A BIOMECHANICAL MODEL FOR TISSUE INJURY IN PELVIC ORGAN PROLAPSE

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INTRODUCTION

The pelvic floor is a complex, soft tissue diaphragm that is suspended across the pelvis and supports the abdominal contents against gravity and activities that raise intraabdominal pressure. During childbirth, the pelvic soft tissues in women must distend greatly to accommodate delivery of a large fetal cranium. During birth, the vaginal connective tissues rapidly undergo a 4-5 fold elongation. After childbirth, the tissues must remodel back into a structure that can provide support for 40-60 years. Mechanical failure of pelvic floor is common in older women, and results in conditions such as pelvic organ prolapse and urinary incontinence. Women face an 11% lifetime risk for undergoing surgery for pelvic organ prolapse. (Olsen, 1997)

The anterior vagina forms the most superior layer of pelvic floor support and the most common site of vaginal prolapse. In contrast to ligamentous supports, the vagina is composite of several tissue types, smooth muscle, connective tissue, and epithelium. (Figure 1) Gynecologic surgeons posit that prolapse occurs due to attenuation or discrete breaks in the fibromuscular layer, followed by abnormal elongation of the vaginal epithelium. Surgeons believe that the fibromuscular layer lends strength to the vagina, and surgical repair aims to repair this layer. This assumption cannot be studied in human women due to the deep, inaccessible location of these tissues.

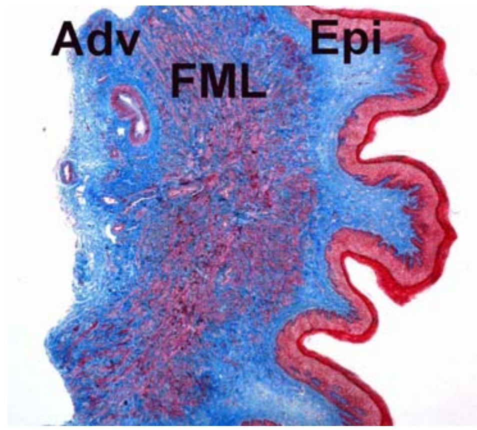

Figure 1. Full thickness histological section of anterior vaginal wall: **Adv,** adventitia; **FML,** fibromuscular layer; **Epi,** epithelium. With trichrome, smooth muscle and epithelium stain red, connective tissue, blue.

We've chosen an animal model in which to study the biomechanics of vaginal support. The Rhesus is similar to humans with respect to its semi-upright posture, its pelvic anatomy, and childbirth. Biomechanical assessment of these tissues represents a novel approach to studying the pelvic floor.

METHODS

Longitudinal strips of full thickness anterior vaginal wall were cut in a standardized "dog bone" shape. Uniaxial tensile failure load was measured on an Instron tensiometer. Deflection was measured by synchronizing optical capture of markers sutured to the specimens. A customized cryo-fixture was used to attach specimen to the tensiometer. The loading rate was 20 mm/min. The tissue dimensions were measured from digital photographs of the gross specimens and by image analysis of histological specimens.

RESULTS AND DISCUSSION

In contrast to tensile testing of ligament or skin, ultimate failure load occurred coincident with a small break in the specimen, while the majority of the tissue is visibly intact. (Figure 2a) Visible (clinical) failure of the tissue didn't occur until the load dropped almost to zero. (Figure 2b)

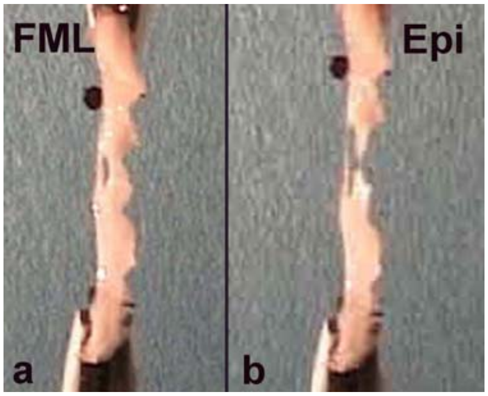

Figure 2. Lateral view of test specimens; **a.** At ultimate failure load, **b.** Just prior to clinical failure

In a majority of specimens, 80%, the 1st break occurred in the fibromuscular layer, separately or coincident with an adjoining layer. (Table 1) The remaining adventitia and epithelium undergo significant plastic elongation before complete loss of continuity of the tissue.

Table 1. Results of testing

First Layer of Break	N=83
FML	56 (67%)
FML+Adv simultaneously	8 (10%)
FML+Epi simultaneously	2 (2%)
All layers simultaneously	2 (2%)
Epi→FML→Adv	7 (8%)
Unable to visualize	8 (10%)

Ultimate elastic elongation was measured as 24% of the original length. Plastic elongation was even greater, 33% of original length before complete loss of continuity.

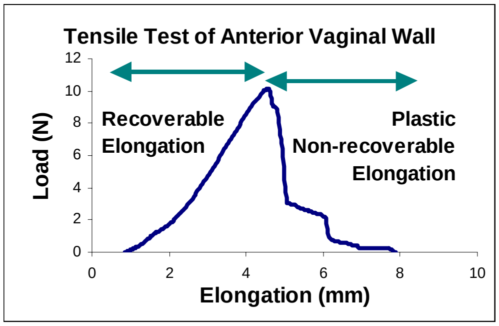

Figure 3. Example of load deflection curve demonstrating elastic and plastic elongation

SUMMARY

Our model closes represents the condition of pelvic organ prolapse, in which abnormal elongation of the vagina is associated with specific defects in the fibromuscular layer. We've shown that the injury required to weaken the vagina can occur without a visible change in the vaginal epithelium. Our work supports the clinical hypothesis that sub-clinical injury to the fibromuscular layer, as may occur during childbirth, markedly reduces the ability of the vagina to resist plastic deformation, thus leading to the development of pelvic organ prolapse.

REFERENCES

Olsen, A. L., V. J. Smith, et al. (1997). "Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence." Obstet Gynecol **89**(4): 501-6.

ACKNOWLEDGEMENTS

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