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Structural, Functional, and Symptomatic Differences between Women with Rectocele versus Cystocele and Normal Support

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Abstract

Background—Prolapse of the anterior and posterior vaginal walls has been generally associated with apical descent and levator ani muscle defects. However, the relative contributions of these factors to the pathophysiology of descent in the different vaginal compartments is not well understood. Furthermore, symptoms uniquely associated with prolapse in these compartments have not been well-characterized.

Objectives—Compare associations between: 1) apical support, 2) levator ani muscles, and 3) pelvic floor symptoms in women with posterior-predominant prolapse, anterior-predominant prolapse, and normal support.

Study Design—This is a cross-sectional study with two case arms: 60 women with posterior prolapse, 90 with anterior prolapse, and a referent control arm with 103 asymptomatic subjects with normal support, determined from pelvic organ prolapse quantification examinations. Levator muscle defects were graded from magnetic resonance imaging. Vaginal closure forces above resting were measured with an instrumented speculum during maximal contraction. Pelvic floor symptoms were measured via the Pelvic Floor Distress Inventory – Short Form.

Results—Mean Point C location in controls was –6.9 cm [1.5] (mean [standard deviation]); and was higher in posterior prolapse (–4.7 cm [2.7], 2.2 cm below controls) than the anterior prolapse group (–1.2 cm [4.1]; 5.6 cm below controls, $p < 0.001$ for all comparisons). Normal-appearing muscles (i.e., muscle without a visible defect) occurred at similar frequencies in posterior prolapse (45%) and controls (51%, $p = 0.43$), but less often in anterior prolapse (28%, $p = 0.03$ for pairwise comparisons). Major levator ani defects occurred at similar rates in women with posterior (33%) and anterior prolapse (42%, $p = 0.27$), but less often in controls (16%, $p = 0.012$ for both pairwise comparisons). Similarly, there were significant differences in generated vaginal closure forces

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across the three groups, with the prolapse groups generating weaker closure forces than the control group ($p=0.004$), but the differences between the two prolapse groups were not significant after controlling for prolapse size ($p=0.43$). Pelvic floor symptoms were more severe for the posterior (mean Pelvic Floor Distress Inventory score 129) and anterior prolapse groups (128) than the controls (40.2, $p<0.001$ for both comparisons); the difference between the two prolapse groups was not significant ($p=0.83$).

Conclusions—Posterior-predominant prolapse involves almost 3-fold less apical descent below normal than anterior-predominant vaginal prolapse. Levator ani defects and muscle impairment are also less impacted. Pelvic floor symptoms reflect the presence and size of prolapse more than the predominant lax vaginal compartment.

Introduction

Pelvic organ prolapse imposes significant quality-of-life and economic burdens.^{1,2} Of the 225,000 surgical operations performed annually in the United States for prolapse,^{3–5} posterior colporrhaphy is performed in 87%.⁶ Despite the prevalence and obvious clinical importance of rectocele,^{6,7} evidence-based understanding of anatomical and physiological factors specific to this form of prolapse are lacking. Recent reviews have documented significant progress in understanding causal mechanisms of prolapse in general, and anterior vaginal wall prolapse in particular,^{8,9} but our mechanistic understanding of the posterior compartment remains poor.¹⁰ Because surgeons often base their choice of operation on a theory of causation, function, and pathophysiology, evidence-based mechanistic studies are needed to decide between competing theories.

Despite a long history of innovation and investigation, pelvic organ prolapse surgery continues to have an unacceptably high failure rate. Two large, randomized trials by expert surgeons revealed anatomical failure with prolapse beyond the hymen in 25% of subjects.^{11,12} Attempts to improve this using synthetic mesh grafts have met with blunted enthusiasm due to significant complication rates. Surgeons continue to select treatments empirically, based on training, experience, and patient demographics. Ideally, surgery would be tailored to objective anatomic and physiologic patient-specific factors, as is done in selecting cardiac treatment based on echocardiography findings. Improving our understanding of the pathophysiology relevant to different types of prolapse will allow evidence-based, patient-tailored treatments to become viable.

Two causal factors common to anterior and posterior prolapse include: 1) apical descent,^{13,14} and 2) levator ani muscle damage.^{15,16} However, the contributions of these components to posterior wall prolapse are poorly understood. In this analysis, we sought to compare apical support in women with posterior and anterior compartment prolapse, as well as in controls with normal support, with the null hypothesis that apical support is similarly compromised in the two prolapse groups. Our secondary aim was to assess levator ani appearance and function in these three groups, with the null hypothesis that women with posterior and anterior prolapse have similar muscles. Finally, we aimed to compare associations between prolapse and pelvic-floor symptoms.

Materials and Methods

This is a cross-sectional study involving two case arms, women with posterior-predominant and anterior-predominant pelvic organ prolapse, along with a control arm of women with normal vaginal support. The data represent the primary analysis of a University of Michigan Institutional Review Board-approved (IRBMED #HUM00012823) case-control study of women with posterior pelvic organ prolapse and controls with normal pelvic support (NIH P50 HD044406). After the study was planned and underway, it became evident that insights from comparing data between women with posterior compartment prolapse to women with normal support would be greatly enhanced by also making comparisons to women with anterior compartment prolapse. Therefore, similar mechanistic data from a contemporaneous study of the biomechanics of anterior vaginal wall support (NIH R01 HD035665) that had similar goals to the posterior compartment prolapse project were included as well (IRBMED #HUM00043445). The two studies had identical recruitment strategies and testing protocols, and were carried out in the same unit by the same personnel. Stress 3D magnetic resonance imaging (MRI) findings from a subset of this study (R01 HD035665) have been published,¹⁷ so the current data represent a secondary analysis of a larger group of individuals. Written informed consent was obtained from all patients in the original studies. Exclusion criteria for both studies included prior pelvic floor surgery, hysterectomy within the year before enrollment, genital anomalies, or a history of radiation therapy or other factors that would pose a risk for MRI or pelvic floor testing such as metal implants, recurrent urinary tract infections, or immunosuppression.

Subject Selection and Testing

The women with anterior or posterior predominant vaginal wall prolapse were recruited from Urogynecology clinics. Controls were recruited by advertisement and institutional pools of research volunteers. The control subjects were selected to be demographically similar as a group in age, race, and parity to the two prolapse groups. All subjects underwent a pelvic organ prolapse quantification (POP-Q) examination¹⁸ in a semi-recumbent lithotomy position, which was used to determine eligibility and group assignment. To be included in the two prolapse groups, women were selected to represent anterior predominant or posterior predominant prolapse. To be in either group, the location of the contralateral vaginal wall could not be abnormal¹⁹ and had to be at least 1 cm higher than the predominant prolapse. These groups were based on the women's POP-Q examinations as follows: Subjects in the posterior wall prolapse group were those who had distal posterior wall prolapse, with POP-Q point Bp 1 cm or more beyond the hymen (+1) with no anterior or apical compartment point below the hymen, and in whom this was the predominant element of the prolapse (the most dependent point of the POP-Q measurements). Similarly, the anterior wall prolapse group was comprised of subjects in whom POP-Q point Ba descended at least 1 cm beyond the hymen (+1 cm) with no posterior or apical points below the hymen. Controls had all vaginal POP-Q points at least 1 cm above the hymen (-1 cm) and were asymptomatic. The level of the hymen as a criterion for prolapse was chosen because it represents subjects who are outside of the normal range for vaginal support as seen in population-based studies of asymptomatic women,¹⁹ and it is the level at which women become symptomatic from their prolapse.^{20–22}

Subjects with similar anterior and posterior wall support in whom Bp and Ba descended the same amount (Bp = Ba), in whom the cervix was the lowest part of the prolapse, or in whom complete vaginal eversion was detected (POP-Q point C = total vaginal length) were excluded.

Pelvic Floor Imaging

All subjects underwent MRI in the axial, coronal, and sagittal planes using a fast spin proton density technique (echo time: 15–30 ms, repetition time: 2100–4000 ms) in the supine position. Scans were obtained using a 1.5 T superconducting magnet (Signa; General Electric Medical Systems, Milwaukee, WI) or a 3 T system (Philips Medical Systems, Best, The Netherlands). Slice thickness was 4 mm, with 5 mm total image spacing. Full details of our imaging technique are described.²³

Levator ani muscle defects were graded on magnetic resonance scans by two independent examiners blinded to group assignment using a system and its inter-rater reliability as previously described.^{24,25} Briefly, the left and right muscles were scored independently, with scores from 0 – 3 assigned to each muscle. A score of 0 was assigned if there was no visible muscle damage to the pubococcygeal muscle, 1 if less than half the muscle bulk was missing, 2 if more than half was missing, and 3 if no significant muscle could be seen in its normal location. If the two reviewers differed on their score assignments, they reviewed the scans together to determine a final score. The scores from both sides were totaled to provide a final defect score ranging from 0 – 6. These scores were used to categorize the levator defects, as they relate to risk association with prolapse,²⁶ as follows: 0, no defects; 1 – 3, minor defects that involve less than 50% of the expected muscle bulk; 4 – 6, or a unilateral grade 3, major defects involving more than 50% of expected muscle bulk.

Functional Testing

Levator ani muscle function was evaluated using an instrumented vaginal speculum to measure vaginal closure force.²⁷ The force was measured both at rest and during a maximal pelvic muscle contraction. The difference between the vaginal closure force at maximal contraction and at rest was recorded as the augmentation of vaginal closure force during maximal pelvic muscle contraction. In addition, resting genital hiatus and perineal body lengths were measured to gauge hiatus characteristics.

Participants completed a women's health questionnaire that included questions from the Duke Pelvic Floor Dysfunction Distress Inventory (PFDI).²⁸ Survey items pertained to one of three PFDI subscales: The Pelvic Organ Prolapse Distress Inventory (POPDI), the Colorectal-Anal Distress Inventory (CRADI), and the Urinary Distress Inventory (UDI). For those experiencing the symptoms identified in the questionnaire, the level of bother for each symptom was recorded as ranging from "not at all" to "quite a bit." The POPDI, CRADI, and UDI were computed using the methods described in Barber et al, by taking mean values of the respective subscale items and multiplying those means by 25 to arrive at the POPDI, CRADI, and UDI scales. The PFDI was then computed as the sum total of the POPDI, CRADI, and UDI scales.

Statistical Analysis

Descriptive statistics, including means and proportions, are presented for each of the three groups. Simple linear regressions were used to assess differences between anterior prolapse, posterior prolapse, and control groups for continuous measures of interest. For binary measures, simple binary logistic regressions were used. Additional simple regression models were used to assess differences between those with prolapse (anterior prolapse and posterior prolapse groups combined) and controls. Further, multivariable linear and logistic regressions were used to compare the anterior prolapse and posterior prolapse groups while controlling for maximum prolapse size. Additional control variables such as hysterectomy status, body mass index, and vaginal parity were also explored when examining group differences between the prolapse and control cohorts. Posterior prolapse, anterior wall prolapse, and control group differences in levator ani defect classifications were investigated with a simple multinomial logistic regression. The posterior and anterior vaginal wall prolapse groups were further investigated with a multivariable multinomial logistic regression that controlled for maximum prolapse size. Missing data were handled by pairwise deletion, which removed cases with missing values on the measure under analysis and utilized all available women. All analyses were conducted in Stata v. 14.1 (StataCorp. 2013. *Stata Statistical Software: Release 14*. College Station, TX: StataCorp LP.)

Power Analysis

A power analysis was conducted using results from previous studies of levator ani injury in women with pelvic organ prolapse.¹⁵ Chi-square tests of independence with an alpha level of .05 were used for our calculations. Major levator ani injuries were identified in 55% of women with pelvic organ prolapse and in 15.6% of controls.¹⁵ Based on these previous findings, we had >99% statistical power to detect a difference of this magnitude in levator ani defects between our prolapse and control groups given our respective sample sizes. Similarly, assuming that 62% of women with rectocele and 87% of women with cystocele had levator ani avulsion,¹⁶ our sample yielded 94.10% statistical power to detect a similar difference between our two prolapse cohorts.

Results

Characteristics of the three groups and the unadjusted overall p-value are presented in Table 1. Of note, no assessment of statistical significance changed after controlling for hysterectomy status, body mass index, and vaginal parity. As expected from the study design, the locations of the anterior (Ba) and posterior (Bp) vaginal walls differed between the subjects in the three study cohorts.

Apical support, as reflected by POP-Q point C, was lower in both prolapse groups than in controls ($p<0.001$ for both comparisons). It was 2.2 cm lower in the posterior group and 5.7 cm lower in the anterior group than in the group with normal support. The 3.5 cm lower position of point C in the anterior group compared with the posterior prolapse group remained statistically significant when controlling for body mass index, prolapse size, parity, and hysterectomy status ($p<0.001$). Compared with the control group, point D was 2.7 cm lower for the posterior prolapse group ($p<0.001$) and 3.4 cm lower for the anterior group

($p<0.001$). The 7 mm difference in point D between anterior and posterior groups did not reach statistical significance ($p=0.09$).

The genital hiatus and perineal body at rest were also significantly different in overall comparisons across the three groups ($p<0.002$). Pairwise comparisons of genital hiatus between the prolapse and control groups were all statistically significant ($p<0.001$ for both comparisons). The average genital hiatus (from urethra to fourchette) while straining was 2 cm (65%) larger in the posterior group compared with controls, and 2.8 cm (90%) larger than controls in the anterior group. The 0.8 cm difference between anterior and posterior groups was significantly different ($p<0.001$) in unadjusted bivariate comparisons. However, after adjusting for maximum prolapse size, the posterior and anterior groups had similar genital hiatus measurements both at rest and during straining ($p=0.131$).

The perineal body was similar in the posterior prolapse and control groups ($p=0.434$), but 0.6 cm (16%) thinner in women with anterior prolapse as compared to the control group ($p<0.001$). Although perineal body length was 0.4 cm shorter with anterior prolapse than posterior prolapse, this difference was not significant after controlling for maximal prolapse size ($p=0.112$).

Major levator ani defects were more common in both pelvic organ prolapse groups compared to controls (Figure 1). Major defects were found 17% more often in women with posterior prolapse and 26% more often in women with anterior prolapse than in controls (posterior prolapse vs. controls: $p=.012$; anterior prolapse vs. controls: $p<.001$). The rate of major defects did not differ significantly between the two prolapse groups ($p=.267$).

By contrast, the absence of visible levator muscle defects (“normal muscles”) was 17% less likely in anterior than posterior wall prolapse women ($p=.03$). Normal muscles were seen 23% less often in anterior prolapse compared with controls ($p=.001$), while women with posterior prolapse had similar rates of normal muscle compared with controls ($p=0.425$).

Vaginal closure forces at rest and the increase in closure force recorded above the resting force measured during maximum voluntary contraction are shown in Figure 2. Closure forces at rest were similar among the three groups overall ($p=0.373$). The closure force increase during maximum voluntary contraction differed among the three groups overall ($p=0.004$). Women with anterior prolapse generated 35% less vaginal closure force than controls ($p<0.001$), while women with posterior prolapse generated a 22% lower closure force compared with controls ($p=.064$). The increase in closure forces did not differ substantially between the two prolapse groups after controlling for prolapse size ($p=0.43$).

Women’s reporting of their symptoms evaluated by the PFDI is presented in Table 2. Both the anterior and posterior prolapse groups had significantly higher overall PFDI scores than women with normal support (both $p<.001$). Prolapse symptom severity, as measured by the POPDI subscale, was similar between the two prolapse groups ($p=.680$). Women with posterior prolapse had higher CRADI scores than those with anterior prolapse ($p=0.014$). However, this result was not statistically significant after adjusting for maximum prolapse size, body mass index, parity, and hysterectomy status ($p=0.170$). There was not a statistically significant difference in the unadjusted comparison of UDI between anterior and

posterior prolapse groups ($p=0.11$). By contrast, after adjusting for maximum prolapse size and demographic characteristics, the UDI score was significantly higher for the anterior prolapse group as compared to the posterior prolapse group ($p=0.047$).

Comment

We reject our first null hypothesis. Apical descent is more strongly associated with prolapse of the anterior than posterior compartments. These data add compartment-specific information to previous studies showing that apical descent is associated with prolapse in general.¹³ Furthermore, our results are consistent with those of Lowder and colleagues, who demonstrated that lifting the apex during pelvic examination elevates the anterior vaginal wall to a greater degree than the posterior wall.²⁹ The anatomical and mechanical factors responsible for this difference remain to be clarified. Significant advances are being made in understanding the mechanisms of apical support,^{17,30–32} which should help clarify which women will and will not benefit from hysterectomy and/or apical suspension. Given that the apex is, on average, more than 2.5-fold further from normal in anterior than in posterior prolapse, surgeons can expect less improvement from apical suspension in women with rectocele compared with cystocele. This may also explain why apical suspension reduces reoperation rates for recurrent prolapse as compared to anterior colporrhaphy alone, whereas the reoperation rates for recurrent prolapse are similar when comparing women who undergo posterior colporrhaphy or posterior colporrhaphy with concomitant apical suspension.³³

We also reject our second null hypothesis, that levator ani muscle appearance and function are similar in posterior and anterior vaginal wall prolapse. Normal muscles are seen more often in women with posterior prolapse than with anterior prolapse. The differences in levator appearance in our cohorts highlight the nuanced relationship between prolapse and these muscles. Differential levator muscle function in the two prolapse groups is also supported by our finding that the resting genital hiatus³⁴ was different in the anterior and posterior prolapse groups. This complements existing literature identifying a longer genital hiatus in women with prolapse than in controls with normal pelvic support.^{15,35–38} Interestingly, though, maximal voluntary closure force was 20% higher in the posterior than in the anterior prolapse group, although this did not reach statistical significance. These observations highlight the complexity of pathophysiology and suggest that posterior and anterior vaginal wall prolapse are not simply mirror images of one another.

Clinically, this information may provide guidance for surgical prolapse repair, suggesting that concomitant apical suspension may be more important in women with anterior as compared to posterior prolapse. Caution must be taken in interpreting these data, however, as the majority of posterior prolapses in our study were distal rectoceles, and may not be representative of women with enterocele. The degree of apical descent in women with posterior vaginal wall descent would likely be greater if there had been more enteroceles/vault eversions in the study. This finding may also be confounded by the fact that the proportion of women in the posterior prolapse group who had previously undergone hysterectomy was also approximately twice that of the anterior prolapse group. While our statistical analysis suggests that the greater apical descent associated with cystocele as compared to rectocele remains after controlling for hysterectomy status, we cannot be

certain that had we included more women with post-hysterectomy enteroceles in the rectocele group, this difference would have diminished or disappeared.

Conventional wisdom suggests that posterior vaginal wall prolapse is associated with perineal body defects, and that perineorrhaphy is recommended with repair of posterior prolapse.³⁹ However, our data suggest that perineal body lengths are similar in women with posterior prolapse and controls, and are actually shorter in those with anterior prolapse. Women with posterior prolapse have 11% longer perineal bodies than those with anterior prolapse. Although length is only one aspect of a complex three-dimensional structure, it is counter to what is typically believed and therefore deserves further study to clarify how posterior colporrhaphy influences support.

Pelvic floor symptoms are classically taught as resulting from prolapse of specific vaginal compartments. Our data show that women with prolapse have more severe symptoms than controls. However, after adjustment, only lower urinary tract symptoms (UDI) remain significantly different between the anterior and posterior prolapse groups, and these differences are modest at best. These findings are similar to those from previous publications,^{20–22,40–42} confirming that most pelvic floor symptoms classically attributed to prolapse of specific vaginal walls reflect the presence of any bulge, rather than compartment-specific complaints.

Strengths of this study include its validated, objective measurements of levator injury and force-generated strength, large sample size, use of validated symptom questionnaires, and statistical control for potentially confounding factors, e.g., prolapse size and hysterectomy status. Our control cohort was composed of healthy, asymptomatic women recruited from the community, rather than those with pelvic floor disorders who had normal support on exam. Several limitations should also be acknowledged. The study design does not allow for longitudinal assessment of causality or calculation of pelvic floor dysfunction prevalence. Because the question we addressed concerned differences between anterior- and posterior-predominant prolapse, we excluded women with apical-predominant prolapse and those with equal descent of the anterior and posterior compartments, so our data may not apply to all women with prolapse. Finally, this analysis involves subjects from two separate but similarly designed case-control studies that included group matching for cases and controls. As such, the final demographic comparisons between the groups may not reflect perfect matching. Furthermore, because of the strong associations between various demographic characteristics and pelvic organ prolapse (such as race and parity), it was not possible to get ideal matching for all characteristics.

Pelvic organ prolapse does not occur in neatly defined categories. To study differences between compartments, we chose a subset of women with predominant prolapse in either the anterior or posterior compartment. To do this, it was necessary to make some decisions about who would and would not be included. In making these decisions, we chose to study women who had prolapse in either the anterior or posterior compartment but who did not have descent outside the normal range in the other compartment. In defining what is normal, we have chosen to use population-based normal values rather than the POP-Q ordinal staging system, because the divisions between stages are arbitrary and were not chosen

based on any evidence. Stage II, for example, includes women whose support is in the normal range (Aa -1) and those with prolapse (Aa +1). Therefore, our conclusions reflect these selection criteria; other combinations of prolapse (for example, equal anterior and posterior prolapse) will require further study. Finally, our study population is relatively racially homogeneous, which limits the generalizability of our findings.

The presence of any prolapse is correlated with apical descent, levator ani impairment, and pelvic floor symptoms. However, while the type of apical support and levator status appears to correlate to specific compartments, pelvic floor symptoms are more reflective of prolapse presence and size than descent of specific vaginal walls.

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References

1. Jelovsek JE, Barber MD. Women seeking treatment for advanced pelvic organ prolapse have decreased body image and quality of life. *Am J Obstet Gynecol*. 2006; 194(5):1455–1461. [PubMed: 16647928]
2. Subak LL, Waetjen LE, van den Eeden S, Thom DH, Vittinghoff E, Brown JS. Cost of pelvic organ prolapse surgery in the United States. *Obstet Gynecol*. 2001; 98(4):646–651. [PubMed: 11576582]
3. Boyles SH, Weber AM, Meyn L. Procedures for pelvic organ prolapse in the United States, 1979–1997. *Am J Obstet Gynecol*. 2003; 188(1):108–115. [PubMed: 12548203]
4. Shah AD, Kohli N, Rajan SS, Hoyte L. The age distribution, rates, and types of surgery for stress urinary incontinence in the USA. *Int Urogynecol J Pelvic Floor Dysfunct*. 2008; 19(1):89–96. [PubMed: 18043958]
5. Wu JM, Kawasaki A, Hundley AF, Dieter AA, Myers ER, Sung VW. Predicting the number of women who will undergo incontinence and prolapse surgery, 2010 to 2050. *Am J Obstet Gynecol*. 2011; 205(3):230 e231–235. [PubMed: 21600549]
6. Silva WA, Pauls RN, Segal JL, Rooney CM, Kleeman SD, Karram MM. Uterosacral ligament vault suspension: five-year outcomes. *Obstet Gynecol*. 2006; 108(2):255–263. [PubMed: 16880293]
7. Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. *Obstet Gynecol*. 1997; 89(4):501–506. [PubMed: 9083302]
8. Lamblin G, Delorme E, Cosson M, Rubod C. Cystocele and functional anatomy of the pelvic floor: review and update of the various theories. *Int Urogynecol J*. 2016; 27(9):1297–1305. [PubMed: 26337427]
9. Arenholt LT, Pedersen BG, Glavind K, Glavind-Kristensen M, DeLancey JO. Paravaginal defect: anatomy, clinical findings, and imaging. *Int Urogynecol J*. 2016
10. Hale DS, Fenner D. Consistently inconsistent, the posterior vaginal wall. *Am J Obstet Gynecol*. 2016; 214(3):314–320. [PubMed: 26348375]
11. Barber MD, Brubaker L, Burgio KL, et al. Comparison of 2 transvaginal surgical approaches and perioperative behavioral therapy for apical vaginal prolapse: the OPTIMAL randomized trial. *JAMA*. 2014; 311(10):1023–1034. [PubMed: 24618964]
12. Nygaard I, Brubaker L, Zyczynski HM, et al. Long-term outcomes following abdominal sacrocolpopexy for pelvic organ prolapse. *JAMA*. 2013; 309(19):2016–2024. [PubMed: 23677313]

13. Rooney K, Kenton K, Mueller ER, FitzGerald MP, Brubaker L. Advanced anterior vaginal wall prolapse is highly correlated with apical prolapse. *Am J Obstet Gynecol.* 2006; 195(6):1837–1840. [PubMed: 17132485]
14. Summers A, Winkel LA, Hussain HK, DeLancey JO. The relationship between anterior and apical compartment support. *Am J Obstet Gynecol.* 2006; 194(5):1438–1443. [PubMed: 16579933]
15. DeLancey JO, Morgan DM, Fenner DE, et al. Comparison of levator ani muscle defects and function in women with and without pelvic organ prolapse. *Obstet Gynecol.* 2007; 109(2 Pt 1): 295–302. [PubMed: 17267827]
16. Dietz HP, Simpson JM. Levator trauma is associated with pelvic organ prolapse. *BJOG.* 2008; 115(8):979–984. [PubMed: 18503571]
17. Chen L, Lisse S, Larson K, Berger MB, Ashton-Miller JA, DeLancey JO. Structural Failure Sites in Anterior Vaginal Wall Prolapse: Identification of a Collinear Triad. *Obstet Gynecol.* 2016; 128(4):853–862. [PubMed: 27607881]
18. Bump RC, Mattiasson A, Bo K, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. *Am J Obstet Gynecol.* 1996; 175(1):10–17. [PubMed: 8694033]
19. Trowbridge ER, Fultz NH, Patel DA, DeLancey JO, Fenner DE. Distribution of pelvic organ support measures in a population-based sample of middle-aged, community-dwelling African American and white women in southeastern Michigan. *Am J Obstet Gynecol.* 2008; 198(5):548 e541–546. [PubMed: 18455530]
20. Ellerkmann RM, Cundiff GW, Melick CF, Nihira MA, Leffler K, Bent AE. Correlation of symptoms with location and severity of pelvic organ prolapse. *Am J Obstet Gynecol.* 2001; 185(6):1332–1337. discussion 1337–1338. [PubMed: 11744905]
21. Swift SE, Tate SB, Nicholas J. Correlation of symptoms with degree of pelvic organ support in a general population of women: what is pelvic organ prolapse? *Am J Obstet Gynecol.* 2003; 189(2): 372–377. discussion 377–379. [PubMed: 14520198]
22. Bradley CS, Nygaard IE. Vaginal wall descensus and pelvic floor symptoms in older women. *Obstet Gynecol.* 2005; 106(4):759–766. [PubMed: 16199633]
23. Larson KA, Luo J, Guire KE, Chen L, Ashton-Miller JA, DeLancey JO. 3D analysis of cystoceles using magnetic resonance imaging assessing midline, paravaginal, and apical defects. *Int Urogynecol J.* 2012; 23(3):285–293. [PubMed: 22068322]
24. DeLancey JO, Kearney R, Chou Q, Speights S, Binno S. The appearance of levator ani muscle abnormalities in magnetic resonance images after vaginal delivery. *Obstet Gynecol.* 2003; 101(1): 46–53. [PubMed: 12517644]
25. Morgan DM, Umek W, Stein T, Hsu Y, Guire K, DeLancey JO. Interrater reliability of assessing levator ani muscle defects with magnetic resonance images. *Int Urogynecol J Pelvic Floor Dysfunct.* 2007; 18(7):773–778. [PubMed: 17043740]
26. Berger MB, Morgan DM, DeLancey JO. Levator ani defect scores and pelvic organ prolapse: is there a threshold effect? *Int Urogynecol J.* 2014; 25(10):1375–1379. [PubMed: 24788366]
27. Miller JM, Ashton-Miller JA, Perruchini D, DeLancey JO. Test-retest reliability of an instrumented speculum for measuring vaginal closure force. *Neurourol Urodyn.* 2007; 26(6):858–863. [PubMed: 17357114]
28. Barber MD, Kuchibhatla MN, Pieper CF, Bump RC. Psychometric evaluation of 2 comprehensive condition-specific quality of life instruments for women with pelvic floor disorders. *Am J Obstet Gynecol.* 2001; 185(6):1388–1395. [PubMed: 11744914]
29. Lowder JL, Park AJ, Ellison R, et al. The role of apical vaginal support in the appearance of anterior and posterior vaginal prolapse. *Obstet Gynecol.* 2008; 111(1):152–157. [PubMed: 18165404]
30. Luo J, Betschart C, Chen L, Ashton-Miller JA, DeLancey JO. Using stress MRI to analyze the 3D changes in apical ligament geometry from rest to maximal Valsalva: a pilot study. *Int Urogynecol J.* 2014; 25(2):197–203. [PubMed: 24008367]
31. Smith TM, Luo J, Hsu Y, Ashton-Miller J, Delancey JO. A novel technique to measure in vivo uterine suspensory ligament stiffness. *Am J Obstet Gynecol.* 2013; 209(5):484 e481–487. [PubMed: 23747493]

32. Swenson CW, Smith TM, Luo J, Kolenic GE, Ashton-Miller JA, DeLancey JO. Intraoperative cervix location and apical support stiffness in women with and without pelvic organ prolapse. *Am J Obstet Gynecol.* 2016
33. Eilber KS, Alperin M, Khan A, et al. Outcomes of vaginal prolapse surgery among female Medicare beneficiaries: the role of apical support. *Obstet Gynecol.* 2013; 122(5):981–987. [PubMed: 24104778]
34. Ashton-Miller JA, DeLancey JO. Functional anatomy of the female pelvic floor. *Ann N Y Acad Sci.* 2007; 1101:266–296. [PubMed: 17416924]
35. Delancey JO, Hurd WW. Size of the urogenital hiatus in the levator ani muscles in normal women and women with pelvic organ prolapse. *Obstet Gynecol.* 1998; 91(3):364–368. [PubMed: 9491861]
36. Fialkow MF, Gardella C, Melville J, Lentz GM, Fenner DE. Posterior vaginal wall defects and their relation to measures of pelvic floor neuromuscular function and posterior compartment symptoms. *Am J Obstet Gynecol.* 2002; 187(6):1443–1448. discussion 1448–1449. [PubMed: 12501044]
37. Ghetti C, Gregory WT, Edwards SR, Otto LN, Clark AL. Severity of pelvic organ prolapse associated with measurements of pelvic floor function. *Int Urogynecol J Pelvic Floor Dysfunct.* 2005; 16(6):432–436. [PubMed: 15660182]
38. Lewicky-Gaupp C, Yousuf A, Larson KA, Fenner DE, Delancey JO. Structural position of the posterior vagina and pelvic floor in women with and without posterior vaginal prolapse. *Am J Obstet Gynecol.* 2010; 202(5):497 e491–496. [PubMed: 20452497]
39. Katz, VL. Anatomic Defects of the Abdominal Wall and Pelvic Floor. In: Lentz, GM, Lobo, RA, Gershenson, DM., Katz, VL., editors. *Comprehensive Gynecology*. 5th. Philadelphia: Mosby Elsevier; 2007.
40. Tan JS, Lukacz ES, Menefee SA, Powell CR, Nager CW, San Diego Pelvic Floor C. Predictive value of prolapse symptoms: a large database study. *Int Urogynecol J Pelvic Floor Dysfunct.* 2005; 16(3):203–209. discussion 209. [PubMed: 15875236]
41. Miedel A, Tegerstedt G, Maehle-Schmidt M, Nyren O, Hammarstrom M. Symptoms and pelvic support defects in specific compartments. *Obstet Gynecol.* 2008; 112(4):851–858. [PubMed: 18827128]
42. Broekhuis SR, Futterer JJ, Hendriks JC, Barentsz JO, Vierhout ME, Kluivers KB. Symptoms of pelvic floor dysfunction are poorly correlated with findings on clinical examination and dynamic MR imaging of the pelvic floor. *Int Urogynecol J Pelvic Floor Dysfunct.* 2009; 20(10):1169–1174. [PubMed: 19597719]

Condensation

Apical support and levator ani impairment are less severe in women with posterior-predominant, as compared to anterior-predominant, prolapse.

Implications and Contributions

1. This study was conducted to provide better insight into differences in anatomy, physiology, and symptoms of women with pelvic prolapse in different vaginal compartments.
2. Women with posterior-predominant prolapse have less apical descent and levator ani muscle injuries than those with anterior-predominant prolapse. Pelvic floor symptoms are associated with the presence of prolapse, rather than compartment-specific descent.
3. This analysis quantifies the differences in apical descent between posterior- and anterior-predominant prolapse, possibly helping explain why apical suspension is more effective for cystoceles as compared to rectoceles. These data also provide some insight into the underlying mechanisms linking levator ani muscle injury to the development of pelvic organ prolapse.

Figure 1. Proportion of subjects in each study cohort with no defects, minor defects, and major LA defects

Statistically significant differences marked with a line.

LA=levator ani

Figure 2. Vaginal closure forces generated by maximum pelvic floor muscle contraction, stratified by prolapse status
Standard error shown.

Table 1
Demographics and Pelvic Support Characteristics of the Prolapse and Control Cohorts

Characteristic	Control (N=103)	Posterior Vaginal Wall Prolapse (N=60)	Anterior Vaginal Wall Prolapse (n=90)	Overall Unadjusted P-value ^a	Posterior vs Anterior P-value ^b
Demographics					
Age, years	56.0 (10.2)	58.2 (10.6)	57.3 (11.0)	.42	0.55
Height, inches	64.4 (2.5)	64.5 (2.3)	64.7 (2.1)	.76	0.41
Weight, pounds	158.7 (33.3)	169 (30.8)	160.3 (32.1)	.13	0.45
Body Mass Index, kg/m ²	26.8 (5.1)	28.6 (5.2)	27 (5.3)	.08	–
Vaginal Parity	2.2 (1.2)	2.4 (1.1)	2.8 (1.5)	.01	–
Forceps Birth	29 (32.6%)	21 (43.8%)	36 (47.4%)	.14	0.50
Hysterectomy	24 (23.3%)	21 (35.0%)	14 (15.6%)	.02	–
Postmenopausal	74 (72.5%)	45 (75.0%)	61 (68.5%)	.67	0.49
Taking Estrogen	7 (6.9%)	9 (15.5%)	9 (10.3%)	.23	0.96
Replacement Therapy					
White Race	86 (83.5%)	57 (95%)	77 (85.6%)	.12	0.06
Pelvic Floor					
POP-Q Point Ba, cm ^c	–1.9 (.8)	–1.0 (1.4)	3.0 (2.0)	<.001	<.001
POP-Q Point C, cm ^c	–6.9 (1.5)	–4.7 (2.7)	–1.2 (4.1)	<.001	<.001
POP-Q Point D, cm ^c	–8.9 (1.5)	–6.2 (3.4)	–5.5 (2.5)	<.001	0.10
POP-Q Point Bp, cm ^c	–2.0 (.7)	2.1 (1.5)	–1.0 (1.6)	<.001	<.001
Maximum Prolapse, cm ^c	–1.6 (.7)	2.1 (1.5)	3.2 (2.1)	<.001	<.001
Genital Hiatus, cm	3.1 (1.0)	5.1 (1.4)	5.9 (1.6)	<.001	<.001
Perineal Body, cm ^d	3.7 (1.1)	3.5 (.9)	3.1 (1.2)	.002	.053
Vaginal Length, cm	10.3 (1.3)	10.0 (1.3)	9.9 (1.1)	.05	0.36
Post-Void Residual, ml	30.3 (25.1)	59.0 (57.5)	63.2 (65.7)	<.001	0.60

Data are presented as mean (SD), or N (%).

^a p values were derived from comparisons of all three groups. No assessment of statistical significance changed after controlling for hysterectomy status, body mass index, and vaginal parity. For perineal body comparisons, we did not control for parity because birth can affect the perineal body.

q values derived from comparison of the posterior and anterior wall prolapse groups after controlling for hysterectomy status, body mass index, and vaginal parity. For perineal body comparisons, we did not control for parity because birth can affect the perineal body.

POP-Q points are reported as centimeters above (+) or below (–) the hymen and hiatus.

Perineal body measurements were made at rest.

Table 2**Symptoms of the Prolapse and Control Cohorts**

Inventory Type	Control	Posterior Vaginal Prolapse	Anterior Vaginal Prolapse	Overall Unadjusted P-value^a	Posterior vs Anterior P-value^b
Pelvic Floor Distress Inventory	40.2 (29.9)	129.2 (63.5)	128.4 (61.0)	<.001	0.66
Pelvic Organ Prolapse Distress Inventory	5.8 (10.1)	39.4 (24.4)	40.8 (22.6)	<.001	0.99
Colorectal-Anal Distress Inventory	21.9 (17.6)	50.8 (24.9)	41.7 (23.4)	<.001	0.17
Urinary Distress Inventory	12.5 (14.5)	38.9 (28.0)	46.0 (32.7)	<.001	0.047

Data are presented as mean (SD).

^a p values were derived from comparisons of all three groups. All assessments remain statistically significant after controlling for hysterectomy status, body mass index, and vaginal parity.

^b p values derived from comparison of the posterior and anterior wall prolapse groups after adjusting for maximum prolapse size, hysterectomy status, body mass index, and vaginal parity.