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Levator Defects Effect Perineal Position Independently of Prolapse Status

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Abstract

Objective—Determine the effect of levator defects on perineal position and movement irrespective of prolapse status.

Study Design—Forty women from an ongoing study were divided into two groups of 20 women with and without severe levator defects. Prolapse status was matched between groups with 50% having stage III or greater anterior wall prolapse. Perineal structure locations were measured against standard axes on MR scans at rest, maximum contraction (Kegel), and maximum Valsalva. Differences in location were calculated and compared.

Results—In women with levator defects, *independent* of prolapse status: (1) at *rest* the perineal body was 1.3 cm and the anal sphincter 1.0 cm more caudal ($p = 0.01$); at *maximum contraction* the perineal body and the anal sphincter were both 1.2 cm more caudal ($p = 0.01$); with *maximum Valsalva* the perineal body was 1.3 cm more caudal and the anal sphincter was 1.2 cm more caudal ($p = 0.01$). (2) At *rest* the levator hiatus was 0.8 cm larger and the urogenital hiatus was 1.0 cm larger ($p = 0.01$). (3) At *rest* the bladder was 0.07 cm more posterior ($p = 0.02$) and with *maximum contraction* 1.9 cm lower ($p = 0.02$). (4) With *maximum Valsalva* the bladder was 1.5 cm lower and displaced further caudally ($p = 0.03$).

Conclusions—Controlling for prolapse, women with levator defects have a more caudal location of their perineal structures and larger hiatuses at rest, maximum contraction, and maximum Valsalva.

Keywords

levator hiatus; MR; perineal body; prolapse; Valsalva

Introduction

Pelvic organ prolapse is a remarkably common and distressing condition that symptomatically affects 3% of the population¹, and requires approximately 200,000 inpatient surgeries annually.² Recent MR and ultrasound studies have shown that levator ani muscle damage is a clinically important factor associated with pelvic organ prolapse.^{3,4} It

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has likewise been shown that patients with prolapse have enlarged genital hiatuses.^{4,5} Biomechanical studies have long indicated the importance of levator function in maintaining pelvic organ support by closing the levator hiatus and reducing tension on the ligamentous support.^{5,6} Clinically, this is relevant as women with enlarged hiatuses and reduced levator function are more likely to experience prolapse recurrence.⁷

This enlarged hiatus could be either the cause or the result of prolapse. Evident damage and weakening of the levator ani muscle as seen in prolapse might result in the inability of the muscle to hold the hiatus closed.³ On the other hand, the presence of a prolapse that distends the hiatus might stretch tissues, making the hiatus larger. Insight into the difference between cause and effect might be gained from comparing hiatus size between women with and without defects, who all have similar degrees of prolapse. If the hiatal enlargement is due exclusively to levator damage, then only one group (those with levator ani damage) would be expected to have enlargement. If it is the result of prolapse, then both groups would have equal degrees of hiatal enlargement. This study compares measures of hiatus dimensions and perineal position while stratifying for prolapse occurrence and size to isolate the effect of levator ani muscle damage separately from the presence or absence of prolapse.

Materials and Methods

Dynamic MR imaging studies were obtained from an ongoing IRB approved study of pelvic organ support. The parent study recruited women with pelvic organ prolapse where at least 1 vaginal wall (anterior, posterior, or apical) point or the cervix was 1 cm or more beyond the hymen. A group of women with normal support where all vaginal points were 1 cm or more above the hymen was recruited to be of similar age, parity and race. Patients were excluded if they had previous surgery for prolapse or pelvic floor dysfunction, had any findings which would distort pelvic anatomy such as a pelvic mass or history of pelvic radiation, or were unable to complete the clinic exam or MR imaging study. Women who had undergone hysterectomy were eligible if the surgery had been done at least 1 year before enrollment and if the indication for surgery did not include pelvic floor dysfunction (eg, pelvic organ prolapse, urinary incontinence, or fecal incontinence).⁸

From the parent study, we compared 20 women with severe levator ani defects (a muscle defect severity score of 5 or 6) to 20 women with normal levator ani muscles (No Defect Group). Levator ani muscle damage was assessed using a scale for each side of the muscle from 0 (no defect) to 3 (complete muscle loss) that results in a total score from 0 to 6 when both sides are added as outlined in Morgan.⁹ We chose scores of 5 or 6 to represent individuals with severe injury and to include sufficient numbers of women with normal support. Each case and control group for levator injury status was selected to have equal numbers of participants with and without prolapse. The limiting factor to both subject selection and study size was the number of scanned participants who had severe levator ani defects who did not have pelvic organ prolapse. There were only ten of such women available in our database who had normal pelvic organ support.

For the purposes of this analysis, cases of prolapse were defined clinically as women in whom the anterior wall of the vagina extended at least a centimeter below the hymen during straining and who had not previously had surgery for prolapse or incontinence. We have used a definition of prolapse based on findings in a population-based sample of normal women that was used to define a normal range, with a vaginal wall at the level of the hymen as the dividing line indicating the end of the normal range.¹⁰ This is also the point at which women begin to have prolapse symptoms (feeling of a bulge). The POP-Q use of the term prolapse for stage 1 support is not based on either a normal range or the occurrence of symptoms and so we have chosen not to use it. Subjects were those women who had a stage

III or IV prolapse as clinically significant prolapse as discussed by Trowbridge.¹⁰ Controls were asymptomatic women in whom no wall reached the hymen during straining and who had not had prior surgery for prolapse or incontinence.

Scans were selected in the following way. The limiting factor to both subject selection and study size was the number of women with normal support who had severe levator ani defects. There were ten of these women available. Scans for the other case and control groups were selected sequentially to be matched for age, prolapse type and severity, vaginal parity, BMI, and hysterectomy status. Subjects were excluded where the MR scans were not of adequate quality due to motion artifact, parasagittal location of the scan plane, or in cases where the maneuvers were not performed properly such that the subject did not perform pelvic muscle contraction or Valsalva as instructed, despite extensive coaching.

MR imaging protocol and technique was completed as described in Hsu.⁸ In brief, imaging was performed on a 1.5 Tesla system (Signa, General Electric, Milwaukee, WI) using a 4-channel torso phased array coil with the subject in the supine position. The field of view was 30 or 36 cm depending on subject size and weight. Subjects were extensively coached and instructed to perform a maximum contraction (C_{max}) and maximum Valsalva (V_{max}). This work deviated from Hsu's⁸ in that additional scans were analyzed with the C_{max} variable. In this acquisition, the operator instructed the subjects to hold their breath as the scan was initiated; after 5 seconds of imaging at rest, they were asked to contract minimally for 5 seconds, moderately for 5 seconds, maximally for 5 seconds, and then to breathe normally for 5 seconds before ending the acquisition.

All midline sagittal images were analyzed by the same expert examiner, who was blinded to levator damage and prolapse status. Analysis began with the placement of a SCIPP line based x- and y-axis to nullify for the effects of pelvic rotation in the MR scanner (Figure 1). The x-axis consisted of the sacro-coccygeal inferior pubic point line, drawn from the inferior portion of the pubic bone to sacro-coccygeal joint (SCIPP Line). A y-axis was drawn perpendicularly to this line at the level of the pubic bone. Movement differences were assessed in both the y-direction and the x-direction as the distance in mm for each axis. The perineal body point (PB) was marked in the region of the perianal skin immediately anterior to the external anal sphincter. A point was placed at the lowest visible point at the intersection the bladder neck and urethra (B). A point was also placed in the external anal sphincter muscle at the most infero-medial point (EAS) (Figure 1). Measures were taken at rest, with contraction (Kegel), and with Valsalva (Figure 2). Point movements are noted not as vectors, but as differences in either the x- or the y-coordinates between different point locations. Next, a point was placed behind the anterior apex of the levator hiatus (LH) and a line was drawn to the inferior portion of the pubic bone as a measurement of LH length. A line was also drawn from the inferior pubic point to the perineal body as a measurement of the urogenital hiatus (UGH) length. Inter and intra rater reliability of point placement and measurement were established during data collection. We have previously published on the reproducibility of these measures, most notably in Hsu, 2006.⁸

The location of different anatomical landmarks were defined by establishing the (x,y) coordinate relative to the established axes. Distances between points were determined using the formula.

$$d = \sqrt{(x_2 - x_1)^2 + (y_2 - y_1)^2}.$$

Statistical analysis was carried out using SPSS version 16.0 (SPSS Inc., Chicago, IL). Displacements were calculated as above. Bivariate relationships were explored with two-sample *t*-tests calculated between groups on the resting, $C_{\max}$, and $V_{\max}$ images for each structure. Variability of subject positioning in the MR scanner was addressed by measuring the SCIPP line angle relative to the scanner axis during $C_{\max}$ and $V_{\max}$.

Results

Participant demographics are presented in Table 1. There was no statistically significant difference between mean age, BMI, vaginal parity, rate of operative delivery, menopausal status, smoking status, and use of HRT. All participants were Caucasian. Consistent with the study design, pelvic organ prolapse quantification (POPQ) did not differ between the two groups (Table 3).

Women with severe levator ani defects showed lower positions of pelvic floor structures at rest. The perineal bodies of women with defects rested 1.3 cm lower along the y-axis than those of women without defects ($p<.01$). The external anal sphincters rested 1.0 cm lower than those of women without defects ($p<.01$). We also noted that the bladder necks were 0.7 cm further away from the pubic symphysis at rest in women with defects than women without defects ($p=.02$) (Figure 3, Table 4).

During $C_{\max}$ and $V_{\max}$ there were also differences in the pelvic floor positions of women with severe levator ani defects. At $C_{\max}$, the perineal bodies and external anal sphincters of women with defects were both 1.2 cm lower than those of women without defects ($p<.01$). At $V_{\max}$ the bladders were 0.8 cm further from the pubic symphysis in women with defects compared to those without ($p<.01$). At $V_{\max}$ the perineal bodies were 1.3 cm lower ($p<.01$) and the external anal sphincters were 1.2 cm lower ($p<.01$) in women with defects compared to those without. The bladder x-coordinates were not statistically different between groups at $V_{\max}$, however, the bladder was 1.9 cm lower ($p=.02$) in women with severe defects at $V_{\max}$ compared to those without defects. There were also differences in the dynamic motion of women with and without levator ani defects. The bladders of women with defects moved 1.5 cm ($p=.03$) further caudally with $V_{\max}$ than the bladders of those without defects (Figure 3, Table 4).

The size of both the levator and urogenital hiatus was larger in women with defects. At rest, the LH was 0.8 cm ($p<.01$) and the UGH was 1.0 cm ($p<.01$) larger in women with severe levator ani defects than in those without. The anatomic differences in width of the LH and UGH were maintained during $C_{\max}$ and $V_{\max}$. At $C_{\max}$ the LH was 0.7 cm ($p<.05$) larger and the UGH was 1.3 cm ($p<.01$) larger in women with defects compared to those without. At $V_{\max}$ both the LH and the UGH were 1.0 cm ($p<.01$) larger in women with severe levator ani defects compared to women without defects (Table 2). There was no significant relationship between SCIPP length and LH or UGH width.

Comment

This study indicates that levator ani defects effect perineal anatomy independent of prolapse status. In women with similar pelvic organ support, the perineal structures were, on average, about a centimeter lower if there was a concurrent severe levator ani muscle defect. By having the occurrence and degree of prolapse the same in both groups, we were able to isolate the effect of levator injury from the presence of the prolapse. Previous publications have alluded to the relationship between prolapse and levator ani defect. Hsu⁸ and Klingele¹¹ determined with MR and clinical evidence that increasing pelvic organ prolapse is associated with increasing perineal descent. The present study takes this analysis one step further by separating this additive effect; it seems that at least some degree of perineal

descent was a result of levator ani muscle defects, not prolapse. Clinically, this is relevant when assessing patients with both prolapse and perineal descent and can figure prominently in determining a treatment plan.

As the perineal tissues descend, the urogenital and levator hiatuses become enlarged. As with perineal descent, this is the effect of levator ani damage on hiatus width. These hypotheses are not new, having been treated extensively by Halban and Tandler¹² in their landmark work on the anatomy of prolapse published in 1907. However, until recently, making measurements in symptomatic and asymptomatic women to test these hypotheses have not been available. Many works, such as Hsu,⁸ previously determined that the levator hiatus is larger in women with prolapse than those without. Ghetti¹³ concluded that a positive relationship exists between both the levator hiatus and urogenital hiatus size and prolapse severity, while Dietz¹⁴ demonstrated a correlation between LH and pelvic organ descent, even in young nulliparous women. Likewise, in prior work, DeLancey and Hurd⁵ also noted that urogenital hiatus size is positively associated with prolapse size and Hoyte¹⁵ demonstrated that there is increased straining perineal descent in women with prolapse as compared to controls. Our study shows hiatus size is associated with levator ani damage, not prolapse, and suggests a potentially causal relationship between this damage and the development of pelvic organ prolapse as a result of a larger hiatus.

Recent biomechanical research has shed light on the mechanisms of prolapse and possible recurrence. Levator ani muscle impairment allows the hiatus to open too easily, exposing the anterior vaginal wall to increased pressure differentials between abdominal and atmospheric pressure.^{16,17} This provides one explanation for why levator impairment is associated with an increased occurrence of muscle impairment.⁷

What is the clinical significance of these findings? If tests could be developed that identified which patients are most likely to experience recurrent prolapse, then these patients could possibly be selected for operations such as mesh placement, that may reduce prolapse recurrence but are associated with increased complications. Strategies to address operative failure either by augmenting the strength of the connective tissue supports by placing surgical meshes or developing techniques to aid hiatal closure carry additional risks and costs. Therefore, establishing which individual women would and would not need these adjunctive measures would improve success without causing more morbidity for those not at risk of failure. The present line of investigation, by helping to clarify the mechanisms involved in pelvic organ prolapse, should provide information that can lead to future diagnostic strategies that will identify women in whom supplemental surgery is justified.

Several factors should be kept in mind while interpreting the results of this study. Notably, subjects were selected from a limited database of case-control studies on prolapse, they were selected for specific subject characteristics to answer our research question. This allowed us to control for confounding factors, but created a sample that was not representative of the population at large. Additionally, at the present time there are not enough scans to separately analyze different types of prolapse (apical, anterior, posterior) and the role of prolapse type and size must await larger samples. Likewise, our sample was comprised of only Caucasian women and these findings would need to be evaluated in other groups to see if the same findings hold true in those populations. Additionally, pelvic organ prolapse is a multifactorial disease and it is not possible to address all aspects (connective tissue, muscle strength, innervation, bony anatomy, aging, parity) in a single study. The results of this investigation must, therefore, be taken in the broader context that includes hypotheses tested in other parts of the support system.

It would be ideal to perform these MR studies in the standing instead of the supine position. However, both the low field strength of open magnets and space constraints do not allow an adult woman to stand in them. Additionally, this present study only takes into consideration two dimensional midline images and does not account for movement in multiple planes. It is reassuring however that Chi,¹⁸ Torricelli,¹⁹ and Hoyte¹⁵ determined that dynamic MR is an accurate and sensitive imaging tool in a research setting for evaluating the complex anatomy that may accompany pelvic floor dysfunction.

By evaluating the independent effect of levator defects on pelvic anatomy, while holding prolapse presence and size constant between groups, this study proved that both perineal descent and increased hiatus width are associated with a result of defect status. The separate effect of prolapse on hiatus size remains to be further clarified. A causal relationship might be posited that levator defects yield perineal descent and greater hiatus width, thus creating an ideal environment for pelvic organ prolapse to occur. By both preventing the occurrence of levator ani avulsion during labor and delivery and by treating or rehabilitating levator ani defects prior to the development of prolapse, there may be reduced need for later and more difficult correction of pelvic organ prolapse.

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Figure 1. Point Placement & Hiatus Diameters

After placement of the x and y axis. Points for structures are marked at rest, Kegel and Valsalva for each subject: Bladder (B), Perineal body (PB), External anal sphincter (EAS), Levator Hiatus (LH), Urogenital hiatus (UGH)

Figure 2. Point placement and hiatus diameters with movement

Points placed in the same locations at rest, maximum Kegel and maximum Valsalva for each subject: Bladder (B), Perineal body (PB), External anal sphincter (EAS), Levator Hiatus (LH), Urogenital hiatus (UGH)

Figure 3. Displacement with maximum Kegel and Valsalva

Movement of characteristic points with maximum Kegel and Valsalva in women with and without major levator ani defects. BN: bladder neck, PB: perineal body, EAS: external anal sphincter, LH: levator hiatus, UGH: urogenital hiatus. Axis measured in cm

Table 1
Subject Demographics

	No Defect (N=20)	Defect (N=20)	p-value
Age	52.8	55.2	0.5
(years)	(13)	(10.1)	
BMI	25.2	26.6	0.4
(kg/m ²)	(5.4)	(4.9)	
Vaginal parity	2.2	2.6	0.3
(number)	(1.2)	(1.1)	
Vaginal parity	2	2	1.0
(median)			
Forceps deliveries	43%	59%	0.4
(percentage)			
Current use of HRT	26%	25%	0.9
(percentage)			

Data are means and (SD). BMI, body mass index; HRT, hormone replacement therapy.

Table 2
Levator and Urogenital Hiatus Diameters

	Levator Hiatus (cm)		Urogenital Hiatus (cm)	
	No Defect (N=20)	Defect (N=20)	No Defect (N=20)	Defect (N=20)
Rest	6.1 (0.9)	6.9* (0.7)	6.6 (1.2)	7.6* (1.0)
Kegel	6.0 (1.2)	6.7 (0.8)	6.2 (1.0)	7.5* (1.2)
Valsalva	6.7 (1.2)	7.7* (1.1)	7.5 (1.3)	8.5* (0.9)

Data are means and (SD).

*
P<0.01 vs. No Defect

POP-Q**Table 3**

	No Defect n=20	Defect n=20	p
Aa	-0.1 (1.9)	0.2 (1.5)	0.7
Ba	1.1 (3.5)	1.4 (2.4)	0.8
C	-3.6 (4.7)	-2.8 (3.9)	0.6
Bp	-0.8 (3.1)	-1.0 (2.0)	0.8
Ap	-1.5 (1.5)	-1.7 (1.2)	0.7

Data are means and (SD).

Table 4

Data Point Coordinates

	Rest Coordinates		C _{max} Coordinates		V _{max} Coordinates	
	No Defect n=20	Defect n=20	No Defect n=20	Defect n=20	No Defect n=20	Defect n=20
B _X	2.1 (1.1)	2.8 (0.6) *	1.8 (1.2)	2.6 (0.6) *	1.8 (1.7)	1.2 (1.7)
B _Y	0.7 (2.2)	0.0 (1.0)	1.2 (2.2)	0.4 (1.0)	-1.6 (2.8)	-3.5 (2.1) *
PB _X	2.3 (1.1)	2.4 (1.1)	2.2 (1.0)	2.6 (1.2)	2.2 (1.3)	2.2 (1.5)
PB _Y	-3.6 (0.8)	-4.9 (0.8) *	-3.2 (0.8)	-4.4 (1.1) *	-4.7 (1.1)	-6.0 (1.0) *
EAS _X	3.2 (1.3)	3.4 (1.1)	3.3 (1.1)	3.8 (1.4)	3.3 (1.5)	3.3 (1.7)
EAS _Y	-5.5 (0.9)	-6.5 (1.0) *	-4.8 (1.0)	-6.0 (0.9) *	-6.1 (1.2)	-7.3 (1.0) *

Data are means and (SD).

* P<0.05 vs. No Defect.

B: Bladder, PB: Perineal Body, EAS: External Anal Sphincter, C_{max}: Maximum Contraction; V_{max}: Maximum Valsalva