ABNORMAL EXTERNAL URETHRAL SPHINCTER ACTIVITY IN PUDENDAL NERVE INJURY RATS

Hypothesis / aims of study
Pudendal nerve injury in the rat has been used as an animal model for studying SUI. However, the effect of pudendal nerve injury on external urethral sphincter function has received relatively little attention. The aim of this study was to establish a durable SUI model in female rats by bilateral pudendal nerve injury. In addition to the LPP test, our studies focused on detrusor and external urethral sphincter (EUS) activity after pudendal nerve injury.

Study design, materials and methods
Sixty female Sprague-Dawley rats (300-380 g) were used in the experiments. The rats were divided into two groups: pudendal nerve injury (PNI) and sham-operated control groups. Cystometrography (CMG) and EUS electromyography (EUS EMG) were performed after induction of urethane anesthesia 6 weeks after pudendal nerve injury. The leak point pressure (LPP) test was used to demonstrate the effect of pudendal nerve transection.

Results
All animals exhibited micturition reflexes during CMGs. A significant decrease in LPP was found in PNI animals. PNI rats exhibited voiding abnormalities including increased volume threshold (VT) for initiating voiding, increased contraction duration (CD) and residual urine (RU), decreased voiding efficiency and changes in the pattern of EUS EMG activity. In PNI rats, the EUS EMG consisted of longer periods of bursting activity and a reduction in the ratio of silent to active periods during voiding.

Interpretation of results
Our results indicate that the pudendal nerve is not essentially for reflex micturition because all of the animals voided during continuous bladder infusion. However, PNI rats exhibited voiding abnormalities, which might be attributed to loss of both the sensory and motor branches of the pudendal nerve.

Because the pudendal nerves are thought to provide the principal innervation of the EUS, the activity of the EUS would be expected to completely disappear after bilateral pudendal nerve transection. However, our data showed that there was still some residual EMG activity during bladder filling in the rats 6 weeks after PNI. Although pudendal nerve regeneration could partially account for the unexpected results, it is also possible, as noted by other investigators, that the EUS receives an innervation via the pelvic nerves.

Our results indicate that the average bursting frequency in normal control rats was 6.25 Hz, which is consistent with previously studies (a burst pattern of 4-8 Hz). In PNI rats, a decrease in the silent period during bursting and an increase in bursting frequency (8.025 Hz) shortened uroflow time, which in turn induced urinary retention.

Concluding message
These results indicate that bilateral pudendal nerve injury impaired voiding efficiency in rats by changing EUS activity.

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