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Hiatal Failure: Effects of Pregnancy, Delivery, and Pelvic Floor Disorders on Level III Factors

Wenjin Cheng[1,2], **Emily English**[1,3], **Whitney Horner**[1], **Carolyn W. Swenson**[1,4], **Luyun Chen**[1,5], **Fernanda Pipitone**[1,6], **James A. Ashton-Miller**[5,7], **John O. L. DeLancey**[1]

[1]University of Michigan, Department of Obstetrics and Gynecology

[2]Beaumont Hospital Dearborn, Department of Obstetrics and Gynecology (current location)

[3]Michigan Medicine, University of Michigan Health-West (current location)

[4]University of Utah, Division of Urogynecology and Pelvic Reconstructive Surgery (current location)

[5]University of Michigan, Department of Biomedical Engineering

[6]University of São Paulo, Hospital das Clínicas (current location)

[7]University of Michigan, Department of Mechanical Engineering

Abstract

Introduction: The failure of the levator hiatus (LH) and urogenital hiatus (UGH) to remain closed is not only associated with pelvic floor disorders, but also contributes to recurrence after surgical repair. Pregnancy and vaginal birth are key events affecting this closure. An understanding of normal and failed hiatal closure is necessary to understand, manage, and prevent pelvic floor disorders.

Methods: This narrative review was conducted by applying the keywords “levator hiatus” OR “genital hiatus” OR “urogenital hiatus” in PubMed. Articles that reported hiatal size related to pelvic floor disorders and pregnancy were chosen. Weighted averages for hiatal size were calculated for each clinical situation.

Results: Women with prolapse have a 22% and 30% larger LH area measured by ultrasound at rest and during Valsalva compared to parous women with normal support. Women with persistently enlarged UGH have 2-3 times higher postoperative failure rates after surgery for prolapse. During pregnancy, the LH area at Valsalva increases by 29% from the first to third

Corresponding author: Wenjin Cheng, Beaumont Dearborn Department of Obstetrics and Gynecology, 18101 Oakwood Blvd., Dearborn, MI 48124, Cell phone: 734-239-1398, wenjinc525@yahoo.com.

Author contributions:
W Cheng: Literature review, data collecting, data analysis, manuscript writing and editing
E English: Literature review, data collecting, manuscript writing and editing
W Horner: Literature review, manuscript writing and editing
CW Swenson: Literature review, manuscript writing and editing
L Chen: Literature review, manuscript editing
Fernanda Pipitone: Literature review, manuscript editing
James A. Ashton-Miller: Literature review, manuscript editing
JO DeLancey: Literature review, data analysis, manuscript writing and editing

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trimester in preparation for childbirth. The enlarged postpartum hiatus recovers over time, but does not return to nulliparous size after vaginal birth. Levator muscle injury during vaginal birth, especially forceps-assisted, is associated with increases in hiatal size; however, it only explains a portion of hiatus variation—the rest can be explained by pelvic muscle function and possibly injury to other Level III structures.

Conclusions: Failed hiatal closure is strongly related to pelvic floor disorders. Vaginal birth and levator injury are primary factors affecting this important mechanism.

Brief Summary:

A narrative review of the hiatal closure mechanism and its relationship to pelvic floor disorders, pelvic floor surgery, pregnancy, and delivery.

Keywords

Urogenital hiatus; levator hiatus; pregnancy; vaginal delivery; pelvic floor disorders

INTRODUCTION

As obstetrician gynecologists, we are responsible for managing birth and caring for women with pelvic floor disorders. During a typical second stage of labor, first the levator and then the urogenital hiatus undergo enlargement that is unprecedented anywhere else in the body. The subsequent failure of a hiatus to close properly because of injuries sustained during vaginal birth is not only a major causal factor in pelvic organ prolapse [1,2], but is also implicated in all pelvic floor disorders [3]. Additionally, a persistently enlarged hiatus is a proven factor strongly associated with operative failure following prolapse repair [4,5]. It is therefore imperative for obstetricians and gynecologists to understand the structures involved in hiatal closure, as well as the failure mechanisms of this important aspect of pelvic floor function. Being unfamiliar with these factors would be like trying to understand the menstrual cycle and its disorders without knowing about the hypothalamic-pituitary-ovarian axis.

MATERIALS AND METHODS

We performed a narrative literature review, identifying 540 studies by searching PubMed for the keywords “levator hiatus” OR “genital hiatus” OR “urogenital hiatus.” We included the studies that discussed hiatal anatomy, measuring, factors associated with hiatal size, and pelvic floor disorders related to hiatus, as well as studies concerning the relationship between hiatus and pregnancy, delivery, levator defect, and pelvic muscle function. We also included studies containing data about other parameters of Level III support as they relate to hiatal enlargement. We searched the studies published before 12/1/2021, and selected studies published afterwards. Hiatal size reported by studies were summarized and reorganized as nulliparous, parous control, prolapse, stress urinary incontinence, the three trimesters in pregnancy, 3-7 months, and 1-2 years postpartum after vaginal delivery, and cesarean delivery. Weighted averages of hiatal size for studies addressing similar questions were calculated for each category.

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RESULTS

HIATAL ANATOMY AND CLOSURE MECHANISMS

There are two hiatuses in the pelvic floor: the levator hiatus (LH) and the urogenital hiatus (UGH) (Figs. 1, 2, 3) [6]. Several anatomical structures in Level III influence their closure [7]. Both hiatuses are bounded ventrally by the pubic bones and laterally by the medial margins of the levator ani muscle. The LH extends dorsally to the sides of the anorectum, while the more caudal UGH extends dorsally to the perineal body. The soft tissue structural integrity of this area involves the pubic portions of the levator ani, perineal membrane, and perineal body [8]. Taken together, these muscular and connective tissue elements form what we call the “perineal complex” that surrounds Level III support. They lie in the lower vagina’s high-pressure zone [9].

Several factors affect hiatal closure: levator ani muscle integrity, “resting” muscle tone, and contraction force controlled through pelvic reflexes, as well as the integrity of the connective tissues of the perineal body and perineal membrane. The levator ani muscle is comprised of three components: pubovisceral (also known as pubococcygeal), puborectal, and iliococcygeal [10].* The pubovisceral has muscle fibers that originate from the dorsal surface of the pubic rami on either side of the pubic symphysis and insert into the vaginal wall, the perineal body, and the inter-sphincteric groove of the anal sphincters, with some fibers passing behind the rectum (Fig. 4) [10,11]. The puborectalis originates bilaterally from the pubic bone lateral to the pubovisceral muscle and from the perineal membrane and forms a sling behind the rectum. These two aspects of the levator form the major muscles closing the LH and UGH, as seen on ultrasound and cadaver dissection [12,13]. The iliococcygeal muscle arises dorsal to the two pubic portions. It originates from the arcus tendineus levator ani on either side of the pelvis and forms a sheet-like structure between the anus and sacrum. The levator ani muscle is innervated by the nerve to the levator ani on its inner surface from the sacral plexus, with a possible branch from the pudendal nerve [14]. Connective tissue structures, specifically the perineal body and perineal membrane (Fig. 2) [8], also form part of the Level III perineal complex. Because the medial aspects of the levator ani muscles are attached to the perineal membrane and perineal body, rupture of this connection can lead to diastasis of the muscle that manifests as increased LH and UGH size. It should be emphasized that these structures are all intimately connected with one another and that alterations in one aspect of this system affects all other parts.

SYSTEMS FOR MEASURING THE HIATUS

Levator Hiatus—The use of 3D transperineal ultrasound to measure the LH was first described by Dietz et al [15]. They defined the minimum LH dimension from the pubic symphysis to the anterior border of the anorectal junction. Thus, in the transverse plane, the LH anterior-posterior (AP) diameter, width, and area can be assessed. MRI can also be used to measure the LH, but the differences in imaging modality can result in different size estimates, with MRI measurements consistently larger than ultrasound (5.0±0.8 cm vs 4.4±0.6 cm) [16]. Clinical measures of hiatal diameters on physical examination use

*In discussing levator injury some authors in the ultrasound literature refer to the pubovisceral muscle as the puborectal muscle.

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different landmarks and so differ from imaging measurements. On physical exam, the distance from the urethra to the anus has been used as a surrogate for LH size, but is only moderately correlated with AP diameter (r=−.67) and LH area (r=.72) on ultrasound during Valsalva [17].

Urogenital Hiatus—The UGH is the opening through which prolapse occurs and is part of the Pelvic Organ Prolapse Quantification (POP-Q) system [18]. Measurement of the UGH with the POP-Q is “from the middle of the external urethral meatus to the posterior midline hymen.” On MRI, the UGH is measured as the distance from the lower edge of the pubic symphysis to the anterior perineal body [19].

Depending on the question at hand, the LH and UGH can be measured either at rest, during Kegel, or with Valsalva. Among patients with prolapse, measurements taken during Valsalva were found to have a stronger association with signs and symptoms of prolapse than resting measurements [20].

NORMAL HIATUS SIZE

“Normal” hiatus size can be defined using measurements from one of two groups: 1) nullipara or 2) asymptomatic parous women without prolapse. Several studies have used these groups to establish what may be regarded as baseline hiatus size, as in Table 1 and the first two columns of Fig. 5. LH measurements were larger in nulliparous women on MRI, with a weighted average LH AP diameter of 4.8±0.7 cm at rest, 5.2±0.8 cm during Valsalva, and 4.7±0.8 cm with Kegel (Table S1).

FACTORS ASSOCIATED WITH HIATUS SIZE

Age, ethnicity, and BMI are all known to affect hiatus size. For example, in nullipara aged 52-85 (n=10), the LH area measured on 2D endovaginal ultrasound was 11% larger compared to those aged 18-40 (n=12), but statistical significance was not reached—perhaps because of a small sample size [21]. In nullipara, MRI-based UGH was 30% larger at rest and 10% larger during Valsalva in women >70 years when compared to those ≤40 years [22]. Hiatus shape also changes with aging. The LH area was shown to be more oval in older women on ultrasound, but more circular in younger women [21]. On MRI, a “U”-shaped LH is more common in older women, while a “V” shape is more common in younger women [22].

Hiatus size also differs with ethnicity. Among nulliparous women aged 18-39, Black women had the largest LH area during Valsalva, followed by White and South Asian women (18.1±5.0 cm vs 15.2±5.0 cm vs 14.6±4.7 cm, P<.0001) [23,24]. In the same study, no difference was found for UGH on physical exam. Women with symptomatic prolapse followed the same pattern for both LH and UGH, with Black women having the largest measurements, followed by Caucasian and South Asian women (LH area at Valsalva: 40.2±9.6 cm vs 37.6±9.8 cm vs 34.2±8.3 cm, P=.006; UGH: 6.5±1.0 cm vs 5.8±0.8 cm vs 5.5±1.0 cm, P=.001). However, Black women had significantly higher vaginal parity in this study, while White women were more likely to have a history of surgery for prolapse [25]. Compared to pregnant nulliparous East Asian women, pregnant nulliparous Caucasian

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women were found to have a 27% larger LH area at Valsalva [26]. The association between ethnicity and hiatus size was significant even after controlling for BMI and age [23,26]. This effect might partially originate from the ethnic differences in bony pelvis [27] or pelvic floor muscle anatomy. For instance, there was 14% and 18% greater pelvic muscle thickness and area in East Asian women compared to Caucasian women [26]. This difference underscores the importance of clarifying ethnicities when we discuss hiatus size in the future.

Increased body weight and BMI have been associated with larger LH. Greater body weight was associated with larger resting LH area and LH AP diameter (r=.39 and r=.38, respectively), while greater BMI was correlated only with a greater AP hiatal diameter (r=.35) [28].

HIATAL FAILURE AND PELVIC FLOOR DISORDERS

Pelvic Organ Prolapse—During the two decades after giving birth, women with an enlarged UGH on physical exam (≥3.5 cm compared to ≤2.5 cm at Valsalva) have increased hazard ratios for prolapse of 11.7 (95% CI: 7.51-18.4), for stress urinary incontinence of 2.3 (95% CI: 1.57-3.40), and for anal incontinence of 1.6 (95% CI: 1.12-2.27) [3]. Overall, a failure of the structures surrounding the hiatus to maintain adequate closure is strongly related to pelvic organ prolapse (Figs. 2 and 3). Women with prolapse have larger average LH AP *diameters* than parous women with normal support measured by ultrasound, not only at rest (6%) and during Kegel (7%), but especially during Valsalva (14%) (Fig. 5, Table 1). Similarly, women with prolapse have larger average LH *areas* at rest (22%), with Kegel (20%), and with Valsalva (30%). With a proposed cutoff for defining an enlarged LH area of 25 cm^2 during Valsalva, 51% of women with Stage II prolapse or larger exceeded this value [29]. Women with prolapse also have 36-81% and 22-50% larger UGH AP diameter on physical examination at rest and Valsalva, respectively, compared to parous women with normal support [17,30-36]. On MRI, UGH AP diameters are also larger at rest (15%), during Valsalva (22%), and with Kegel (7%) in women with prolapse versus those without [37]. When comparing women with prolapse to nulliparous women without prolapse, the differences are even larger (Table 1).

An increase in prolapse size is associated with an increase in hiatus size at rest and during Valsalva. Women with Stage III or IV prolapse had a 32% and 71% larger straining UGH, respectively, compared to women with Stage II prolapse [36]. Some studies found UGH on physical exam were similar or even smaller in Stage IV prolapse compared to Stage III prolapse [32,35], which might be explained by a smaller dilating effect with complete protrusion [38]. Both LH (r=.64, $P \leq .001$) and UGH (r=.81, $P \leq .001$) size at Valsalva were shown to be correlated with prolapse size, as measured by the area protruding below the hymen on sagittal MRIs [39]. However, this pattern does not apply to nulliparous women. At Valsalva on physical exam, parous women (N=1,437) with Stage II, III, and IV prolapse had a 29%, 75%, and 25% larger UGH, respectively, while among nulliparous women (N=154), UGH did not vary significantly (Stage 0/I 2.4±0.6 cm, Stage II 2.8±0.8 cm, Stage III 2.8±0.8 cm, Stage IV 2.6±1.7 cm) [35].

Hiatus size also differs depending on the type of prolapse present. For example, compared to posterior prolapse, women with anterior prolapse had a larger UGH at rest (21%) with

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Valsalva (14%) and Kegel (26%) on MRI, but not in all maneuvers for LH [37]. This might be partially explained by the result from the same study that a major levator defect is more commonly seen in patients with anterior wall prolapse (42%) compared to patients with posterior prolapse (36%), which is another important factor associated with enlarged hiatus [35] that will be discussed later in this review. There is also a definite association between hiatus size and apical support. A study addressing this question found a moderate correlation between UGH and loss of apical support (r=.46-.59) measured with POP-Q [36]. A stronger correlation was found between UGH and apex location (r=.84, P=.001) when measured on MRI [40]. Women with posterior-predominant prolapse had three-fold less apical descent compared to those with anterior-predominant vaginal prolapse [31], highlighting a difference that still needs to be explained from a mechanistic point of view.

Whether an enlarged hiatus causes prolapse or prolapse causes an enlarged hiatus has long been debated. A longitudinal study of 1,200 parous women found that UGH enlargement precedes the development of prolapse—suggesting, but not proving, a causal relationship [1]. The median estimated time from the first vaginal delivery to development of prolapse at least 1 cm below the hymenal ring was 33.4 years for women with a 3 cm UGH and 5.8 years for women with a UGH ≥4.5 cm [41]. UGH enlargement was found to happen faster among women who later developed prolapse compared to women who did not (0.56 cm vs 0.15 cm per 5-year period, P<.001) [1]. UGH enlargement and faster enlargement are both characteristics of women who eventually develop prolapse.

Stress Urinary Incontinence (SUI)—There is less hiatal enlargement in women with SUI compared to women with prolapse. Compared to normal women, women with SUI had a 15% larger LH area both at rest and during Kegel, while women with prolapse had a 32% larger LH area at rest and during Kegel, respectively [42]. Average LH area increases in women with versus without SUI are −8% at rest, 11% during Valsalva, and −5% during Kegel (Fig 5) [43-47]. Similarly, women with SUI have an 18% larger UGH on physical examination (2). SUI during pregnancy and postpartum has been associated with a larger hiatus. A longitudinal study during pregnancy showed that the proportions of women with SUI at 12 weeks pregnant, 36 weeks pregnant, and 6 months postpartum were 19%, 47%, and 38%, respectively; women with SUI had 10%, 7%, and 8% larger LH areas during Valsalva at each of those time points [43].

Fecal/Anal Incontinence—There are fewer studies about the relationship between fecal incontinence and hiatus dimensions. The evidence points to a weaker correlation between fecal incontinence and hiatus size compared to that between hiatus size and prolapse or SUI. A positive correlation was found between LH area and fecal incontinence symptoms assessed by the Cleveland Clinic Florida Incontinence Scoring System (ρ=.43, P=.76; N=52) among patients with a previous vaginal delivery and fecal incontinence. A follow-up study from the same team with a larger sample size found a lower correlation between LH area and fecal incontinence symptoms, but it remained statistically significant (ρ=.26, P=.03; N=84) [48,49]. In a small cohort study using MRI to compare hiatus size between elderly women with and without fecal incontinence, UGH and LH were not found to be significantly different [50].

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SURGERY AND THE HIATUS

Enlarged Hiatus and Operative Failure—Prolapse recurrence has been associated with a preoperative enlarged hiatus and a persistently large hiatus after surgery. Recent studies have evaluated the effect of hiatus change before and after surgery. For example, 4-6 weeks after sacrocolpopexy, patients with a persistently enlarged UGH (≥4 cm) had a higher failure rate (14.0%) than patients with an improved UGH (5.7%) and patients with a stable normal UGH (4.0%) [4]. Likewise, after native tissue vaginal vault suspension, composite anatomic failure occurred significantly more often in the persistently wide UGH group (51.3%) compared to the improved group (16.6%) and the stably normal group (6.3%) [5].

The LH area at Valsalva measured by ultrasound before anterior colporrhaphy was 12% larger in women with postoperative failure one year after surgery, with odds ratios (per 1 cm^2) of 1.06, 95% CI: 1.01-1.11), which were not significant when measured at rest or Kegel [51]. In this study, 40% and 50% of the patients received concomitant posterior and apical repair, respectively. A more recent study found that a larger LH area at Valsalva (≥25 cm^2)and the presence of levator avulsion are independently associated with both anatomic and symptomatic failure one year after anterior colporrhaphy with or without posterior repair. Odds for anatomic failure with an enlarged hiatus were 2.5 (95% CI: 1.63-3.88) and for levator avulsion, 2.0 (95% CI: 1.3-3.0). Odds for symptomatic failure with an enlarged hiatus were 2.4 (95% CI: 1.1-6.5) and for levator avulsion, 2.6 (95% CI: 1.1-6.0) [52]. The anatomic recurrence in this study was defined as at or below POP-Q Stage II. Another study using MRI showed that with every 1 cm increase of preoperative LH at rest, there would be an 8-fold increased recurrence rate within a mean follow-up of 13 years [53].

How Much Does Surgery Affect Hiatus Size?—The fact that an enlarged hiatus is associated with operative failure raises the question of how surgery might reduce hiatal size. There are two ways in which an operation might affect the hiatus. First, surgical repair eliminates the dilating effect of the prolapse during Valsalva that can push the hiatus open. Once the prolapse is gone, the hiatus may recover and become smaller both at rest and during Valsalva, as it does in some pessary patients [54,55]. Second, some techniques of posterior colporrhaphy aim to reunite separated perineal structures to reduce hiatus size [56,57]. Additionally, a woman with a larger hiatus may be more likely to have a posterior reconstructive procedure than one with a normal hiatus so that without randomization, differences in outcome may not be apparent.

When one considers the change in anatomy with posterior colporrhaphy, with sutures placed at the level of the perineal body, it is not hard to imagine UGH shortening after the procedure. Evidence for this comes from POP-Q being performed right before and after posterior repair when patients were still under anesthesia, when the UGH was found to have decreased 0.6 cm after surgery (13.5%) [58].

Apical suspension has also been shown to reduce the UGH when measured during Valsalva postoperatively, regardless of concomitant posterior colporrhaphy. Patients who received sacrocolpopexy without concomitant vaginal wall repairs had a 14% decrease in the UGH at rest upon physical exam 14 weeks after surgery [59]. Reductions of 42% and 35% in the UGH measured with POP-Q were also achieved eight months postoperatively in patients

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who received apical suspension with and without Level III repair, such as posterior repair or perineorrhaphy [60].

There is also a potential additive effect on UGH size from combined procedures. Patients who received sacrocolpopexy without posterior colporrhaphy had a 1.3±1.1 cm decrease in the UGH, while patients with concomitant posterior colporrhaphy had a 2.0±1.2 cm decrease in the UGH (P=.03), with a mean follow-up length of 9.8 months [61]. Among women who underwent sacrospinous ligament fixation, postoperative UGH size was similar between those with and without posterior repair (2.7±1.0 cm vs 2.3±0.8 cm, P=.177), likely due to the baseline UGH being significantly larger in women who received concomitant posterior repair (5.0±1.5 cm vs 4.0±1.2 cm, P=.02) [62]. Similarly, posterior repair with apical suspension was not shown to be associated with a higher rate of anatomical success compared to patients who received concomitant posterior repair two years after surgery, likely also due to the larger baseline hiatus in women who received posterior repair [63].

Compared to the UGH, there are fewer studies examining LH change after surgery. Unlike the UGH, the LH AP diameter was not found to be decreased 14 weeks after sacrocolpopexy [59]. Studies with mixed prolapse surgery types showed that the LH area at rest decreased by 21% one month after surgery, while the LH area at Valsalva decreased by only 9% 3-12 months after surgery [64,65]. A novel puborectalis sling placed at the completion of prolapse surgery was shown to decrease the LH area at Valsalva by 30% three months after surgery; these results were sustained two years after surgery [66]. The procedure had a 66% anatomic recurrence rate (POP-Q ≥Stage II in the anterior/posterior, and ≥Stage I in the central compartment) and 30% symptomatic recurrence rate 2.5 years after surgery [66]. This study recruited women with a pre-operative LH area during Valsalva ≥35 cm^2, with an average of 44 cm^2, which might explain the high recurrence rate. The criteria used in that study were also considerably stricter than the evidence-based standard established by the NIH Pelvic Floor Disorders Network for anatomical success and may have influenced the results [67,68].

DELIVERY MODE, LEVATOR DEFECT, MUSCLE STRENGTH, HIATUS, AND PROLAPSE

Hiatus Alterations During Pregnancy—The LH and UGH both increase in size during pregnancy in preparation for the remarkable enlargement that must occur during vaginal birth (Fig. 5, Table 1). For example, from the first to third trimester, the LH area measured by ultrasound increased on average by 13% at rest, 10% during Kegel, and 29% during Valsalva [43,69-76] (Table 1). The mechanism of hiatus enlargement during pregnancy is not fully known, but explanations might include changes in the muscle [77], increased connective tissue “stretchiness” [78], and increased abdominal pressure [79]. Since studies reveal that women with a smaller LH are more likely to undergo instrumented delivery and to sustain levator avulsion [74,80], these are relevant issues.

Hiatus Size After Vaginal Delivery—The unprecedented enlargement of the hiatus during vaginal delivery first stretches the LH, then the UGH during the second stage. Hiatal size gradually diminishes over time during recovery. Biometric data for Caucasian neonates was used to calculate the muscle fiber stretch in the LH during vaginal delivery, which

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ranged from 25-245%, with an average of 107% when predicted lengths during birth were divided by hiatal measures taken at Valsalva [81]. Similarly, a model-based study using *in vivo* MRI data reported the UGH stretch to be 255% [82].

Immediately after either spontaneous or instrumented vaginal deliveries, LH measures at Valsalva and Kegel are larger than before delivery, then gradually decrease during recovery, while measures made at rest show mixed results (Fig. 6, Table 1, Tables S5 and S6). The decrease in hiatus size after vaginal delivery mostly happens in the first 4-6 months [19,83], and is most rapid in the first two weeks [84].

Current evidence shows that while LH size decreases gradually after vaginal delivery, it does not return to first trimester size and does not normalize to nulliparous metrics in all women, especially when measured at Valsalva. When weighted averaged LH areas 3-7 months after delivery are compared to first trimester measures, they are larger by 15% at rest, 29% during Valsalva, and 11% with Kegel (Table 1). The values during rest and with contraction do not differ to the same extent as with Valsalva. Compared to the first trimester, LH areas 1-2 years after vaginal delivery are slightly smaller at rest (3%) and Kegel (5%), but 11% larger during Valsalva by weighted average (Tables S2 and S6).

Increased vaginal parity has been associated with further increase in hiatus size beyond that found after the first birth. The mean LH area during Valsalva of women with no history of vaginal delivery, one vaginal delivery, and four vaginal deliveries were 21.5 cm^2, 29.1 cm^2, and 30.9 cm^2, respectively. Multivariate analysis controlling for confounding factors such as age, BMI, and surgical history, found the hiatal areas were 29.2 cm^2, 30.5 cm^2, and 32.8 cm^2, respectively [85].

Birth-related Levator Injury and Hiatus Size—Tears to the pubovisceral portion of the levator ani have been reported to be 15%, 21%, and 52% following spontaneous, vacuum, and forceps-assisted vaginal delivery in a recent published systemic review [86]. The tears are found in the substance of the muscle and, with more severe injuries, involve tearing of the origin of the pubovisceral part of the levator muscles, resulting in complete avulsion and sometimes complete atrophy of the muscle with time [87]. These injuries affect hard tissue in the form of fracture of the pubic rami, as well as tearing of soft tissue of the pubic symphysis and midline separation of the perineal membrane and perineal body, although further research is needed to properly visualize and document these latter injuries [87-89].

Women with levator injury have a larger hiatus both in the short and longer term. For example, 3-7 months after vaginal delivery, ultrasound imaging has confirmed women with levator avulsion as having larger LH areas at rest (2-11%), during Valsalva (4-16%), and during Kegel (14-18%) compared to women without levator avulsion [71,74,76]. Likewise, measurements made 11 years after vaginal delivery continue to show larger median LH areas in women with avulsions: 32% at rest, 38% during Valsalva, and 48% larger during Kegel, as well as larger UGH AP diameters at rest (25%) and during Valsalva (33%) compared to women without levator injury avulsion [90].

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There is conflicting evidence about the degree to which women with levator defects can close a hiatus upon volitional contraction of their remaining levator muscles (iliococcygeus and puborectalis). Since the iliococcygeal and puborectal muscles remain intact even with complete pubovisceral muscle tear, there is reason for why this could occur [91]. For example, women with unilateral major levator defect had a significantly greater LH area reduction compared to women with bilateral major levator defect (18% vs 9%, P=.04), but the hiatal reduction was similar between subjects without and with only unilateral major levator defect (P=.9) [92]. No significant difference in hiatus size reduction was found with volitional levator contraction [37,90,93]. In most studies, the LH and UGH both enlarge more with Valsalva in the presence of a levator defect. In women with unilateral levator avulsion, the hemi-LH area at Valsalva was 14% larger on the injury side compared to the intact side [92,94]. On MRI, the LH and UGH AP diameters lengthening during Valsalva were 44% and 72% larger, respectively, in women with major defects compared to women with minor or no defects [37].

Pelvic Muscle Function and Hiatus Size—As discussed above in the section on Hiatal Anatomy and Closure Mechanisms, stronger pelvic floor muscles are one of several factors associated with smaller hiatal dimensions. Vaginal resting pressure, maximal contraction force, and pelvic muscle endurance are three parameters commonly used to assess pelvic muscle function.

In nulliparous pregnant women at rest, the LH area on ultrasound was found to be moderately associated with vaginal resting pressure (r=−.45), but not with maximal contraction force or muscle endurance [95]. The LH area during contraction was moderately correlated with maximal contraction force (r=−.37) and endurance (r=−.35). In a study of women with and without prolapse, the LH AP diameter at rest on MRI was found to be moderately associated with maximal vaginal contraction force (r=−.35), but not with resting closure force [37]. In women with prolapse, the LH area at rest measured by ultrasound was found to be moderately correlated with vaginal resting pressure (r=−.46), maximal contraction force (r=−0.41), and pelvic muscle endurance (r=−.40); 26% of the variance in the LH area at rest could be explained by resting pressure and maximal voluntary contraction, while vaginal resting pressure contributed more (β:−.39, P<.001) than maximal contraction force (β:−.25, P=.01) [96]. In one study using ultrasound, LH AP diameter shortening during contraction was moderately correlated with maximal contraction force (r=.53-.68) [97], but not in another study (r=.20) that used MRI [37]. So, the size of the hiatus and muscle function are related to one another, but not strongly so. Hiatus size, logically, may not be completely explained by muscle strength because there could be a strong muscle surrounding a large hiatus or a weak muscle surrounding a small one.

Pelvic muscle strength has been found to influence not only the LH, but also the UGH. For example, women with pelvic muscle peak pressure <20 cm H_20 are more likely to have a UGH ≥3.5 cm on physical exam compared to women with pelvic muscle peak pressure ≥20 cm H_2O (36% vs 17%) [98]. However, only a weak correlation was found between UGH size and vaginal closure force during Valsalva or Kegel in a study using MRI [37].

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It is well recognized that pelvic muscle strength can be improved by pelvic muscle exercise [99]. In an evaluation of exercise and hiatus size after six months of intensive pelvic muscle training, LH areas at rest and Valsalva were decreased by 6% and 8% respectively in women with prolapse [100]. Similarly, after four months of pelvic muscle training in women with prolapse, the average LH AP diameter and UGH diameter each decreased by 8% during levator contraction, but no significant change was found at rest [97]. The study also found LH AP diameter shortening during contraction and vaginal squeeze pressure significantly increased, by 36% and 30% respectively, after pelvic muscle training. Interestingly, women performing general exercise during pregnancy had a larger LH area than non-exercisers at 37 weeks gestation (mean difference rest: 1.6±0.7cm, P=.02; Kegel: 1.1±0.5cm, P=.04), but no difference in delivery outcome [101].

Hiatus Size After Forceps and Vacuum Delivery—Both forceps- and vacuum-assisted vaginal delivery are associated with a subsequently larger hiatus size compared to spontaneous vaginal delivery. In a cohort study, women who were delivered with forceps had a 22% larger LH area at Valsalva 3-4 months after delivery compared to at 36-38 weeks of gestation; this was compared with a 9% LH enlargement after spontaneous vaginal delivery and 11% enlargement after vacuum-assisted vaginal delivery [76]. The same study found the LH area during Valsalva decreased 5% after second stage cesarean delivery.

Forceps delivery is associated with a greater risk for major levator injury on MRI (OR: 11.0-25.9) [102] and levator avulsion on ultrasound (OR: 2.9, 95% CI: 1.3-6.7) [103] compared to spontaneous vaginal delivery. Compared to vacuum-assisted vaginal delivery, forceps delivery is still associated with higher risk for levator avulsion (OR: 4.4, 95% CI: 1.42-13.62) [104]. Neither Kearney et al nor Caudwell-Hall et al found significant associations between vacuum delivery and levator injury [102,103]; however, Garcia-Mejido et al found a higher levator avulsion rate for vacuum compared with spontaneous delivery (34% vs 10%) [105], indicating more studies are needed.

Among women who had levator avulsion, forceps-assisted delivery was associated with a 16% larger median LH area during Valsalva, but UGH and LH areas during Kegel and muscle strength remained unchanged [90]. In the same study, forceps use was not significantly associated with hiatal enlargement or poorer muscle strength in the absence of levator avulsion. Rates of midpelvic forceps delivery were strongly correlated with rates of surgery for pelvic organ prolapse 10 years later (r=.72) [106]. In women with levator avulsions, forceps delivery was associated with a 1.7 times higher prolapse rate compared to women without forceps use, but the difference was not statistically significant in women without levator avulsion [107]. Therefore, the association between forceps and hiatus size, muscle strength, and prolapse is likely mediated by the presence of levator avulsion.

Hiatus Size After Cesarean Section—Measuring the hiatuses after cesarean section allows the effects of pregnancy to be evaluated separately from the distention resulting from vaginal birth. At six months postpartum, the LH area was smaller in women delivered by cesarean section with or without laboring than in their third trimester (Fig. 6), with reductions ranging from 5-20% at rest, 6-23% during Valsalva, and 8-21% during Kegel [43,71-73,75]. Patients who underwent cesarean section before labor and in the first stage

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of labor had 2.0 cm^2 and 2.2 cm^2 decreases in LH area at Valsalva from third trimester to four months postpartum, respectively, while women who underwent cesarean section during second stage labor had a smaller decrease (0.9 cm^2) [76]. Moreover, the LH area at rest 4-6 months after cesarean section was not significantly different from that in the first trimester, but the LH area during Valsalva and Kegel was significantly smaller (9% and 6% respectively) than in the first trimester [71]. One to two years after pre-second stage cesarean section, the weighted averages of LH AP diameter and area in all maneuvers are smaller than the weighted averages in the first trimester (Table 1). This implies that hiatus size in women following cesarean section might be able to recover back to pre-pregnancy values. Although levator avulsion is typically seen after vaginal birth, there are occasional reports of avulsion on ultrasound for women who had cesarean section in the second stage of labor [108], especially when labor was prolonged [109]. Among women with a history of only cesarean birth, relative to a GH size of ≤2.5 cm, the association between UGH size and prolapse was significant, with hazard ratios for UGH during Valsalva for GH =3.0 cm and GH ≥3.5 cm being 2.7 (95% CI, 1.2-6.2) and 8.0 (95% CI, 3.7-17.2), respectively [3]. This indicates that UGH enlargement is a risk factor for prolapse regardless of mode of delivery.

OTHER LEVEL III PARAMETERS

It must be remembered that hiatal size and levator injury are only two aspects of Level III pelvic floor status, which can also be affected by factors such as injury to the connective tissues of the perineal complex surrounding the lower birth canal, including the perineal membrane and perineal body [88]. Pelvic floor failure can also involve deepening of the pelvic floor as measured by levator bowl volume, levator subtended volume, and mid-sagittal levator bowl area, all of which are correlated with the UGH and LH (Fig. 7) [33,39,110-113]. By definition, the levator bowl includes the whole 3D volume below the pubovisceral plane and above the levator, and it captures both the change in descent and lateral bulging of the levator ani [114]. Rodrigues et al. introduced levator ani subtended volume, the volume between the pubovisceral plane and the LH plane, as a metric to capture this phenomenon [111,112]. In addition, there are parameters intended for the description of hiatal shape such as the “V-U index” [22], and a new levator plate shape analysis method using principal components that evaluates the levator plate when traced on a mid-sagittal view on MRI; this allows one to identify the levator plate shape difference at full length on both the ventral-dorsal and cephalic-caudal directions [53].

DISCUSSION

The literature contains evolving data on hiatal dimensions in the presence of levator muscle injury, changes in levator muscle strength, delivery mode, and pelvic floor dysfunction. Measurement of hiatal dimensions is one important aspect of Level III status. An enlarged hiatus is associated with pelvic organ prolapse and, to some extent, SUI. Vaginal birth —especially forceps-assisted—is associated with significant enlargement in the hiatus. Enlargement of the hiatus during pregnancy can be interpreted as preparation for the remarkable enlargement that occurs during the second stage of labor. Levator avulsion and reduced pelvic muscle contraction force are associated with an enlarged hiatus but explain less than half of the variation in hiatus size. Therefore, other factors that have not been

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as fully studied such as changes in the perineal membrane and neuromuscular control will need further study to advance our understanding of hiatal closure mechanisms. Factors such as hiatus-associated passive tissue injuries, age, ethnicity, BMI, and genetics might explain the rest of the variance. The two hiatuses—LH and UGH—as anatomic features are key parameters for evaluating current Level III status. Advances in our understanding of the causes of an enlarged hiatus and exactly how it is related to pelvic floor disorders may lead to better prevention strategies, prediction of future pelvic floor dysfunction, and reduction in prolapse recurrence after surgery.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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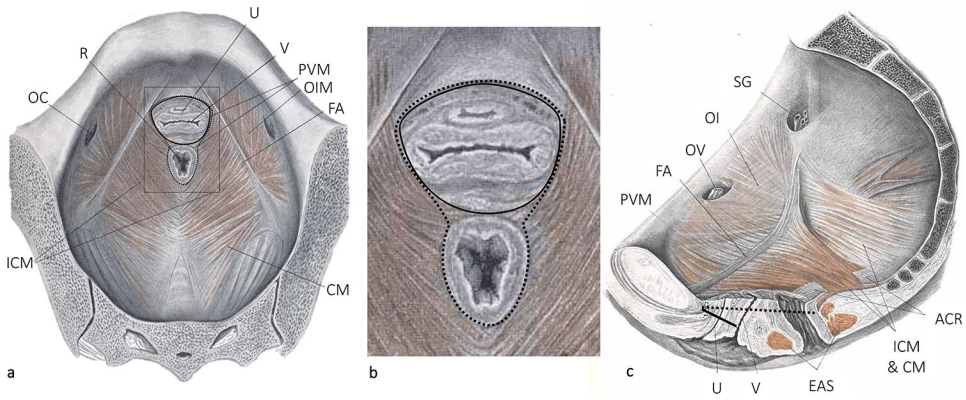

Fig 1. Cadaver dissection of the female pelvic floor

a) View from above after removal of the upper pelvic organs.

b) Closeup view. Shown are the borders of the LH (dotted line) and the UGH (solid line).

c) Left lateral view of the pelvis, showing antero-posterior diameters of LH (dotted line) and UGH (solid line).

LH, levator hiatus; UGH, urogenital hiatus; CM, coccygeus muscle; ICM, iliococcygeal muscle; OC, obturator canal; OIM, obturator internus muscle; OV, obturator vessels; SG, superior gluteal vessels; PVM, pubovisceral muscle; FA, fascial arch (=arcus tendinous fascia pelvis); R, rectum; U, urethra; V, vagina; EAS, external anal sphincter; ACR: anococcygeal raphe.

From Halban & Tandler, 1907 [6]

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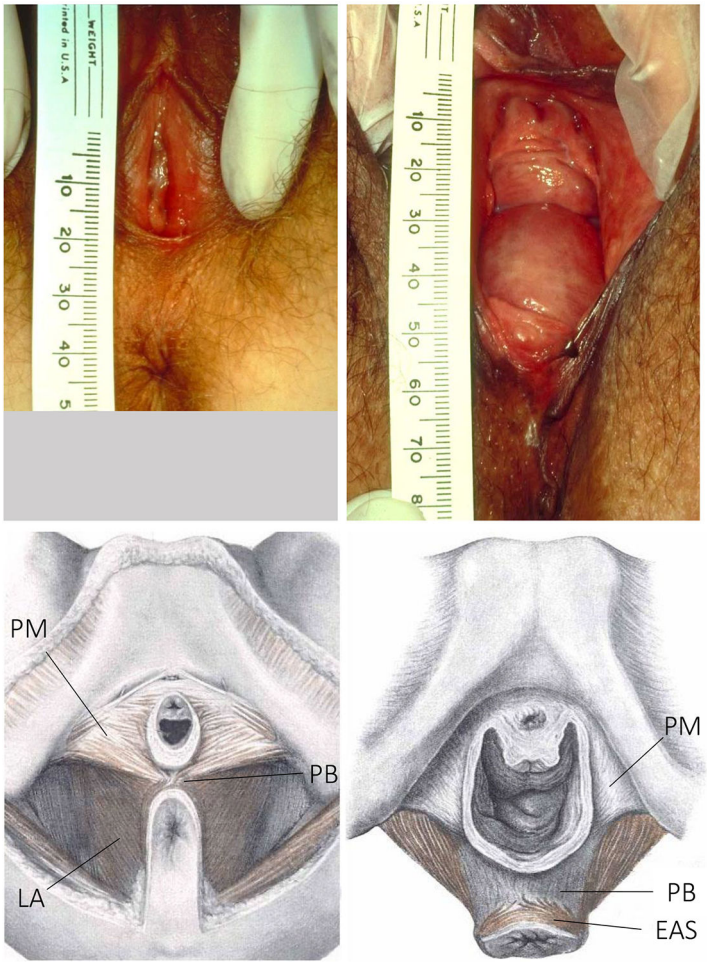

Fig 2. Comparison between normal pelvic support (left) and prolapse with failed hiatal closure (right) in lithotomy view

Top row: UGH as seen in clinical exam.

Bottom row: Lithotomy view of UGH and its relationship to perineal membrane and perineal body in Level III of pelvic support in cadaver dissection. Note close relationship between the perineal membrane and hiatal opening and the significant distortion with prolapse. Lack of information about the role of the perineal membrane in hiatal closure and prolapse is an important knowledge gap.

UGH, urogenital hiatus; EAS, external anal sphincter; LA, levator ani muscle; PB, perineal body

Bottom row from Halban & Tandler, 1907 [6]

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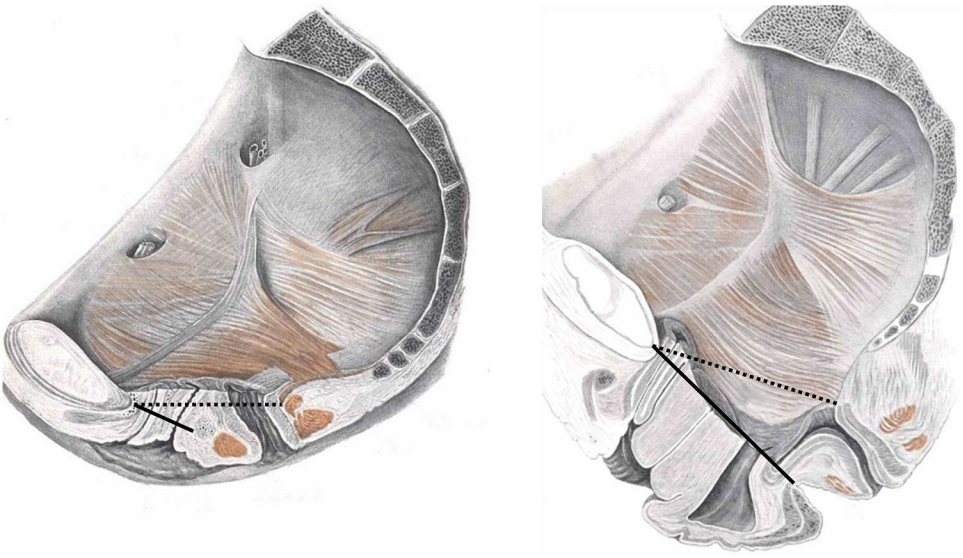

Fig 3. Comparison between normal pelvic support (left) and prolapse with failed hiatal closure (right) in mid-sagittal view

Comparisons shown after removal of upper pelvic organs. Urogenital hiatus shown as solid line and levator hiatus shown as dotted line.

From Halban & Tandler, 1907 [6]

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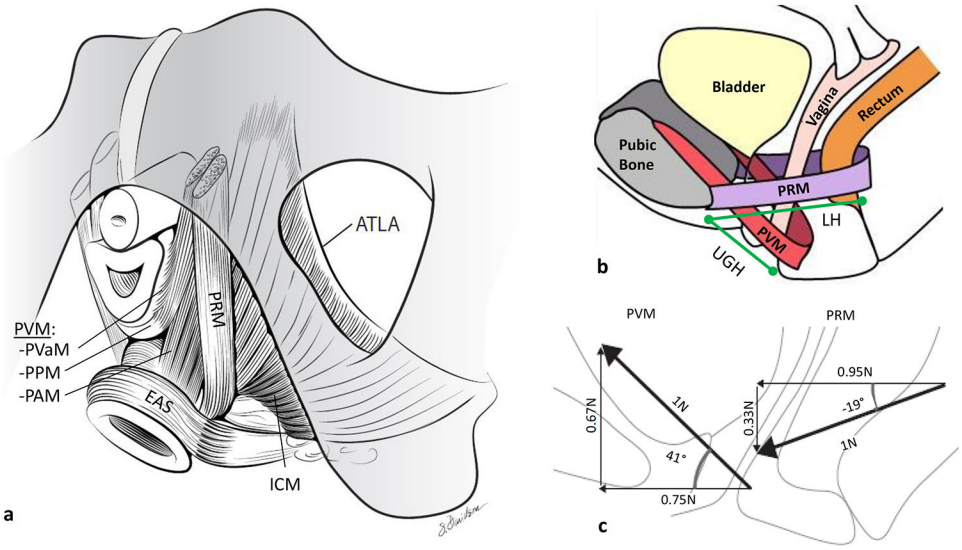

Fig 4. Schematic view of levator ani muscles, UGH, and LH

a) View of levator ani muscles from below after the vulvar structure and perineal membrane have been removed. Pubovisceral (PVM) and puborectal (PRM) muscles forms the sling around UGH and LH, respectively. PVM includes three parts: pubovaginal muscle (PVaM), puboperineal muscle (PPM), and puboanal muscle (PAM).

b) Diagrammatic illustration of muscle loops showing muscle fiber directions for the PVM and PRM relevant to the UGH and LH (green).

c) Horizontal and vertical components of the pubovisceral muscle (PVM) and puborectal muscles (PRM) lines of action in the sagittal plane in a standing posture. The thick arrows show the average direction of the lines of action of the pubovisceral and puborectal muscles relative to the horizontal for a theoretical 1 N force. Thin lines indicate how much of that force acts to “close” and “lift” each hiatus.

Note: Vectors are shown larger than the background anatomy to avoid an overlap in the display.

LH, levator hiatus; UGH, urogenital hiatus

Modified from Kearney, 2004 [10] and Betschart, 2014 [11]. ©DeLancey

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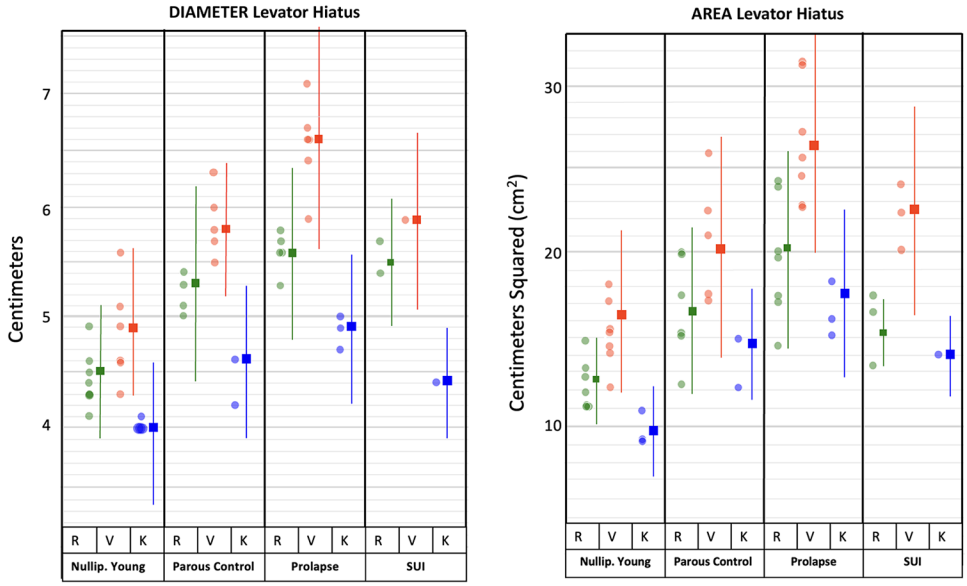

Fig 5. Parity and pelvic floor disorders affecting hiatus size based on values for LH AP diameter and area measured by transperineal ultrasound reported in the literature

Dots represent the means of individual studies listed in Tables S1, S9, S10, and S11. Squares represent weighted means based on the studies shown as dots. Standard deviations bars represent the weighted values.

LH, levator hiatus; AP, anterior-posterior; R, rest; V, Valsalva; K, Kegel; SUI, stress urinary incontinence; Nullip., nullipara.

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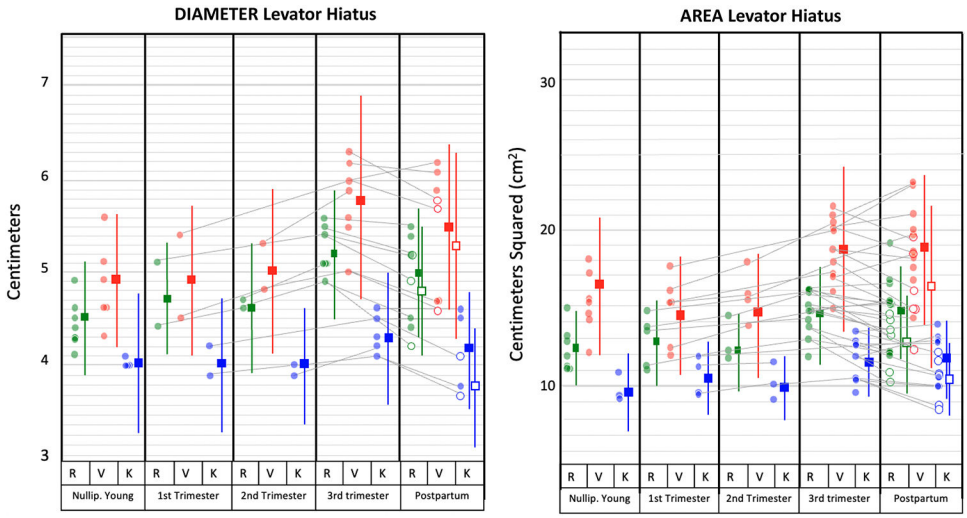

Fig 6. Pregnancy and postpartum hiatal size

Plots of LH size in nullipara along with changes in LH during pregnancy and after birth. Note the generally lower values than in Fig 4. Dots represent the means of individual studies listed in Tables S1-S7. Data that come from a single study are linked by light dotted lines. Squares represent weighted means based on the studies shown as dots. In postpartum columns, filled dots/squares are data for vaginal delivery and open dots/squares are data for cesarean section.

Error bars represent weighted standard deviation.

LH, levator hiatus; R, rest; V, Valsalva; K, Kegel

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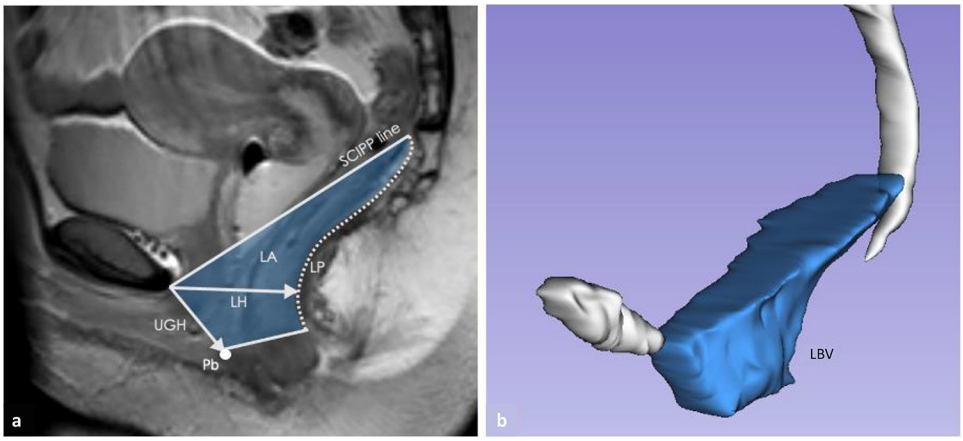

Fig 7. LH, UGH measurements and levator shape

a) Perineal body (Pb), levator plate (LP), levator area (LA), sacrococcygeal inferior-pubic point (SCIPP) line in mid-sagittal view

b) 3D view of levator bowl volume (LBV)

LH, levator hiatus; UGH, urogenital hiatus

Modified from Nandikanti, 2019 [110] and Cheng, 2022 [113]

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Table 1.

Weighted average of LH AP diameters and LH area measured by ultrasound at rest, Valsalva, and Kegel

Measure Type		Nullip Young	Parous Controls	First Trimester		Second Trimester		Third Trimester		Postpartum Vaginal Delivery (3-7 months)		Postpartum Cesarean Section (3-7 months)		Prolapse			SUI		
		Mean±SD	Mean±SD	Mean±SD	%[a]	Mean±SD	%[a]	Mean±SD	%[a]	Mean±SD	%[a]	Mean±SD	%[a]	Mean±SD	%[a]	%[b]	Mean±SD	%[a]	%[b]
LH AP diameter (cm)	R	4.5±0.6	5.3±0.9	4.7±0.6	4%	4.6±0.7	2%	5.2±0.7	16%	5.0±0.7	11%	4.8±0.7	7%	5.6±0.8	24%	6%	5.5±0.6	22%	4%
	V	4.9±0.7	5.8±1.1	4.9±0.8	0%	5.0±0.9	2%	5.8±1.1	18%	5.5±0.9	12%	5.3±1.0	8%	6.6±1.0	35%	14%	5.9±0.8	20%	2%
	K	4.0±0.6	4.6±0.7	4.0±0.7	0%	4.0±0.6	0%	4.3±0.7	8%	4.2±0.6	5%	3.8±0.6	−5%	4.9±0.7	23%	7%	4.4±0.5	10%	−4%
LH area (cm^2)	R	12.3±2.4	16.6±4.9	12.8±2.8	4%	12.2±2.6	−1%	14.5±3.1	18%	14.7±3.1	20%	12.7±3.2	3%	20.2±5.8	64%	22%	15.2±2.0	24%	−8%
	V	16.4±4.9	20.3±6.4	14.6±3.9	−11%	14.7±4.4	−10%	18.7±5.4	14%	18.9±5.0	15%	16.3±5.4	−1%	26.3±6.9	60%	30%	22.5±5.9	37%	11%
	K	9.7±2.5	14.7±3.2	10.5±2.3	8%	9.8±2.1	1%	11.5±2.3	19%	11.7±2.5	21%	10.4±2.3	7%	17.6±4.9	81%	20%	14.0±2.3	44%	−5%

[a] Percentage difference compared to nulliparous young women

[b] Percentage difference relative to parous controls

LH, levator hiatus; AP, anterior-posterior; Nullip, nulliparous; SUI, stress urinary incontinence; R, rest; V, Valsalva; K, Kegel

NOTE: Details are provided in Tables S1-S5, S7, and S9-S11

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