PUEDNAL NERVE STRETCH REDUCES EXTERNAL URETHRAL SPHINCTER ACTIVITY IN RATS

Hypothesis / aims of study
Vaginal childbirth injury, a major risk factor for stress urinary incontinence (SUI), results in damage to the pelvic floor and the innervation of the external urethral sphincter (EUS) via pudendal nerve stretch (PNS) and crush [1]. Animal models such as vaginal distension (VD), pudendal nerve crush (PNC), and combinations simulate SUI, but animals quickly recover normal urethral resistance to leakage [2]. An integrated model involving physiologic mechanisms of injury, such as pudendal nerve crush and stretch and VD, would more accurately replicate human vaginal childbirth injury. We hypothesize that PNS reduces EUS electromyography (EMG) activity with a resultant decrease in urethral resistance, as measured by leak point pressure (LPP).

Study design, materials and methods
Female virgin Sprague-Dawley rats were anesthetized with urethane. The urethra and bilateral pudendal nerves were exposed through an anterior transpubic approach. EMG of the EUS was performed using parallel needle electrodes during bladder filling via a suprapubic catheter (5ml/h). The pudendal nerve was marked with a surgical marker and bilateral PNS was performed by insinuating either a shodded Debakey forceps or Castroviejo surgical calipers under the nerve and opening the instrument, stretching the nerve by spreading apart the ischiorectal fossa between the pelvic sidewall and vagina. Digital photographs before and during stretch were used to quantify nerve stretch. EUS EMG was repeated immediately following and then at 10, 30, 60, and 120 minutes after bilateral stretch, and were compared to pre-stretch values. Similarly, LPP was assessed at each time point as a measure of urethral outlet resistance. Friedman test followed by post hoc pair-wise comparisons were used to evaluate results at different time points with p<0.05 indicating a statistically significant difference. Data is presented as mean ± standard error of the mean.

Results
Sixteen rats weighing approximately 260g underwent bilateral PNS. Stretch was 54 ± 14% on the left and 47 ± 9% on the right and were not significantly different. There was an immediate and significant decrease in EMG amplitude (p < 0.01) and frequency (p < 0.01) following PNS (Figure 1), and amplitude remained significantly lower 10 minutes after stretch (p < 0.01). LPP also dropped immediately following stretch (median 45cm H₂O to median 39cm H₂O), although this was not significant (p = 0.09; Figure 2).

Interpretation of results
Although there was an immediate and significant reduction in neuromuscular activity of the EUS, the decrease in LPP did not reach statistical significance. The threshold to cause neuromuscular deficit with stretch may therefore be lower than that needed to cause significant functional change, indicating a high sensitivity of the EUS to PNS.

Concluding message
PNS shows promise as a model of pudendal nerve injury during vaginal childbirth, with demonstrable change in EUS EMG immediately following stretch that shows short-term recovery following a short duration of stretch. The changes in EMG, and relationship between these changes and degree of stretch, warrant further study with different degrees and durations of stretch, and longer follow-up to evaluate the time course of recovery.
Figure 1. EUS EMG Amplitude and Frequency Before and After Stretch

Figure 2. LPP Before and After Stretch

References
1. AJOG 2005; 192: 1669.