In Vivo Inhibitory Effects of Stimulation at the Central End of the Pelvic Nerve Severed from Urinary Bladder on Urinary Bladder Contraction in Rats

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Abstract—Two- or five-Hz electrical stimulation of the central end of the left pelvic nerve severed from the urinary bladder in rats inhibited bladder contraction induced by intravesical infusion of Tyrode’s solution. Inhibition of bladder motility by 2-Hz nerve stimulation appeared after pretreatment with strychnine (0.3 mg/kg, i.v.), naloxone (1 mg/kg, i.v.) and picrotoxin (1 mg/kg, i.v.). Hypogastric nerve stimulation, however, did not affect bladder contraction. These results suggest the presence of an inhibitory mechanism on the pelvic motoneuron activated by contralateral pelvic nerve stimulation in rats.

It has been reported that in cat urinary bladder, antidromic stimulation of pelvic detrusor motor nerve fibers results in suppression of spontaneous detrusor reflex activity (1-3). In the present experiment, I confirmed in rats that stimulation of the nerve bundle containing the pelvic motor nerve and sensory nerve from the urinary bladder inhibited the urinary bladder contraction induced by expansion of the bladder wall in a reflex manner. I therefore studied the effects of drugs on this inhibition.

The methods for recording bladder contractions and the separation of pelvic nerve fibers were described in our previous papers (4, 5). Male Wistar rats (weighing 250-350 g) were anesthetized with urethane (1.0 g/kg, s.c.) and α-chloralose (25 mg/kg, s.c.). After exposure of the bladder, a needle (1/4) attached to one end of a silicone tube (O.D., 1.0 mm and I.D., 0.5 mm, 30-40 cm in length) was inserted into the bladder through the left ureter, which was then ligated around the needle. In order to induce bladder contraction, Tyrode’s solution lacking glucose was infused intravesically through the silicone tube at a constant rate (0.8-1 ml/10 min), and the pressure signals were measured with a pressure transducer (Nihon Kohden, MPU-0.5) connected to a silicone tube via a T-tube. Cotton-wool swabs soaked with Tyrode’s solution were laid on the bladder to keep it moist, and the swabs were warmed with a lamp. The urinary branch of all of the left pelvic nerve fiber bundle was separated from the fibers innervating the rectum and cut near the prostate. The central cut end of the urinary branch and the peripheral one of both the right and left hypogastric nerves were electrically stimulated (1 msec duration and 5 V strength delivered from a stimulator (Nihon Kohden, S-5039)). During hypogastric nerve stimulation, the contraction of the left vas deferens was recorded via an isotonic transducer (Nihon Kohden, TD 112S) with a 4-g load. The vas deferens severed from the epididymis was carefully isolated from the connective tissue so as not to injure the blood vessel, and its cut end was connected to the transducer. Drugs, strychnine HNO₃, picrotoxin (Wako Pure Chem.) and naloxone HCl (a gift from Sankyo), were i.v. injected through the femoral vein.

When the central end of the pelvic nerve bundle was stimulated at 2 or 5 Hz for 3-5 min, bladder contraction was inhibited during the stimulation, and the bladder pressure increased as intravesical infusion of solution continued (Fig. 1A). Even stimulation at 1 Hz inhibited bladder contraction in about one
third of the rats. Therefore, the effects of drugs on inhibition induced by stimulation at 2 Hz were studied. Strychnine at a dose (0.3 mg/kg, i.v.; Fig. 1B) abolishing the recurrent inhibition by antidromic stimulation on the spontaneous contraction in cats (1), picrotoxin at a dose (1 mg/kg, i.v.) below the convulsive threshold, and naloxone at a dose (1 mg/kg, i.v.; Fig. 1C) reversing the inhibitory effect of morphine on bladder contraction did not affect the inhibition induced by electrical stimulation. When the hypogastric nerves were stimulated at 5 or 20 Hz at the onset of bladder contraction, the pattern of contraction was not affected, and the vasa deferentia in both sides were contracted (Fig. 2).

In cats the inhibitory mechanism involved in the antidromic stimulation of pelvic motor neurons has been suggested to be activation of the inhibitory interneurons by recurrent collaterals of pelvic motor nerves, inhibiting the contralateral pelvic motoneurons (Renshaw-type inhibition) (1–3). Inhibition induced by stimulation of pelvic afferent nerves from the bladder has been reported in the pudendal motoneuron of the cat bladder (6, 7). Therefore, in rats the inhibition of bladder contraction induced by stimulation of the pelvic nerve bundle at the central cut end was thought to result from the inhibition of the pelvic motoneuron. The present results did not suggest the presence of Renshaw-like cells in rats. In the experiments with cats (1), it was described that dihydro-β-erythroidine did not potentiate the inhibition induced by antidromic nerve stimulation and the effects of strychnine upon it were inconsistent. In the spinal cord or the pelvic ganglia, GABA, opiate and adrenergic receptors are involved in the inhibitory mechanism of bladder motility (5, 8–10). In rats, the adrenergic nervous system had no inhibitory action (Fig. 2, Ref. 11) and opiate receptors were not involved in the inhibition. The involvement of GABA receptors could not be clarified because a higher dose of picrotoxin could not be examined. Although the transmitter could not
be elucidated, interneurons and projection neurons would be involved in the inhibitory mechanism on contralateral motoneurons, since these neurons decussate in the spinal cord (12).

Fig. 2. Response to hypogastric nerve stimulation in the vas deferens (top trace) and the urinary bladder contraction (bottom trace) induced by intravesical infusion of Tyrode’s solution in rats during hypogastric nerve stimulation. Vertical bar in the top trace indicates the contraction of the vas deferens and that in the bottom trace indicates the bladder pressure. Hypogastric nerve stimulation was performed during the period marked (u).

References