THE JOURNAL OF
Obstetrics and Gynaecology Research

doi:10.1111/jog.12303 J. Obstet. Gynaecol. Res. Vol. 40, No. 4: 1037–1045, April 2014

Do we protect the pelvic floor with non-elective cesarean? A study of 3-D/4-D pelvic floor ultrasound immediately after delivery

Serdar Aydın[1], Muazzez Ayça Tuncel[1], Çağrı Arıoğlu Aydın[2] and Cemal Ark[1]

Obstetrics and Gynaecology, [1]Kanuni Sultan Süleyman Research and Training Hospital, İstanbul, and [2]Karadeniz Ereğli Hospital, Zonguldak, Turkey

Abstract

Aim: To compare levator defect, loss of tenting, change in biometric measurements of the levator ani and genital hiatus according to the mode of delivery, length of the labor, Bishop score, birthweight and head circumference immediately after delivery.

Methods: One hundred and seventy-one primiparous women who delivered either by vaginal delivery or cesarean were prospectively evaluated. Two 3-D volumes (one at rest, one on Valsalva maneuver) were recorded in the supine position after voiding, and levator biometry, levator defect and loss of tenting were determined on the axial plane.

Results: Of 171 nulliparous women, 84 had vaginal delivery and 87 had cesarean delivery. All hiatal dimensions on resting and maximal Valsalva were found to be higher in the vaginal delivery group. Levator defect rate was found to be significantly higher in the vaginal delivery group ($P < 0.0001$). We found a positive correlation with head circumference, fetal weight and first stage labor length in women who delivered vaginally. In the cesarean delivery group, mean fetal head circumference, fetal weight, length of first stage of labor and Bishop score were higher in women with levator ani defect. Loss of tenting rate was significantly higher in vaginal delivery women ($P = 0.03$).

Conclusion: Labor itself, and factors such as fetal head circumference and fetal weight that cause prolongation of labor, can induce levator ani muscle defect or microtrauma which in turn can cause morphological alterations of the levator hiatus.

Key words: cesarean section, levator ani muscle, levator hiatus, pelvic floor anatomy, pelvic floor imaging.

Introduction

Vaginal delivery is frequently cited as an important risk factor for pelvic organ prolapse.[1–3] Birthweights, mode of delivery and length of the second stage of labor have been shown to be additional risk factors.[4,5] Controversies regarding the potential protective effect of cesarean delivery persist. Pregnancy itself causes pathological changes to the pelvic floor.[6–8] Incomplete recovery in anatomy and function may follow macroscopic trauma to the pelvic floor or pregnancy itself can lead to connective tissue remodeling and disruption of the normal pelvic floor function.[9]

Received: May 24 2013.
Accepted: September 11 2013.
Reprint request to: Dr Serdar Aydın, Obstetric and Gynecology Department, Kanuni Sultan Süleyman Research and Training Hospital, Turgut Özal Cad., Küçükçekmece İstanbul, Turkey. Email: serdariks@yahoo.com

The levator ani muscle (LAM) complex, especially the pubovisceral muscle, is the main structure of the pelvic floor which supports the pelvic floor like a hammock. Several forms of such injury to the LAM have been recognized, including stretching resulting in subsequent widening of the pelvic floor organs, neurological injury described in several studies with impairment of LAM strength, anteromedial avulsion and partial or total detachment of the pubovisceral sling from the pubic bone.[10–12]

Recent studies using magnetic resonance imaging and ultrasonography have found LAM trauma in 25–30% of parous women who delivered vaginally.[1,13] LAM avulsion is a risk factor for ballooning and associated with a decrease in pelvic floor muscle strength.[10,14,15] It is also a risk factor for pelvic organ prolapse, especially in anterior and central compartments.[14,16] Alteration in LAM biometry and function after avulsion may also increase the risk of prolapse recurrence after surgical repair.[17–19]

Until recently, magnetic resonance was the only imaging method available for assessing the LAM improvement of the 3-D pelvic floor, however, ultrasound now enables us to evaluate the LAM with minimal discomfort to the patient and at much less cost. The advent of 3-D pelvic floor ultrasound made it possible to access axial planes similar to that employed in magnetic resonance imaging (MRI).

Transperineal ultrasound has been used extensively for pelvic floor assessment with recent advances in imaging technology. By 4-D ultrasound, rendered volume allows observation of axial plane images of the LAM during maneuvers such as Valsalva. Debate has increased in recent years regarding a possible link between LAM trauma during vaginal delivery and subsequent pelvic floor disorders, such as pelvic organ prolapse and urinary stress incontinence.[1,20,21] Cesarean delivery before the beginning of labor may have a potential protective effect against LAM injury.[3,11,12,22] It is therefore important to determine the subgroup of women at risk related to the mode of delivery and potential obstetric factors associated with LAM trauma.

The aim of the study was to compare LAM defect, loss of tenting, biometric measurements of LAM and genital hiatus according to the mode of delivery, length of labor, Bishop score, birthweight and head circumference (HC) immediately after delivery. We also sought to define the critical timing of the cesarean for protection of the pelvic floor.

Methods

This prospective cross-sectional observational study was performed at a tertiary perinatal center with 18 000 deliveries per year. One hundred and seventy-one primiparous women who delivered singleton babies either by vaginal delivery or cesarean were prospectively evaluated within 36 h of delivery. Parameters such as age, episiotomy, mode of delivery, duration of labor, birthweight, HC and antenatal biparietal diameter (BPD) measures were obtained from the clinical files. Length of the first and second stage of labor was obtained from partograph. Obstetric ultrasounds were performed within 48 h before delivery and fetal biometric measures were recorded. All women gave informed written consent before investigation.

Exclusion criteria were operative deliveries (forceps or vacuum assisted), women without regular and active contractions, multifetal pregnancy, previous vaginal or cesarean delivery, handicap in lithotomy position, refused consent and uncooperative patient for effective Valsalva maneuver.

All static ultrasound volume acquisitions were performed by the same experienced sonographer (M. A. T.), trained in pelvic floor ultrasound within 36 h of delivery. Two 3-D volumes (one at rest, one on Valsalva maneuver) were recorded in the supine position after voiding, automatic image acquisition taking approximately 4 s each. A GE Voluson 730 expert ultrasound system (GE Penta Healthcare) with a 4–8-MHz curved array 3-D/4-D ultrasound transducer was used. Imaging was performed with women in lithotomy position with empty bladder. The probe was covered with a sterile film and placed on the perineum in the sagittal plane. The field of view angle was set to a maximum of 70° in the sagittal plane and the volume acquisition angle to 85° in the axial plane. In addition, 3-D render-mode images were made. It was not feasible to not know the delivery mode because of the episiotomy scar after vaginal delivery and dressings as an indicator of cesarean incisions. Analysis of stored volumes was conducted offline by another experienced investigator blinded to clinical and mode of delivery data.

The effectiveness of Valsalva was ascertained by observing 2-D ultrasounds before acquiring volumes. The method of obtaining hiatal dimensions and planes of minimal hiatal dimensions was performed as described by Dietz *et al*.[23] and found to be reproducible by others.[24,25] The plane of minimal hiatal dimensions is identified in the mid-sagittal plane, evident as

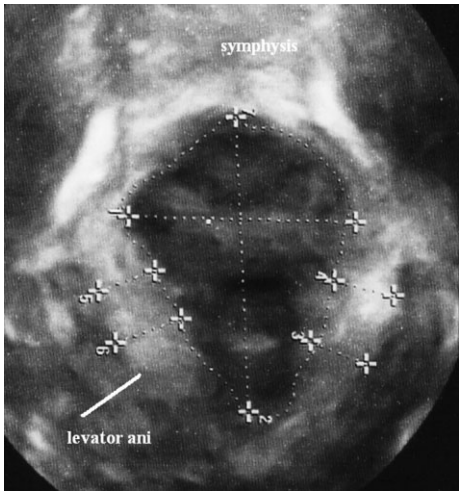

Figure 1 Levator biometric measurement at the plane of minimal hiatal diameter. Markers show: 1, hiatal transverse diameter; 2, hiatal anteroposterior diameter; and 3–6, levator thickness. Dotted line marks levator area.

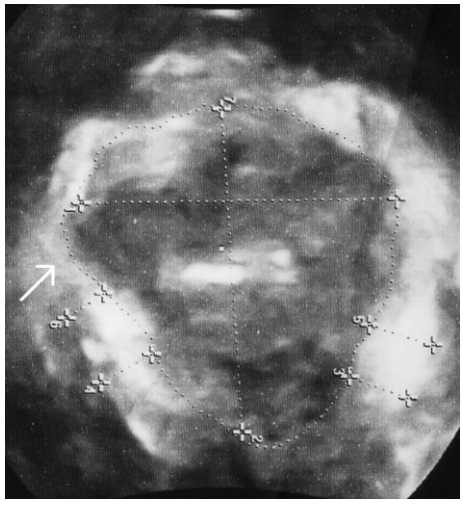

Figure 2 Right-sided levator defect. White arrow shows levator defect. Markers show: 1, hiatal transverse diameter; 2, hiatal anteroposterior diameter; and 3–6, levator thickness. Dotted line marks levator area.

the minimal distance between the hyperechogenic posterior aspect of the symphysis pubis and the hyperechogenic anterior border of the pubovisceral muscle just posterior to the anorectal muscle. LAM thickness is determined at the plane of minimal hiatal dimensions rather than 1–1.5 cm above the actual LAM hiatus where LAM thickness is maximal as described by Dietz *et al*.[23] The following parameters were assessed for this study: maximum diameters of the LAM hiatus (anteroposterior and transverse) at rest and on Valsalva maneuver; area of the LAM hiatus at rest and on Valsalva; and pubovisceral muscle thickness (left and right of the rectum), difference in anteroposterior and transverse diameter of hiatus and LAM hiatus area between rest and Valsalva (Fig. 1).

A LAM defect was diagnosed when there was a discontinuity between the inferior pubic ramus and the puborectalis muscle, which is evident as a V-shaped loop enclosing the urethra, vagina and anorectum, defining the plane of minimal hiatal dimensions as previously described (Fig. 2).[26] Part of the anterior wall and partially lateral wall where it joins the endopelvic fascia forms a 'smile' shape similar to a tent in axial planes. The absence of ventrolateral vaginal sulci described in previous studies in axial planes, either unilateral or bilateral, is accepted as loss of tenting (Fig. 3).[27] A test–retest study on 20 patients was performed by the same investigators for the diagnosis of LAM defect and the results were analyzed with κ statistics (Cohen's κ of 0.72).

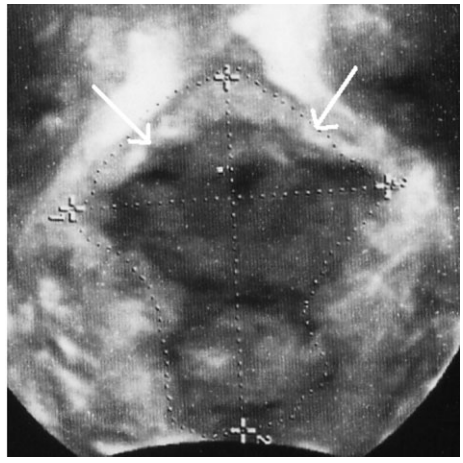

Figure 3 Bilateral loss of tenting. White arrows show loss of tent appearance. Markers show: 1, hiatal transverse diameter; and 2, hiatal anteroposterior diameter. Dotted line marks levator area.

Fetal HC and BPD were measured antenatally 48 h before labor by experienced sonographers. The length of the first stage of labor was calculated from the beginning of regular and at least three strong uterine contractions in 30 min according to the women's statements, tocography and palpations to full dilatation or when cesarean delivery took place. The Bishop score traditionally used for assessment of labor induction was used for estimation of total stress to pelvic floor

and prediction of injury, calculated from charts just before cesarean delivery (Table 1).

Institutional ethics committee approval was obtained for our study (BEAH 2011 6-06). Statistical analysis was performed after normality testing (histogram analysis and/or Kolmogorov–Smirnov) using SPSS version 11.5. Student's *t*-test was performed to compare parametric variables and the Mann–Whitney *U*-test was used for non-parametric variables. χ^2-Test table statistics were used for the proportion of categorical variables. $P < 0.05$ was considered statistically significant.

Results

Mean maternal age at the time of delivery was 24.1 years in the vaginal delivery groups and 26.3 years in the cesarean delivery group. Mean body mass indexes were similar. All patients were nulliparous and 18 (10.8%) women gave a history of first trimester miscarriage or termination. Of 171 women, 84 had vaginal delivery and 87 had cesarean delivery.

Levator hiatus biometric measurement: LAM thickness, hiatal area, anteroposterior diameter, transverse diameter on resting and maximal Valsalva are shown in Table 2. LAM thickness was higher in the vaginally delivered group. All hiatal dimensions on resting and maximal Valsalva were found to be higher in the vaginal delivery group. Mean hiatal area was 20.3 cm^2 on resting and 22.1 cm^2 on Valsalva in the vaginally delivered group. Area measurements were 15.3 cm^2 and 16.4 cm^2 in the cesarean and vaginal delivery groups, respectively, with measurements significantly narrower in the cesarean delivery group.

Levator defect rate was significantly higher in the vaginal delivery group (60/84, 71.4%) than the cesarean delivery group (35/87, 40.2%) ($P < 0.0001$). Maternal age was similar in both groups. When we analyzed the association with BPD, HC, fetal weight, Bishop score and length of the first stage of labor, we found a positive correlation with HC, fetal weight and the first stage labor length in women who delivered vaginally (Table 3). HC (36.5 ± 2.9 cm) was higher in vaginally delivered women with LA defect than without defect (mean HC 34.6 ± 2.2). Fetal weight was also heavier in women with a defect detected using perineal ultrasound immediately after vaginal delivery (3194 ± 443 g with defect vs 2925 ± 331 g without defect). Mean length of the first stage of labor was 9.2 h in women with a defect and 7 h in women without defect. In the cesarean delivery group, mean HC, fetal weight, length of the first stage of labor and Bishop score were higher in women with LAM defect. Mean Bishop score was 5.9

Table 1 Bishop scoring system used for assessment of total stress to pelvic floor during first stage of labor

Score	Dilatation (cm)	Effacement (%)	Station	Cervical consistency	Cervical position
0	Closed	0–30	–3	Firm	Posterior
1	1–2	40–50	–2	Medium	Mid-position
2	3–4	60–70	–1, 0	Soft	Anterior
3	≥5	≥	3	—	—

Table 2 Levator hiatal biometric measurements of women in vaginal and cesarean delivery groups

	Vaginal delivery (mean ± SD) $n = 84$	Cesarean delivery (mean ± SD) $n = 87$	*P*
Levator thickness†	0.99 ± 0.18	0.94 ± 0.17	**0.04**
Hiatal area (resting)‡	20.3 ± 4.3	15.3 ± 3.4	**<0.0001**
Hiatal area (Valsalva)‡	22.1 ± 4.9	16.4 ± 3.7	**<0.0001**
Hiatal anteroposterior diameter (resting)†	6.1 ± 0.9	5.5 ± 0.7	**<0.0001**
Hiatal anteroposterior diameter (Valsalva)†	6.1 ± 0.9	5.4 ± 0.7	**<0.0001**
Hiatal transverse diameter (resting)†	4.6 ± 0.5	4.1 ± 0.5	**<0.0001**
Hiatal transverse diameter (Valsalva)†	5 ± 0.6	4.4 ± 0.6	**<0.0001**
Δ Hiatal area‡	1.8	1.1	**0.04**
Δ Hiatal anteroposterior diameter†	0.01	–0.14	0.06
Δ Hiatal transverse diameter†	3.33	3.31	0.8

†cm. ‡cm^2. Bolding indicates statistical significance. SD, standard deviation.

Table 3 Fetal measurements and labor factors that affect the levator ani muscle defect injury

	Vaginal delivery			Cesarean delivery		
	No LAM defect (mean ± SD)	LAM defect (mean ± SD)	*P*	No LAM defect (mean ± SD)	LAM defect (mean ± SD)	*P*
BPD (mm)	88.3 ± 3.3	88.9 ± 4	0.5	88 ± 9.1	90.8 ± 4.2	0.09
HC (cm)	34.6 ± 2.2	36.5 ± 2.9	**0.02**	35.4 ± 3	37 ± 2.8	**0.02**
Fetal weight (g)	2925 ± 331	3194 ± 443	**0.003**	3019 ± 520	3403 ± 415	**<0.0001**
Maternal age	24.5 ± 5	24 ± 4.1	0.6	26.3 ± 5.3	26.3 ± 4.7	0.9
1st stage of labor length (h)	7	9.2	**0.001**	4.6	9	**<0.0001**
Bishop score				3.3	5.9	**<0.0001**

Bolding indicates statistical significance. BPD, biparietal diameter; HC, head circumference; LAM, levator ani muscle; SD, standard deviation.

Table 4 Fetal measurements and labor factors that affects loss of tenting

	Vaginal delivery			Cesarean delivery		
	No loss of tenting (mean ± SD)	Loss of tenting (mean ± SD)	*P*	No loss of tenting (mean ± SD)	Loss of tenting (mean ± SD)	*P*
BPD (mm)	89.5 ± 3	88.4 ± 4.1	0.2	87.7 ± 10	90.4 ± 4.2	0.1
HC (cm)	35.2 ± 2.6	36.3 ± 2.2	0.1	35.1 ± 3.5	36.7 ± 2.3	**0.03**
Fetal weight (g)	3025 ± 331	3158 ± 464	0.2	3047 ± 578	3286 ± 578	**0.03**
Maternal age	24.1 ± 4.9	24.2 ± 4.1	0.9	26	26.5	0.6
1st stage of labor length (h)	7.7	9.0	**0.04**	4.7	7.8	**0.006**
Bishop score				3.2	5.8	**0.002**

Bolding indicates statistical significance. BPD, biparietal diameter; HC, head circumference; LAM, levator ani muscle; SD, standard deviation.

Table 5 Effect of levator defect on levator hiatus biometric measurements

	Vaginal delivery			Cesarean delivery		
	No LAM defect (mean ± SD)	LAM defect (mean ± SD)	*P*	No LAM defect (mean ± SD)	LAM defect (mean ± SD)	*P*
Levator thickness†	1 ± 0.2	1 ± 0.2	0.8	1 ± 0.2	0.9 ± 0.2	0.1
Hiatal area (resting)‡	18.3 ± 4.4	21.1 ± 4.1	**0.007**	15.4 ± 3.5	15.2 ± 3.2	0.7
Hiatal area (Valsalva)‡	18 ± 3.9	23.8 ± 4.3	**<0.0001**	16.3 ± 3.7	16.7 ± 3.9	0.6
Hiatal anteroposterior diameter (resting)†	5.9 ± 0.9	6.2 ± 0.9	0.2	5.6 ± 0.7	5.5 ± 0.9	0.7
Hiatal anteroposterior diameter (Valsalva)†	5.7 ± 0.9	6.3 ± 0.9	**0.009**	5.4 ± 0.8	5.4 ± 0.7	0.9
Hiatal transverse diameter (resting)†	4.5 ± 0.5	4.7 ± 0.6	0.2	4.1 ± 0.5	4.1 ± 0.6	0.7
Hiatal transverse diameter (Valsalva)†	5.1 ± 0.9	4.6 ± 0.9	**0.003**	4.3 ± 0.6	4.5 ± 0.7	0.1
Δ Hiatal area‡	–0.3	2.7	**<0.0001**	0.8	1.4	**0.04**
Δ Hiatal anteroposterior diameter†	–0.2	0.1	**0.01**	–0.2	–0.1	0.4
Δ Hiatal transverse diameter†	0.1	0.4	**0.007**	0.3	0.4	**0.04**

†cm, ‡cm^2. Bolding indicates statistical significance. BPD, biparietal diameter; HC, head circumference; LAM, levator ani muscle; SD, standard deviation.

in women with LA defect who delivered abdominally and 3.3 without defect at the time of delivery.

The loss of tenting rate was 58 of 84 (69%) women in the vaginal delivery group and 46 of 87 (52.9%) in the cesarean delivery group, namely, it was significantly higher in the vaginal delivery women ($P = 0.03$). In the vaginal delivery group, loss of tenting was more frequent when the first stage of labor length was longer (Table 4). In the cesarean group, higher mean HC, fetal weight, length of the first stage of labor and Bishop score was associated with higher loss of tenting rate.

The effect of LAM defect on the LAM biometric measurements immediately after delivery is shown in Table 5. In the vaginal delivery group, all hiatal dimensions and areas were correlated with LAM defect. Resting hiatal area, Valsalva hiatal area and change in the area between Valsalva and resting were larger among women with LAM defect. Greater dimensions, resting and Valsalva hiatal anteroposterior diameters, resting transverse diameter and change in transverse and anteroposterior diameters were strongly associated with LAM defect. Furthermore, Δ hiatal area and Δ

hiatal transverse diameter was greater in women in the cesarean delivery group with LAM defect.

Discussion

We encountered unexpected results when we analyzed the results of the research. LAM defect rate was extremely high in both groups which is incompatible with previous studies.

In the non-elective cesarean section group, LAM defect rate was nearly twice that of the vaginal birth group. We also found LAM defects following non-elective cesarean which indicates a discrepancy with previous studies. We examined elective an cesarean group in a pilot study before beginning our study and we did not find any defect in the LAM. Although we configured our study to investigate the effect of labor itself, uterine contractions and plane of fetal head on pelvic floor, the evidence of LAM tear after cesarean delivery was unexpected in terms of published data until Albrich *et al*. showed LAM defect 48 h after emergency cesarean.[28] Furthermore, it is difficult to explain how realization of LAM avulsion before crowning of the fetal head causes the distension of the puborectalis muscle. Although it is important to discuss our results, we must also consider our limitations and the timing of the evaluation.

Levator ani muscle avulsion prevalence was reported at a rate of 18.8% by Valsky *et al*. and 39.5% by Albrich *et al*., within 48 h after delivery and using the 3-D perineal ultrasound assessment.[28,29] We found that up to 71.4% of LAM defects arise within 36 h of delivery. This spectacular result could be explained by operator fault or false-positive results. Minor anomalies in the puborectalis muscle described by 3-D perineal ultrasonography could explain our incompatible results. The 'levator defect' and 'avulsion defect' are not used synonymously in this study, avulsion being a complete detachment of the muscle from bone. Minor forms of such defects and any discontinuation or hypoechogenic lesions seen in axial sections have been noted as a defect in our study. Recently, Dietz *et al*. clarified the definitions of complete and partial trauma of the LAM: if all three central slices, namely, the slice at the plane of the minimal hiatal dimensions plus the two above, were abnormal they called it a complete avulsion, with partial avulsion diagnosed as when any three to eight slices were abnormal.[30] We have included all abnormalities in our results, whether complete or partial. These defects may be caused by hematoma, partial muscle tear and edema.

Such minor defects just after delivery may help to explain pathophysiological mechanisms like recovery failure, neurological trauma, loss of muscle strength and uncoordinated function. So, our study supports the studies that consider avulsion and vaginal birth not to be the only reason for pelvic organ prolapses. All labors were actively managed in our clinic. It is important to standardize vaginal birth; thus, all women in the vaginal delivery group and most of women in the cesarean group had oxytocin either for induction or augmentation and mediolateral episiotomy applied to all primiparous women.

Comparison of our LAM defect rates with avulsion rates following first vaginal delivery reported by previous studies of between 19% and 39% is not appropriate due to different assessment times.[28,29] The LAM defect rate following first vaginal delivery and emergency cesarean section defined by Albrich *et al*. was 39.5% within 48–72 h after delivery which is approximately half our results, however, timing of our assessment is within 24–36 h after delivery.[28] This difference could be explained by regression of edema, intramuscular bleeding, fluid collection and straining not being completed in 24–36 h and active management of labor. In addition, there is no information about the first or second stages of labor, HC and other risk factors in the study by Albrich *et al*.[28] As a continuation of this study the measurements were carried out after 3 months of delivery, at which point we found a lower LAM defect rate. Therefore, the definition of 'defect' does not fully meet that of 'avulsion' as elucidated by Dietz's group.[23]

In the present study, one of the inclusion criteria was that all patients were primiparous with active and regular contractions. The use of the first stage of labor, Bishop score (which is traditionally used for predicting the success of labor induction) and fetal measurements such as HC, BPD and fetal birthweight are positive points of the study. We believe use of the Bishop score to calculate total stress on the pelvic floor up to delivery is innovative and controversial. There is no data about the association between the length of the first stage of labor and the Bishop score with pelvic floor ultrasound findings just after or after delivery (either vaginal or cesarean).

Lanzarone *et al*. reported that LAM hiatal dimensions are associated with the length of the second stage of labor.[31] In contrast, Falkert *et al*. found no association with the duration of the second stage of labor, but the weight and HC of the baby showed a positive correlation with the area of LAM hiatus.[32] There is no data in the published work matching LAM defect and the

length of the first stage of labor. Although there is no objective criterion of measurement for the length of the first stage of labor, we calculated it according to the patient's declaration, and files and partographs. The length of the first stage of labor could be associated with LAM defect rather than LAM avulsion which is due to crowning of the fetal head. It must be noted that all patients in the cesarean group had regular contractions demonstrated with tocograph and most of the indications for cesarean deliveries were cephalopelvic disproportion, fetal distress and labor dystocia. The distribution of indications, most of them due to problems with or resistance to the passage of the baby, may help to explain defects in the cesarean group.

Chin *et al*. reported that patients who had undergone cesarean during active labor had a greater risk of future urinary incontinence as comparable with patients who had undergone elective cesarean prior to the onset of labor.[33] Novellas *et al*. published a study demonstrating LAM muscle anomalies with MRI just after cesarean delivery of the primiparous women, and reported that hypersignaling of the muscle, thinning or thickening, or rupture of the muscular insertion in the pubococcygeus–puborectalis muscle of the women were reported anomalies after cesarean section with active labor.[34] They demonstrated that women who had undergone active labor during cesarean had 2.7-times more abnormalities than the patients with cesareans without labor. The average cervical dilatation was 6.2 cm and the average duration of labor was 5.65 h in the active labor group. Lesions involving denervated or ischemic muscle can also present hypersignaling and it is reasonable to assume that during active labor there is the possibility of nerve lesions or transient ischemia leading to muscle damage.[35–38] This evidence confirms that the act of labor itself has a deleterious effect on the pelvic floor that is independent of any mechanical deformation caused by crowning of the fetal head. In addition, Branham *et al*. had already published data on LAM lesions at 6 weeks post-partum.[39]

The timing of the assessment of our study differs from others that mostly evaluate at least 6 weeks post-partum. The timing was convenient because the women were still in hospital where 50–60 live births per day take place and the turnaround of patients is very fast. Early assessment of the pelvic floor enables accessibility of paravaginal connective tissue immediately after stress or trauma caused by labor and pressure of the fetal head before remodeling or recovery processes have occurred within approximately 6 weeks after delivery. Positioning and mobilization of the patient is easier, and congestion and edema of connective tissue of the pelvic floor reduces within 24–48 h of trauma. Investigation of microtrauma and minor hemorrhage was planned before remodeling. We will report the data that – as a continuation of our study – we evaluated in the same patient group after 3 months to compare findings to see whether or not they were compatible with early puerperium.

Loss of tenting described as an absence of ventrolateral vaginal sulci in axial planes is used for evaluation of paravaginal support structures. Evaluation of tenting and associated paravaginal fascial support may be very early, but it could hold clues for follow-up clinical examination and perineal ultrasound 3 months after delivery. There is no study that evaluates tenting or loss of tenting immediately after delivery. Dietz *et al*. reported absence of tenting at rest in 32 of 57 (57%) patients, but this was not correlated with other clinical findings, and 21 of 57 (37%) patients on Valsalva were weakly associated with clinically observed lateral defects.[40] Our result with regard to loss of tenting was 69% in the vaginal delivery group and 52.9% in the non-elective cesarean group at rest, which is compatible with Dietz *et al*.'s findings.[40]

The effect of LAM avulsion on hiatal dimensions and function was clearly demonstrated by Abdool *et al*.[15] They reported that the relative risk of abnormal distensibility was 3.5 in unilateral and 3.96 in bilateral avulsion. Our LAM defects were associated with Δ hiatal area and Δ hiatal transverse diameter in both cesarean and vaginal deliveries. Although these findings may have clinical significance with regard to the described LAM defects, association with pelvic organ prolapse and LAM hiatal distensibility must be supported by our planned study that will evaluate results 3 months after delivery and another well-designed prospective study.

However, we can say that vaginal delivery causes enlargement of pelvic hiatus and LAM defect, and that cesarean delivery is associated with similar defects. According to our results, labor itself, and factors such as HC and fetal weight that cause prolongation of labor, induce LAM defect or microtrauma which in turn can cause morphological alterations of the LAM hiatus. Regular uterine contractions and total stress to the pelvic floor may play a role in LAM and paravaginal deformation. Although there are various limitations to our study, considerable factors relating to the fetus and the mother causing prolongation of labor may predict LAM defect and the following pelvic floor dysfunction. Our controversial data needs confirmation or

contradiction by well-designed prospective studies for the identification of the subgroup of women at risk related to potential obstetric factors and mode of delivery associated with LAM trauma, and the critical timing of cesarean for the protection of the pelvic floor.

Acknowledgments

The authors would like to thank all study participants.

Disclosure

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

References

1. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. *Obstet Gynecol* 2005; **106**: 707–712.
2. Kearney R, Miller JM, Ashton-Miller JA, De-Lancey JO. Obstetric factors associated with levator ani muscle injury after vaginal birth. *Obstet Gynecol* 2006; **107**: 144–149.
3. Dietz HP. Pelvic floor trauma following vaginal delivery. *Curr Opin Obstet Gynecol* 2006; **18**: 528–537.
4. Rortveit G, Brown JS, Thom DH, Van Den Eeden SK, Creasman JM, Subak LL. Symptomatic pelvic organ prolapse: Prevalence and risk factors in a population-based, racially diverse cohort. *Obstet Gynecol* 2007; **109**: 1396–1403.
5. Dannecker C, Anthuber C. The effects of childbirth on the pelvic-floor. *J PerinatMed* 2000; **28**: 175–184.
6. Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med* 2003; **348**: 900–907.
7. Viktrup L, Rortveit G, Lose G. Risk of stress urinary incontinence twelve years after the first pregnancy and delivery. *Obstet Gynecol* 2006; **108**: 248–254.
8. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG* 2000; **107**: 1460–1470.
9. Wijma J, Weis Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract during pregnancy. *BJOG* 2001; **108**: 726–732.
10. Dietz HP, Shek C, De Leon J, Steensma AB. Ballooning of the levator hiatus. *Ultrasound Obstet Gynecol* 2008; **31**: 676–680.
11. Snooks SJ, Swash M, Henry MM, Setchell M. Risk factors in childbirth causing damage to the pelvic floor innervation. *Int J Colorectal Dis* 1986; **1**: 20–24.
12. Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: A neurophysiological study. *Br J Obstet Gynaecol* 1990; **97**: 770–779.
13. 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**: 46–53.
14. 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**: 295–302.
15. Abdool Z, Shek KL, Dietz HP. The effect of levator avulsion on hiatal dimensions and function. *Am J Obstet Gynecol* 2009; **201**: 89.e1–89.e5.
16. Dietz HP, Simpson JM. Does delayed childbearing increase the risk of levator injury in labor? *Aust N Z J Obstet Gynaecol* 2007; **47**: 491–495.
17. Dietz HP, Chanatasorn V, Shek KL. Avulsion of the puborectalis muscle is a risk factor for recurrence after anterior repair. *Int Urogynecol J* 2009; **20**: S172–S173.
18. Adekanmi OA, Freeman R, Puckett M, Jackson S. Cystoceles: Does anterior repair fail because we fail to correct the fascial defects? A clinical and radiological study. *Int Urogynecol J* 2005; **16**: S73.
19. Model A, Shek KL, Dietz HP. Do levator defects increase the risk of prolapse recurrence after pelvic floor surgery? *Neurourol Urodyn* 2009; **28**: 888–889.
20. Gainey HL. Post-partum observation of pelvic tissue damage. *Am J Obstet Gynecol* 1943; **46**: 457–466.
21. Bedwell P, Dietz H. Does levator trauma increase the risk of urodynamic stress incontinence? *Int Urogynecol J* 2008; **19**: S15–S16.
22. Weidner AC, Jamison MG, Branham V, South MM, Borawski KM, Romero AA. Neuropathic injury to the levator ani occurs in 1 in 4 primiparous women. *Am J Obstet Gynecol* 2006; **195**: 1851–1856.
23. Dietz HP, Shek C, Clarke B. Biometry of the pubovisceral muscle and levator hiatus by three-dimensional pelvic floor ultrasound. *Ultrasound Obstet Gynecol* 2005; **25**: 580–585.
24. Yang J, Yang S, Huang W. Biometry of the pubovisceral muscle and levator hiatus in nulliparous Chinese women. *Ultraosund Obstet Gynecol* 2006; **28**: 710–716.
25. Hoff Braekken I, Majida M, Ellstrom Engh M, Dietz HP, Umek W, Test–retest BK. and intra-observer repeatability of two, three and four dimensional perineal ultrasound of pelvic floor muscle anatomy and function. *Int Urogynecol J* 2007; **19**: 227–235.
26. Dietz HP, Steensma AB. The prevalence of major abnormalities of the levator ani in urogynaecological patients. *BJOG* 2006; **113**: 225–230.
27. Dietz HP, Steensma AB, Hastings R. Three-dimensional ultrasound imaging of the pelvic floor: The effect of parturition on paravaginal support structures. *Ultrasound Obstet Gynecol* 2003; **21**: 589–595.
28. Albrich SB, Laterza RM, Skala C, Salvatore S, Koelbl H, Naumann G. Impact of mode of delivery on levator morphology: A prospective observational study with three-dimensional ultrasound early in the postpartum period. *BJOG* 2012; **119**: 51–60.
29. Valsky DV, Lipschuetz M, Bord A *et al*. Fetal head circumference and length of second stage of labour are risk factors for levator ani muscle injury, diagnosed by 3-dimensional transperineal ultrasound in primiparous women. *Am J Obstet Gynecol* 2009; **201**: 91.e1–91.e7.
30. Dietz HP, Bernardo MJ, Kirby A, Shek KL. Minimal criteria for the diagnosis of avulsion of the puborectalis muscle by tomographic ultrasound. *Int Urogynecol J* 2011; **22**: 699–704.

31. Lanzarone V, Dietz HP. Three-dimensional ultrasound imaging of the levator hiatus in late pregnancy and associations with delivery outcomes. *Aust N Z J Obstet Gynaecol* 2007; **47**: 176–180.
32. Falkert A, Endress E, Weigl M, Seelbach-Göbel B. Three-dimensional ultrasound of the pelvic floor 2 days after first delivery: Influence of constitutional and obstetric factors. *Ultrasound Obstet Gynecol* 2010; **35**: 583–588.
33. Chin HY, Chen MC, Liu YH, Wang KH. Postpartum urinary incontinence: A comparison of vaginal delivery, elective, and emergent cesarean section. *Int Urogynecol J Pelvic Floor Dysfunct* 2006; **17**: 631–635.
34. Novellas S, Chassang M, Verger S, Bafghi A, Bongain A, Chevallier P. MR features of the levator ani muscle in the immediate postpartum following caesarean delivery. *Int Urogynecol J* 2010; **21**: 563–568.
35. Tunn R, Delancey JO, Howard D, Ashton-Miller JA, Quint LE. Anatomic variations in the levator ani muscle, endopelvic fascia, and urethra in nulliparas evaluated by magnetic resonance imaging. *Am J Obstet Gynecol* 2003; **188**: 116–121.
36. Tunn R, DeLancey JO, Howard D, Thorp JM, Ashton-Miller JA, Quint LE. MR imaging of levator ani muscle recovery following vaginal delivery. *Int Urogynecol J Pelvic Floor Dysfunct* 1999; **10**: 300–307.
37. Khoury NJ, el-Khoury GY, Kathol MH. MRI diagnosis of diabetic muscle infarction: Report of two cases. *Skeletal Radiol* 1997; **26**: 122–127.
38. Snooks SJ, Setchell M, Swash M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 1984; **2**: 546–550.
39. Branham V, Thomas J, Jaffe T *et al*. Levator ani abnormality 6 weeks after delivery persists at 6 months. *Am J Obstet Gynecol* 2007; **197**: 65.e1–65.e6.
40. Dietz HP, Pang S, Korda A, Benness C. Paravaginal defects: A comparison of clinical examination and 2D/3D ultrasound imaging. *Aust N Z J Obstet Gynaecol* 2005; **45**: 187–190.