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SPINAL INHIBITORY CONTROL OF PELVIC EFFERENT ACTIVITY TO THE BLADDER – AN EXPERIMENTAL STUDY IN THE CAT

Aims of Study

Two spinal inhibitory systems are known to influence pelvic efferent activity to the bladder: one involves afferents from the ano-genital region, the other recurrent collaterals of bladder preganglionic neurones (1 - 4). Both the afferent and recurrent inhibitory systems are quite powerful and normally sufficient to abolish an ongoing micturition reflex. In the present study, the electrophysiological properties of these inhibitory systems were compared. It was thought that such a characterisation might help to specify the functional role of these inhibitory mechanisms.

Methods

Adult female cats were anaesthetized with α-chloralose (55 mg/kg). A catheter was inserted into the bladder through a slit in the proximal urethra to allow bladder filling or draining and intravesical pressure recordings. After a laminectomy of the L6-L7 vertebrae, the dorsal roots S1-S4 were transected on the right side. Bladder pelvic nerves were exposed bilaterally while left in continuity. Bladder test reflexes were evoked on the left side, where the nerve branches were mounted for stimulation near the bladder, and the evoked ipsilateral efferent activity recorded from the central end of a small pelvic nerve branch transected close to the bladder. To focus the investigation on spinal inhibitory mechanisms, the spinal C fiber bladder reflex was used as a test. C-fibre reflexes were evoked by low frequency stimulation with trains of three shocks (0.5 ms, 10 ms intervals) at appropriate intensity. The right pelvic nerve was mounted for stimulation and used to elicit recurrent inhibition through antidromic activation of bladder pre-ganglionic fibres. Dorsal clitoris nerves on each side were mounted for stimulation. In most trials, the bladder was left empty and open to avoid interference due to activation of bladder Aδ mechanoreceptor afferents distally to the stimulation site. Naloxone (0.01-0.5 mg/kg, i.v.) was used to further characterize transmission in the inhibitory pathways.

Results

Stimulation of small to medium sized myelinated afferents in the dorsal clitoris nerve elicited a pronounced inhibition of the bladder C-fibre reflex. Brief trains (< 200 ms, 10 Hz, 0.1-0.5 ms pulses) were sufficient. The inhibition had a short latency, about 20 ms, and was short lived. With single conditioning trains its duration only exceeded that of dorsal clitoris afferents activation by some 200 ms. These characteristics (afferent origin, immediate build up and termination) appear tailored to prevent the appearance of a micturition reflex during an increased bladder sensory load.

Repetitive electrical stimulation (0.5 ms pulses, 20 Hz, for 20 s) of the right bladder pelvic nerve at intensities sufficient to recruit bladder preganglionic neurones consistently depressed the C-fibre test reflexes. Lower stimulation intensities were ineffective, and transection of the right pelvic nerve central to the stimulation site suppressed the effect. The inhibition thus arose from activation of the bladder parasympathetic preganglionic neurones and had a central pathway, hence most likely via recurrent collaterals of these neurones (1,2). The functional properties of this inhibition significantly differed from those of dorsal clitoris inhibition. The depression had a slow build up, requiring several seconds to develop after the end of the conditioning stimulation. Its amplitude and duration strongly depended on the frequency and duration of the conditioning stimulus. A prolonged activation of bladder parasympathetic neurones was necessary to evoke the depression. Typically, it appeared for train stimulus durations of more than 5 s, and frequencies above 5Hz. After longer trains and at higher frequencies (30 s, 20 Hz), a maximal depression of the test down to 20% of control was observed, and several minutes were needed before complete recovery. This time course strongly resembles that of the depression occurring after large spontaneous bladder contractions.

Naloxone greatly enhanced all bladder reflexes and transformed ongoing phasic bladder efferent activity into a continuous discharge. In this situation dorsal clitoris inhibition remained virtually unchanged, but recurrent inhibition was completely abolished. This was true also when the size of the test reflexes was decreased to control levels by decreasing the test stimulation. The effect was reversible, and complete recovery occurred 40-60 minutes later.

Conclusions

Recurrent and dorsal clitoris evoked inhibition strongly affect both the spinal bladder C-fibre reflex and the ordinary Aδ micturition reflex. However, the two inhibitions display different properties. Those of dorsal clitoris evoked depression are suited to prevent the occurrence of micturition reflexes during sexual activity. Recurrent inhibition appears to be specifically linked to previous bladder preganglionic activity; its time course resembles that of the depression occurring after spontaneous reflex bladder contractions; and it is abolished by naloxone in parallel with the latter. This strongly suggests that this spinal recurrent inhibitory pathway contributes to the termination of the phasic detrusor contractions.

References

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