enterocele was successfully performed in each case without intraoperative complications. The average estimated blood loss was 104 mL (range 20-300 mL).

Two postoperative complications were noted in the study group. One woman with a history of cardiac arrhythmia had postoperative atrial fibrillation. Postoperative ileus and pneumonia necessitating readmission 14 days after the operation developed in the other patient. The average length of hospitalization was 1.3 days (range 1-3 days), with 12 patients being discharged after a 1-day postoperative stay.

The mean follow-up period was 6.3 months (range 1-17 months). No patients had evidence of persistent or recurrent pelvic prolapse at the 4-week postoperative visit. On longitudinal follow-up at 6-month intervals, 2 patients were noted to have mild, asymptomatic vaginal vault decensus without signs of associated enterocele or rectocele. Neither of these defects was as large as before the operation. The first patient was a 77-year-old, para 4, postmenopausal white woman with a previous history of abdominal hysterectomy and retropubic urethropexy who underwent vaginal repair of her grade 2 enterocele. She was noted to have persistent grade 1 vaginal vault decensus at 6 weeks and at 12 months after the operation. The second patient was a 69-year-old, para 3, postmenopausal woman with a history of previous abdominal hysterectomy and Burch colposuspension. After vaginal repair of her grade 2 enterocele, she was noted to have good support of the vaginal apex and posterior vaginal segment at her 6-week postoperative visit but grade 1 prolapse of the vaginal

vault subsequently developed 5 months after the operation. Neither patient has required subsequent therapy.

Comment

The effective surgical correction of pelvic prolapse has continued to challenge the gynecologic surgeon for centuries. Although our understanding and surgical treatment of pelvic relaxation have evolved significantly during the last several years, the optimal surgical cure of the enterocele remains controversial. This may be due to several factors. First, the diagnosis of an enterocele is sometimes challenging, and enterocele is often overlooked on clinical examination. Second, a precise understanding of the anatomic defect that is responsible for the enterocele has been poorly reported in previous articles in the literature. Third, pelvic prolapse seems to be a multifactorial process involving multiple anatomic sites in the vagina, making a comparative assessment of surgical outcomes difficult. Despite these limitations, increased understanding of the anatomy and pathophysiology of the enterocele should result in modifications of our surgical approach, which it is hoped will result in improved surgical outcomes.

Historically, both transvaginal and transabdominal culdoplasty techniques have been used to prevent and repair enteroceles. In 1912 Moschcowitz[4] reported his experience in treating rectal prolapse with 6 to 8 permanent sutures placed in a concentric pattern beginning at the base of the cul-de-sac and continuing until the entire pouch of Douglas was obliterated. Although Moschcowitz[4] did not originally describe his technique as a surgical cure for enterocele, this procedure has formed the cornerstone of abdominal repair of enterocele for many years. Nichols and Randall[9] described a similar method of obliterating the cul-de-sac of Douglas as suggested by Halban,[10] who had proposed that a series of stitches be placed in a sagittal direction. Uterosacral ligament plication has also been described in the surgical treatment of the enterocele, with the goal of obliterating the cul-de-sac.[5] Despite these abdominal procedures, enterocele repair has classically been performed through the vaginal approach. Ward's description[2] in 1922 elucidated the cardinal principles of a midline dissection of the posterior vaginal wall with location of the enterocele separate from any rectocele present, excision with high ligation of the enterocele, and reapproximation of the uterosacral ligaments as close to the rectum as possible. Many surgeons have endorsed this basic technique, achieving favorable intermediate but poor long-term results. Other surgeons have advocated culdoplasty for enterocele correction and prevention. McCall's classic posterior culdoplasty,[3] described in 1957, obliterates the redundant cul-de-sac of Douglas by means of a series of continuous sutures incorporating the uterosacral ligaments and the posterior peritoneum, which are brought together in the midline. Despite the variety of procedures described for the surgical correction of enterocele, few descriptions have approached the enterocele as a hernia with associated anatomic repair.

A critical understanding of the anatomy of pelvic supports forms the basis for effective anatomic restoration and surgical correction. The vagina is essentially a flattened fibromuscular tube that is lined by vaginal epithelium and enveloped by endopelvic fascia. The anatomy of pelvic support has been previously described by DeLancey.[11] According to his description, there are 3 principal levels of vaginal support. Suspension of the upper quarter of the vagina (level 1) is provided by the cardinal-uterosacral ligament complex. Lateral attachment of the middle half of the vagina (level 2) is achieved by the paracolpium, extending to the lateral pelvic sidewalls. Finally, the lower quarter of the vagina (level 3) is maintained by fusion of the lower vagina to the urogenital diaphragm and perineal body. In addition to these various levels of pelvic support, the vagina has structural integrity provided by the pubocervical fascia anteriorly and the rectovaginal septum posteriorly. Although the fascial layers have been described anatomically as well as surgically, histologic analysis by Weber and Walter[12] has revealed that they in fact are a component of the smooth muscular wall of the vagina. In light of these findings, the gynecologic surgeon must acknowledge that the pubocervical and rectovaginal fascia are surgical entities that may be used as descriptive terms, rather than histologic entities that reflect their true composition.

Within the framework of this basic understanding of the anatomy of pelvic supports, Richardson[7] postulated that all types of vaginal prolapse, whether anterior or posterior, are actually hernias that represent a break in the continuity of the fibrous tissue tube or a loss of its suspension, attachment, or fusion to adjacent structures. He had previously described lateral, transverse, and midline defects in the pubocervical fascia as the cause of anterior vaginal segment prolapse and advocated a site-specific fascial defect repair in the surgical treatment of cystocele. Recently, he eloquently described the anatomic defects in the etiology of rectocele and enterocele. Structurally, a vaginal enterocele results from direct contact of the peritoneum with the vaginal epithelium with no intervening fascia. Vaginal enteroceles can be classified in 3 groups according to the location of the break in the fibrous tissue tube of the vagina: anterior, apical, and posterior. The apical enterocele, limited to the patient who has undergone hysterectomy, is the most common of these defects. This defect results when there is failure of fusion or reattachment of the anterior pubocervical fascia to the posterior rectovaginal fascia at the vaginal

apex. The resulting fascial defect allows direct contact of the peritoneum with the underlying vaginal epithelium, eventually stretching and clinically resulting in an enterocele. On the basis of this hypothesis, Richardson recommended that surgical correction of the enterocele defect must involve reconstruction of the vaginal fibrous tissue tube, reestablishment of the suspension and lateral attachment of the reconstructed vaginal tube, and excision of the redundant peritoneum and vaginal epithelium.

Our current surgical approach to enterocele repair is based on the anatomic considerations and surgical recommendations made previously by Richardson regarding the site-specific fascial defect repair of pelvic prolapse. Patients with posterior vaginal wall prolapse underwent site-specific fascial defect repair of the rectocele and enterocele, as well as resuspension of the apical supports. Our preliminary experience with this approach demonstrated that the enterocele fascial defect could be accurately located and corrected through the vaginal approach in 100% of the patients studied. Unfortunately, this observational series is limited by the small number of patients enrolled in this study. This small number was due to the strict inclusion criteria, which excluded patients with significant prolapse of the anterior vaginal segment or vaginal vault in an effort to provide a uniform outcomes analysis. Women with pelvic support defects rarely have a single site of involvement, and longitudinal assessment of surgical outcomes is difficult for patients undergoing complex, multisite reconstructive operations. In addition, the short time frame of follow-up (mean 6.3 months) limits the strength of our outcome data. Initial results are encouraging and may predict long-term outcomes. Shull et al[13] reported that absence of any pelvic support defect at the 6-week visit is associated with a 3% likelihood that the patient will require subsequent reconstructive surgery within 2 to 5 years. Because pelvic prolapse is a time-dependent, multifactorial process, long-term follow-up may result in a higher rate and an increased grade of recurrent vault decensus or enterocele. This report is intended as a preliminary description of the surgical technique as well as an analysis of short-term outcomes, and we hope to report long-term results in this series of patients in the future.

Previous articles on surgical techniques in the treatment of the enterocele have focused primarily on obliteration and correction of the posterior cul-de-sac. However, according to Richardson's anatomic description of the enterocele as caused by site-specific fascial defects, the enterocele occurs either at the vaginal apex or along the posterior vaginal wall. Thus the cul-de-sac is a stable structure posterior to the enterocele sac. Previous culdoplasty techniques have been reported to carry suboptimal cure rates in the treatment of enterocele. This may be due to the chance inclusion of either the fascial margins or the uterosacral ligaments during suture placement. A more logical approach seems to be direct location of the site-specific defect and subsequent repair. It is to be hoped that this surgical approach will provide improved outcomes on a long-term basis. In developing a surgical plan for the management of pelvic prolapse, the astute clinician must realize that our understanding of the anatomy and pathophysiology of such complex disease processes is continually evolving and should adapt his or her surgical technique to incorporate these new developments in the hopes of achieving better outcomes.

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Discussion

Dr Ralph Chesson, New Orleans, Louisiana. Independent of this review, I recently attended the Advanced Laparoscopy Training Center in Marietta, Georgia, which facilitated my understanding of the procedure performed by these authors. The anatomic visualization by the laparoscopic expert Bill Saye and anatomist Cullen Richardson at this course greatly enhanced conceptualization of the anatomic surgical repair of an enterocele.

The concept that an enterocele is the loss of continuity of the uterosacral complex with the pubocervical fascia or the rectovaginal septum is the key concept of this article. The authors have selected a well-defined group of patients who had almost pure enterocele, allowing them to isolate the entity of enterocele and its repair. By means of a laparoscopic approach to locate the uterosacral ligaments, a suture was placed through the uterosacral ligament for eventual apical elevation of the vaginal cuff. A vaginal approach was then used to enter the enterocele, locate the pubocervical fascia, and locate the rectovagi-

nal septum. The previously located uterosacral ligaments were then reapproximated to the ipsilateral pubocervical fascia and rectovaginal septum, thereby reestablishing the continuity of these structures. No attempt was made to obliterate the cul-de-sac from the abdominal approach or to plicate the uterosacral ligaments on the vaginal approach. In their short follow-up of these patients there were no significant failures.

The uterosacral ligaments, pubocervical fascia, and rectovaginal septum are all fibromuscular structures that are neither ligament nor fascia. Their strength lies in their combined integrity. The repair of this vaginal hernia with already injured fibromuscular tissue instead of with fascia may jeopardize the long-term success of this procedure. Studies of the integrity of the uterosacral ligaments are necessary to establish how long these tissues will last when used for reconstruction.

Urinary symptoms were present in 6 patients (35%), and only 1 patient (6%) had an enterocele through the introitus. This series of almost pure enteroceles eliminated the patients with severe prolapse, and this restriction may be reflected in the lack of failures. I would like to promote the use of the Pelvic Organ Prolapse Quantification,[1] the classification system endorsed by this Society to help in descriptions used in our journals.

I have several questions for the authors. We have been using 2-0 permanent sutures in our similar vaginal repairs, and we have had recurrent minor problems with granulation tissue. I would like to know what type of permanent suture you are using and whether you have had problems with granulation tissue. Three patients were noted to have reports of incontinence and 3 were noted to have reports of voiding dysfunction. Were urodynamic studies performed on these patients after the prolapse was reduced? Did your procedure relieve their symptoms?

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Dr Miklos (Closing). I agree with Dr Chesson's recommendation that the Pelvic Organ Prolapse Quantification classification system would have been a more descriptive and current way to describe the prolapse in our series, but the grading system previously described by Baden and Walker was the most convenient to use because 3 different centers were involved in collecting data for this study. We acknowledge the role of the Pelvic Organ Prolapse Quantification classification system in the contemporary diagnosis of pelvic prolapse and hope to use it in future studies.

In response to Dr Chesson's comments regarding suture erosion into the vagina, we have found that suture erosion has occurred in a small percentage of patients at the 4-week postoperative visit and is probably related to inadequate closure of the vaginal epithelium at the time of the operation. These patients are initially treated with pelvic rest and transvaginal estrogen cream as long as no signs of infection are noted. If suture erosion is still visible 6 months after the operation, we remove the suture transvaginally. By this time adequate surgical scarring, with resulting repair of the enterocele defect, should have occurred. Because we remove the suture at 6 months if needed, we have had no problems with persistent granulation tissue. We routinely use 2-0 Ethibond (Ethicon, Inc, Somerville, NJ) suture on an MO-7 needle for most of our transvaginal work, being careful not to leave large suture tags that may result in irritation and subsequent erosion of the vaginal epithelium.

In our series 3 patients were noted to have reported symptoms consistent with urge incontinence and 3 patients were noted to have voiding dysfunction. Those patients with incontinence were initially treated with conservative therapy consisting of anticholinergic medication and strict timed voiding. Follow-up evaluation included urodynamic testing, which revealed that none of these 3 patients had genuine stress incontinence. The low incidence of stress incontinence in our series may be attributable to our exclusion of patients with significant anterior vaginal prolapse. The voiding dysfunction in 3 of our patients may have been related to their pelvic prolapse, and urodynamic testing was performed with reduction of the prolapsed vaginal segment with the lower half of a bivalve speculum. Anecdotally, we noted that the patients with preoperative voiding dysfunction had improvement after the operation, but this improvement may have been due to the previously mentioned conservative therapy.

The incidence of postoperative dyspareunia associated with narrowing of the vagina after use of this surgical technique has been negligible. In our series 9 of the 17 patients had sexual dysfunction associated with the prolapse before the operation. From 3 to 4 of these patients had persistent mild dyspareunia after the operation, but I believe that this will improve with time as the surgical site heals and the vagina stretches. This anatomic approach should not result in a decrease in the vaginal caliber. After meticulous dissection of the vaginal epithelium from the underlying rectovaginal fascia, the rectovaginal fascia was exposed. We have found that the rectovaginal fascia does not stretch or shorten and seems to be consistent in its length, approximately 5 to 7 cm, despite the prolapse being as large as 9 to 10 cm outside the vagina. This measurement is largely due to stretching of the vaginal epithelium. Because the rectovaginal fascia is constant in length and width, anatomic reapproximation to the pubocervical fascia does not result in significant narrowing or shortening of the vagina, provided that the patient has an adequate amount of posterior wall fascia. Regarding uterosacral ligament plication, we tend to avoid this procedure because we believe that it is not an anatomic repair and may result in compensatory abnormalities.