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ROLE OF PUBORECTALIS MUSCLE IN THE GENESIS OF URETHRAL PRESSURE

Hypothesis / aims of study

Urethral pressure is a major determinant of urinary continence function and urethral sphincters (internal smooth and external, striated rhabdosphicter - RHB) play an important role in the genesis of urethral pressure. Pelvic floor muscles are recognized to provide a supportive function in the urethral pressure regulation and urinary continence mechanisms. Puborectalis (PRM), a "U" pelvic floor shaped muscle is important in the genesis of anal and vaginal pressures in humans[1, 2]. However, the role of PRM in the genesis of urethral pressure is not clear. The aim of our study is to characterize the urethral pressure in an animal model and specifically understand the contribution of PRM contractions to urethral pressure.

Study design, materials and methods

Female rabbits (n=10) were anesthetized and urethral pressure was measured using a manometric catheter equipped with 3 mm diameter sleeve sensor. Pressures were recorded (mm Hg) at rest, after administration of pharmacological agents and then during electrical stimulation of the PRM and RHB muscles. A pulse generator connected to a constant current unit with currents ranging from 1 to 6 mA, in steps of 1 mA increment (frequency 50 Hz, pulse duration of 0.2 ms) was used for electrical stimulation. Phenylephrine (PE; 5-50 ug/kg), tamsulosin (TAM; 50 μg/kg), sodium nitroprusside (SNP; 20 μg/kg) and pancuronium bromide (PB) (0. 4 mg/kg) were used to define the relative contribution of smooth and striated muscles to the urethral pressure.

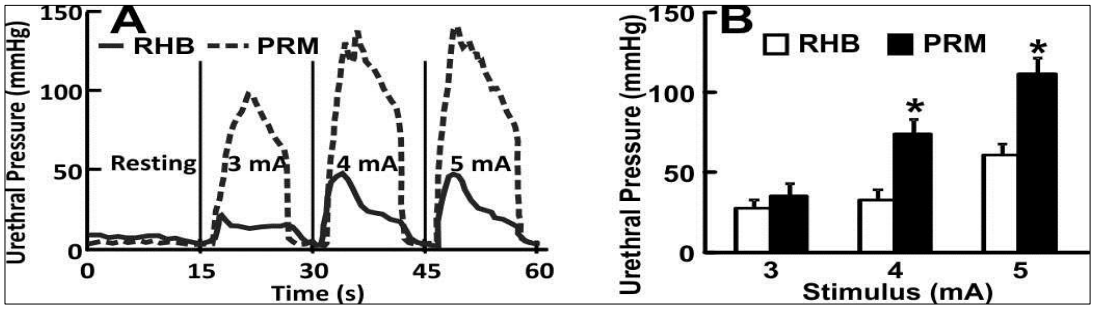
Results

At rest, maximum UP (mmHg) recorded was 12.6±6.2 mmHg. SNP (20 μg/kg) infusion resulted in 30-40 % decrease in the resting UP (7.2± 0.2 mmHg). Administration of PE, an alpha adrenergic agonist produced a dose-dependent increase (17±6, 25±5, 29±6 for 5, 10, 50 μg/kg; i.v.) in UP. TAM (50 μg/kg), a α_{1a}-selective receptor blocking agent, antagonized the PE induced increases in UP (4.1±2.5 after TAM). Electrical stimulation of the PRM muscle resulted in a stimulus dependent increase in the urethral, vaginal and anal canal pressures where as RHB stimulation produced an increase in UP alone. UP increase after PRM stimulation was significantly ($P < 0.05$) higher when compared to the RHB stimulation (Figure 1 A-B). PB administration completely abolished the PRM/ RHB contractions.

Interpretation of results

Our results suggest the involvement of both smooth and striated muscle sphincters in the genesis of resting urethral pressure. Stimulus dependent increase in the PRM active muscle contractions resulting in several fold increase in the UP and its abolition by PB confirms the involvement of pelvic floor skeletal muscle in the UP. Dose dependent increase in the UP produced by PE and its antagonism by TAM suggests the involvement of α_{1a}-adrenergic receptors in mediating these pressure responses.

Figure 1: Effect of RHB/PRM Electrical Stimulation on Rabbit Urethral Pressure



Concluding message

Our data suggest that PRM contractions increase urethral pressure. We propose that pelvic floor contraction plays an important role in the urinary continence mechanism by increasing urethral pressure.

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What were the subjects in the study?	**ANIMAL**
Were guidelines for care and use of laboratory animals followed or ethical committee approval obtained?	**Yes**
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