**Hypothesis / aims**

Cross-sensitization among pelvic structures may contribute to chronic pelvic pain of unknown etiology and involves convergent neural pathways of noxious stimulus transmission from two or more organs. It has been speculated that convergence of sensory information from discrete pelvic structures may occur at different levels of nervous system hierarchy; locally (peripherally) via axon collaterals in the spinal cord (dorsal root reflexes), and/or in the central nervous system. In the present study, we aimed to directly test the hypothesis that rectal distension (RD) can alter the mechanosensitive properties of urinary bladder afferents at a peripheral afferent nerve level by using established techniques of measurement of primary bladder single unit afferent activities (SAAs) and artificial stable RD.

**Study design, materials and methods**

Female Sprague-Dawley rats were used. Under urethane anesthesia (1.5 g/kg intraperitoneally), a catheter and a balloon-attached catheter were inserted into the bladder and the rectum, respectively. Both L6 dorsal roots were cut and fine filaments were dissected from the left L6 dorsal roots and placed across a bipolar electrode for monitoring SAAs. SAAs of the nerves primarily originating from the bladder were identified by electrical stimulation of the pelvic nerve and by bladder distension. Nerves with conduction velocities (CV) more than 2.5 m/sec were designated as Aδ-fibers and those with CV less than 2.5 m/sec as C-fibers. RD was applied by filling the balloon with saline.

First, to exclude the direct mechanical effect of RD on bladder pressure, intravesical pressure was assessed under empty and saline-filled (at 30 cmH2O of intravesical pressure) states of the bladder with application of RD.

To evaluate the effect of continuous RD on bladder SAAs during bladder filling, first, SAAs were recorded during cystometry with filling saline into the bladder at a rate of 0.08 ml/min until an intravesical pressure of 30 cmH2O without RD as the baseline, and then the SAA measurement repeated 3 times under different RD conditions at the rectal balloon pressure of 20, 40, and 60 mmHg.

SAAs were averaged at 5 cmH2O interval of pressure or at equally divide into five parts of volume in the filling phase, and the average unitary activity was totalled as an integrated activity during the whole filling phase, based on pressure and volume, respectively. For comparison of these integrated activities, the values are expressed as a percentage of the base-line activity.

**Results**

Nineteen rats were used in this study. No significant direct mechanical influence of RD on the bladder pressure either empty or full bladder state was confirmed in 4 rats (data not shown). Thirty single unit SAAs (Aδ-fibers: n=11, CV=5.20 ± 0.71 m/sec, C-fibers: n=19, CV=1.28 ± 0.12 m/sec.) were isolated from 15 rats. RD facilitated bladder SAAs of both Aδ- and C-fibers during bladder filling in a strength-dependent manner.

**Conclusion**

The present study demonstrates that rectal distension can increase bladder mechanosensitive afferent activities of both Aδ- and C-fibers as a consequence of peripheral neural cross-talk.