A 3-D FINITE ELEMENT MODEL OF ANTERIOR VAGINAL WALL SUPPORT FOR EVALUATING MECHANISMS UNDERLYING CYSTOCELE FORMATION

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INTRODUCTION

Anterior vaginal wall prolapse (hernia), clinically known as ‘cystocele’, is the most common form of pelvic organ prolapsed in women (Hendrix 2002). It is also the site with the highest rate of persistent and recurrent support defects (Shull 2000). However the mechanism of cystocele still remains unknown. The goal of this study was to develop a 3-D finite element model based on living woman’s anatomy to investigate how intraabdominal pressure affects prolapse size in the presence of increasing defects in the two systems of anterior vaginal wall support: muscular support and connective tissue support, including apical support and paravaginal support.

METHODS

A 3-D volumetric model was created from magnetic resonance images (MRI) of a 34 year-old healthy nulliparous woman to establish the geometry of anterior vaginal wall and surrounding support tissues using 3D Slicer 2.1b1 (ad modam Chen 2006) is shown in Figure 1A & B. The volumetric model was then simplified using the I-DEAS Surfacer (Figure 1C) and then imported into ABAQUS (Version 6.6-1 ABAQUS, Inc) for meshing and finite element analysis (Figure 1D). The anterior vaginal wall and levator ani muscles were modeled as deformable shell elements. The posterior compartment including posterior vaginal wall and rectum were modeled as a deformable 3-D solid. 3-D truss elements were used to represent the ligamentous connections to vaginal wall. There are total 11553 nodes and 11968 elements. All the model elements were assigned hyperelastic material properties based on literature values (Yamada 1970, Bartscht 1988) and our own tissue testing. Simulations were run by modeling defects as % loss in tissue stiffness and incrementing intra-abdominal pressure.

Figure 1, Model development. A: 3-D volume rendering model of anterior support system including pubic bone; B: 3-D volume rendering model without pubic bone; C: Geometry simplified surface model; D: 3-D finite element model with mesh, boundary condition (orange pin representing ligaments and muscle origin is pinned to pubic bone and pelvic sidewall), and abdominal pressure loading. PB: pubic bone; UT: uterus; V: vagina; R: rectum; CL: cardinal ligament; US: uterosacral ligament; ATFP: arcus tendineus fascia pelvis; ICM: iliococcygeus
muscle; PCM: pubococcygeus muscle; AVW: anterior vaginal wall; PC: posterior compartment; PS: paravaginal support; IAP: abdominal pressure

The model was validated by comparing the mid-sagittal anterior vaginal wall deformation under increasing abdominal pressure with that measured in a dynamic mid-sagittal MRI of a patient with stage III cystocele performing the same maneuver. The simulated prolapse size was measured as vertical distance between the most dependent point of deformed vaginal wall to ATFP origins from pubic bone on mid-sagittal plane.

**RESULTS**

A typical 3-D displacement-time simulation sequence is shown in Figure 2 with increasing intraabdominal pressure, which is run on a IBM T42 with Intel Pentium 1.8GHz processor and 1.25GB RAM for 456mins. The main finding is that prolapse size was sensitive to impairments in apical connective tissue, paravaginal connective tissue, as well as to abdominal pressure (Table 1).

![Image](image)

**Figure 2.** The sequential development of a simulated cystocele at 4 time steps (left-to-right). In this simulation, levator ani muscle had a 60% defect, apical and paravaginal support were set be to 50% defects and abdominal pressure rose from 0 to 99 cmH2O. The first row shows three-quarter views, the second row sagittal cut views, while the plot gives the pressure (y-axis, in cm H2O) at each time point (x-axis).

<table>
<thead>
<tr>
<th>Defect Severity</th>
<th>Abdominal Pressure (cmH2O)</th>
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<tbody>
<tr>
<td>Apical Defect</td>
<td>50</td>
</tr>
<tr>
<td>Para-Vaginal</td>
<td>0.7</td>
</tr>
<tr>
<td>Defect</td>
<td>0%</td>
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<tr>
<td></td>
<td>50%</td>
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**Table 1:** Prolapse size (in cm) of the simulated cystocele with different defect combinations under different maximum intraabdominal pressure (in cmH2O).

**DISCUSSION**

This is one of the first attempts to simulate cystocele formation using 3-D finite element analysis. The model correctly reproduces the development of a cystocele under increasing intraabdominal pressure, as seen clinically. The model lends itself to be used to systematically examine the interactions of different combinations of anatomic defects in causing cystocele formation in a way that would not be ethical in living humans.

**CONCLUSIONS**

A cystocele can form due to impairments in muscle and/or connective tissue supports of the anterior vaginal wall.

**REFERENCES**


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