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ACUTE EFFECTS OF VAGINAL DISTENSION IN THE FEMALE RAT: BIOMECHANICS AND HISTOLOGY

Hypothesis / aims of study

Pelvic floor injury frequently complicates vaginal delivery [1]. Animal models have demonstrated urinary incontinence and histopathology day or weeks after vaginal distension (VD) [2,3]. Acute effects have received less attention. The purpose of this study was to assess the biomechanics and acute structural effects following VD.

Study design, materials and methods

Ten Fr. Foley catheters were used to perform VD in 8 virgin female Sprague-Dawley rats. Procedures and care were in accordance with institutional guidelines. To pre-condition each balloon, the balloon was filled and emptied in a stepwise fashion (0.2ml steps) to 3ml while pressure in the balloon was measured, until a consistent pressure-volume filling relation was achieved.

Anesthetized rats underwent serial vaginal dilation to accommodate the vagina to larger capacities. A pre-conditioned catheter was inserted into the vagina, secured with a single skin suture, and attached to the pressure gauge. The balloon was inflated in 0.2ml increments with water (n=4) or radiographic contrast media (n=4) while balloon pressure was recorded. Anterior-posterior and lateral x-rays were taken at each volume step. Balloons were filled to 3ml and maintained for 1 hour. Two rats underwent sham VD according to the same protocol, but without inflation.

The 4 rats that underwent radiographic study and the 2 control rats were euthanized by intracardiac perfusion with aldehyde fixative with the filled balloon *in situ*. The vagina and urethra were carefully dissected and immersion fixed. The organs were bisected and the left half was immersion-fixed in formalin and processed for paraffin sections. The right half was processed for epon semithin sections. Histological data was analysed qualitatively.

Quantitative results are presented as mean ± standard error. A Friedman test was used to compare e*x vivo* and *in vivo* balloon pressures during VD at each volume to identify significant changes in vaginal biomechanical properties. $P<0.05$ indicated a significant difference.

Results

During *ex vivo* filling, pressure in the balloon increased rapidly to a peak pressure of 638 ± 48 mmHg at 0.6 ± 0.1ml after which it decreased (Figure 1). During *in vivo* filling, pressure in the balloon was significantly greater than *ex vivo* filling at all volumes greater than 0.6ml. *In vivo* balloon pressure increased to a peak pressure of 895 ± 17 mmHg at 1.9 ± 0.1ml, rapidly dropped after peak pressure, and maintained a steady pressure at higher volumes.

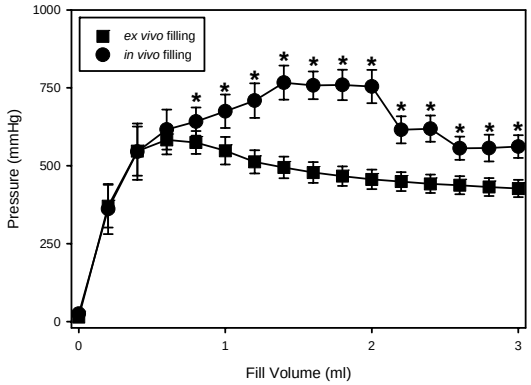

Fig. 1. *Ex vivo* and *in vivo* balloon filling relations.

Intravaginal pressure rose to a peak of 436 ± 27 mmHg at the same volume of peak pressure as in vivo pressures, dropping in a fashion similar to *in vivo* pressure at higher volumes.
Serial radiographs (Figure 2) demonstrated that the shape of the intravaginal balloon started nearly spherical and similar to the *ex vivo* configuration. Intravaginal balloon shape changed to prolate spheroidal at an intermediate filling stage (1.4ml), but returned to a spherical shape in conjunction with the intravaginal pressure decrease.

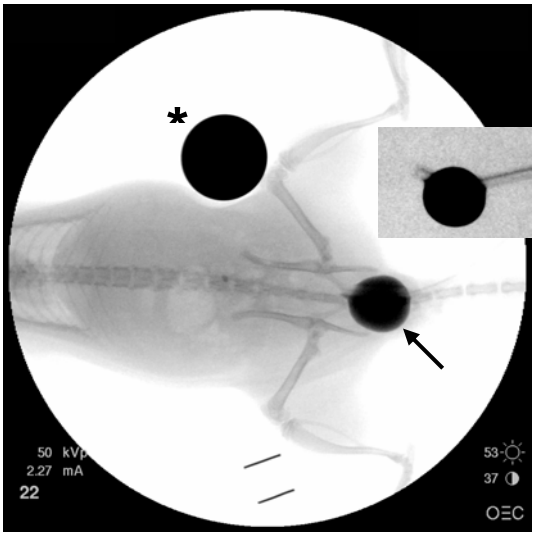

Fig. 2. Example anterior-posterior x-ray of distending balloon in rat vagina at 1.4ml (arrow). Inset is balloon inflated to the same volume *ex vivo*. Large circle, ***,** is a 30mm standardized sphere for length calibrations.

The dorsolateral aspect (near the vagina) of the external urethral sphincter skeletal muscle in the sham-distended animals demonstrated intracellular edema and acute mitochondrial vacuolation. Vaginal and urethral mucosal layers were thick and normal in appearance. In contrast, vaginal and urethral mucosae of the animals distended to high volumes were thin but not torn. The skeletal muscle demonstrated mitochondrial vacuolation but not edema. The extracellular space between the vagina and urethra was expanded. Three distended animals demonstrated significant signs of hemorrhage. In none of the animals were there signs of gross tearing of vaginal or urethral tissues. Compressed vascular structures were consistent with ischemic changes, although the submucosal venous plexus in the urethra had a variable appearance despite the uniformly good perfusion.

Interpretation of results

Multiple interpretations of the drop in intravaginal pressure exist, including hemorrhage or vascular disruption, muscle tears, or relaxation of vaginal muscle contraction, all of which would enable release of vaginal pressure. There was histopathological evidence of hemorrhage in some animals, but no evidence of gross vaginal tears. Future studies will investigate if the drop in pressure is due to muscle relaxation and/or hemorrhage.
The seemingly paradoxical observation of edema in the skeletal muscle of sham-distended animals needs further investigation. Accommodation of the vagina to increasing sizes and insertion of a catheter in the vagina was sufficient to cause detrimental effects in the urethra, which could be due to either trauma or ischemia. Due to constricted space in the pelvic floor at high distension volumes, fluid may leave the intracellular space and go extracellular, reducing edema. This theory is supported by the expanded extracellular space between the vagina and urethra in VD animals.

Concluding message

Our results suggest dramatic alterations occur during VD, which may contribute to the development of urinary incontinence and other pelvic floor disorders.

References

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