

PROSTAGLANDIN E₂ RELEASE IN BLADDER ISOLATED FROM CHRONIC SPINAL RATS**Aims of Study**

Injury to the spinal cord can lead to hyperreflexic bladder because of the emergence of a spinal micturition reflex pathway. Recent studies have shown that various factors contribute to the increased excitability into the bladder afferent neurons in the spinal rats (1). It has been reported that prostaglandins (PGs) act as local modulators of reflex micturition in the pathophysiological condition (2). Furthermore, it has been reported that the increased local stretch of the mouse bladder induced an expression of cyclooxygenase-2, which was the rates limiting enzyme in PG synthesis (3). In the present study, we have measured the releases of PGE₂ in bladder strips isolated from chronic spinal rats, and have evaluated the effects of epithelium and smooth muscle stretching on PGE₂ releases.

Methods

Spinal cord was transected at the level of Th 8-9 in adult female Sprague-Dawley rats. After 8 weeks, the rats were sacrificed under pentobarbital anesthesia, and bladder specimen was obtained. Bladder strips with or without epithelium were suspended in organ bath filled with Krebs-Henseleit solution, and tension developments were recorded. The effects of elevation of the resting tension (0 to 4 g) induced by muscle strip stretching on PGE₂ releases were evaluated. Sham operated rats were used as a control group. In each resting tension, 1 ml of Krebs-Henseleit solution in the bath was collected, and the amount of PGE₂ released from bladder strip was measured by radioimmunoassay.

Results

In spinal rats, PGE₂ releases from bladder strips with or without epithelium were significantly higher, as compared to control bladder strips. In both spinal and control rats, the elevation of the resting tension by stretching caused increases in PGE₂ releases in bladder strips with epithelium, and % increases in PGE₂ releases from strips of spinal rats were significantly higher than that of control rats. However, in bladder strips without epithelium of both spinal and control rats, elevation of resting tension did not have significant changes in PGE₂ releases (table).

Table. Effects of epithelium and resting tension on PGE₂ release from bladder strips

Group	Epithelium	Basal PGE ₂ release: pg/mg tissue (Resting tension 0)	% Increase in PGE ₂ release			
			Resting tension, g			
			1	2	3	4
Spinal rats	With (n=7)	8.53±0.52	61.8±12.0	126.4±20.1	182.7±29.8	208.4±32.3
	Without (n=7)	4.02±0.43	22.0±5.5	28.2±4.3	27.4±5.0	30.0±5.6
Control rats	With (n=7)	3.42±0.34	32.2±4.8	52.0±7.7	78.0±8.6	80.6±10.3
	Without (n=7)	2.22±0.22	12.3±5.8	18.5±4.5	20.4±4.0	22.0±4.4

Conclusions

The present data demonstrated that there was the increased synthesis of PGE₂ from bladder strips in spinal rats, and that the elevation of resting tension caused increases in PGE₂ releases. The increased PGE₂ releases may contribute to detrusor hyperreflexia in spinal cord injury. It is also suggested that epithelium plays an important role on PGE₂ releases from bladder in spinal rats.

References

- 1) Progress in Neurobiology 57: 583-606, 1999
- 2) J. Physiol. 495: 492-440, 1996
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