Does pregnancy affect pelvic organ mobility?

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Abstract

Background: It is generally accepted that parity is a strong predictor of pelvic organ prolapse and incontinence. However, controversy persists as to whether this effect is due to pregnancy itself or parturition.

Aims: To define the contribution of early and late pregnancy to bladder mobility.

Methods: Case control series, using 88 non-pregnant control subjects recruited for a heritability study on urinary incontinence and prolapse, matched for age and ethnicity with 28 pregnant women seen at 10–17 weeks and again at 32–39 weeks’ gestation. Main outcome parameters were ultrasound measures of anterior, central and posterior compartment descent as well as joint mobility measurements.

Results: Patients in early pregnancy showed higher values for bladder mobility in comparison with non-pregnant controls, although this did not reach significance for all parameters. However, when late pregnancy data were tested against non-pregnant controls, this difference reached significance for all tested parameters. Uterine and rectal descent did not show any significant differences between groups. Results for joint mobility were inconsistent.

Conclusions: This study supports the hypothesis that bladder and urethral mobility increase in pregnancy. This effect is already noticeable at 6–18 weeks. As similar changes are also seen in elbow hyperextension, a generalised effect on connective tissue biomechanics, likely hormonal, can be hypothesised.

Key words: pregnancy, prolapse, ultrasound, urinary incontinence.
were performed, with the most effective used for numerical evaluation. No attempt was made to standardise Valsalva strength since this would have required catheter placement. We used a variety of commercially available ultrasound equipment, including Toshiba EcoCee, GE Kretz Voluson 730, ATL HDI 3000, Hitachi EUB 240, Dornier AI 5200) with 3.5–7 MHz curved array transducers. Since electronic calipers are standardised for reproducibility according to industry-wide standards, measurements are generally regarded as comparable between systems.\(^{10}\) Main outcome parameters were ultrasound measures of anterior, central and posterior compartment descent,\(^8\) as well as upper extremity joint mobility measured according to Brighton.\(^{11}\) The ultrasound measures included the retrovesical angle on Valsalva (RVA-S), urethral rotation on Valsalva, bladder neck mobility or descent (BND) on Valsalva, maximal descent of a cystocele, of the uterus and the rectal ampulla on Valsalva. Figure 1 illustrates the first three of those parameters.

Statistical analysis was performed after normality testing (histogram analysis and/or Kolmogorov–Smirnov testing), using Minitab Version 13 (Minitab Inc., State College, PA, USA). Two sample t-test statistics were used for continuous, normally distributed parameters. A \(P < 0.05\) was considered statistically significant.

All participants gave informed written consent. Ethics Committee approval had been obtained from the local Ethics Committees (SESAHS EC approval 99/184 and QIMR P434 (H0202-01-004)).

### Results

Both groups were matched for ethnicity (all Caucasian) and age (means of 21.1 vs. 21.9 years, n.s.). Continence status was not formally used as a matching parameter; however, the difference between the two groups regarding stress continence occurring at least once a month (8/88 vs. 5/28, \(P = 0.2\)) was non-significant. Body mass index was higher in the pregnant group (22.7 in the non-pregnant group, 25.5 in early pregnancy, \(P = 0.01\)).

### Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Nonpregnant (n = 88)</th>
<th>Early pregnancy (n = 28)</th>
<th>Late pregnancy (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVA-S (degrees)</td>
<td>142 (SD 24.5)</td>
<td>153.6 (SD 21.8)*</td>
<td>158 (SD 23.8)*</td>
</tr>
<tr>
<td>Urethral rotation (degrees)</td>
<td>33.1 (SD 27.1)</td>
<td>43.6 (SD 26.7)</td>
<td>47.5 (SD 24.7)*</td>
</tr>
<tr>
<td>Bladder neck mobility (mm)</td>
<td>17.6 (SD 9.2)</td>
<td>21.3 (SD 10.2)</td>
<td>22.8 (SD 8.6)*</td>
</tr>
<tr>
<td>Cystocele descent (mm)</td>
<td>13.5 (SD 9.5)</td>
<td>9.2 (SD 9.9)*</td>
<td>5.2 (SD 10.6)**</td>
</tr>
<tr>
<td>Uterine descent (mm)</td>
<td>32.7 (SD 13.4)</td>
<td>37.6 (21.3)</td>
<td>33.2 (SD 19.8)</td>
</tr>
<tr>
<td>Rectal descent (mm)</td>
<td>10.5 (SD 16)</td>
<td>8.8 (SD 19.7)</td>
<td>17.6 (StD 20.5)</td>
</tr>
</tbody>
</table>

Urethral rotation and Bladder neck mobility signify differences between measurements at rest and on Valsalva. Cystocele, uterine and rectal descent signify lowest stations reached on Valsalva relative to the symphysis pubis.

\*\(P < 0.05\) vs. non-pregnant, \**\(P < 0.01\) vs. non-pregnant. RVA-S, retrovesical angle on Valsalva.
Patients in early pregnancy showed consistently higher values for anterior vaginal wall mobility in comparison with non-pregnant controls, although this did not reach significance for all parameters (see Table 1). When late pregnancy ultrasound data were tested against non-pregnant controls, this difference reached significance for all measures of anterior vaginal wall descent (see Table 1). Women in the third trimester of pregnancy showed approximately 5 mm more bladder neck descent than age- and race-matched non-pregnant controls. Uterine and rectal descent did not show any significant differences between groups.

The results for joint mobility were inconsistent (see Table 2); fifth finger hyperflexion was more marked in non-pregnant women \( (P = 0.04 \text{ in late pregnancy vs. non-pregnant}) \), but elbow hyperextension showed higher values in pregnancy \( (P = 0.085 \text{ in early}, P = 0.001 \text{ in late pregnancy}) \).

### Conclusion

This case-control study supports the hypothesis that pelvic organ mobility is increased in pregnant women when compared to non-pregnant age- and race-matched controls. This finding is consistent with a recently published case control study using clinical prolapse assessment according to the POP-Q (prolapse quantification) system introduced by the International Continence Society.12 O’Boyle et al. found that all women in the non-pregnant group showed a prolapse grading of 0 or 1 for anterior vaginal wall descent on Valsalva, whereas almost half of the pregnant women were graded as a stage 2 descent. While we did not perform clinical examinations in order to optimise compliance, the ultrasound method used in this study has been validated against Baden–Walker and ICS POP-Q classifications,9 and was recently shown to have good interrater reliability.15

It is acknowledged that a case-control series has obvious methodological weaknesses. Optimally, a study assessing the effect of pregnancy on pelvic organ mobility should recruit non-pregnant women and follow them through gestation. Recruitment of non-pregnant women would also allow investigations into a possible link between luteal and/or placental hormones on the one hand and increased pelvic organ mobility on the other hand. Relaxin, one of the most promising candidates in this regard, did not show any significant correlations with early pregnancy pelvic organ mobility,14 although this study tested absolute values, not increases in mobility from the non-pregnant to the pregnant state.

In this case-control series, there seemed to be a distinct effect of early pregnancy on pelvic organ mobility. In addition, there was a further increase in pelvic organ mobility in late pregnancy. As this change is also seen in elbow hyperextension, a generalised effect on connective tissue biomechanics, possibly hormonal, can be hypothesised, as opposed to an exclusively mechanical effect due to the growing uterus.

The average increase in bladder neck descent attributable to early and late pregnancy might well exceed 5 mm, which could be of clinical relevance, given that the average increase seen after normal vaginal delivery reached 7.2 mm when pre and postnatal measurements were compared.4 Bladder neck descent is clearly not the only determinant of stress urinary incontinence however, and the importance of pregnancy-related changes for future symptoms of pelvic floor dysfunction is controversial. The clinical relevance of the above findings therefore remains to be defined.

Another interesting aspect of a potential hormonal effect on pelvic organ mobility lies in the fact that one might reasonably assume pelvic organ mobility to be an indirect measure of pelvic connective tissue biomechanics. Pelvic organ mobility has been shown to be associated with delivery mode,15,16 supporting this hypothesis. As hormonal effects on pelvic connective tissue biomechanics are assumed to play a role in some mammals,17 it is conceivable that such an alteration of connective tissue biomechanics could also occur in the human female. Such an effect can be measurable in vivo by determining pelvic organ mobility on Valsalva, using pelvic floor ultrasound as a bioassay for hormonally altered biomechanical properties of pelvic floor connective tissue and muscle. There is a need for further studies in this field to correlate altered biomechanics with the hormones of pregnancy.

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### References

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