urinary bladder to a higher extent than in control rat and human detrusor muscle. The upregulated response was of the B_1-receptor type and seems to include an increase in both the generation of prostanoids along the cyclo-oxygenase pathway, and in a *de novo* syntesis of receptors or specific proteins involved in the activation pathway of the BK receptor.

91

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THE EFFECT OF NITRIC OXIDE IN ISCHEMIA-REPERFUSION RAT BLADDER

Aims of Study: Since there is an increasing evidence suggesting that nitric oxide (NO) plays important roles in ischemia-reperfusion injury in the bladder,[1,2] we evaluated the effect of NO inhibitors on ischemia-reperfusion injury in the rat utilizing muscle bath and continuous cystometory studies.

Methods: Rat abdominal aorta was clamped with a small clip to induce ischemia-reperfusion (I-R) injury in the rat bladder dome as previously reported.[1] Since our preliminary experiments revealed that 30 minutes reperfusion caused more severe damage than 5, 10 or 20 minutes reperfusion, rats in the I-R and I-R+NA, I-R+NM groups were exposed to 30 minutes and 7 days reperfusion.[1] In the I-R+NA, I-R+NM groups, L-NAME (30 mg/kg) and L-NMMA (30 mg/kg) were injected i.p. 30 minutes prior to the ischemia, respectively, since our previous report indicated that this dose was effective in causing a significant reduction in the histological damages induced by ischemia-reperfusion in the rat urinary bladder.[1,2] Furthermore, detrusor pressure during voiding (Pdet) and capacity of the bladder were evaluated with continuous cystometry (C.CMG, infusion speed 12.6 ml/hr) in control, 30 minutes ischemia and 30 minutes ischemia-30 minutes reperfusion rats with or without the treatment of L-NAME (30 mg/kg).[3]

Results: Contractile responses to carbachol of the bladder strips, C.CMG and bladder capacity are shown in the Table 1 and 2. The contractile responses of the rat bladder dome under 30 minutes ischemia differed slightly but not significantly from those of controls. Reperfusion (30 min) gave significant reduction in contractile response to carbachol in the rat bladder (40.5% of control group). The treatment with L-NAME (30 mg/kg) significantly prevented the injury of reperfusion (59.4% of control group). Seven days after the induction of ischemia-reperfusion, the contractile response to carbachol was significantly improved compared to 30 minuets reperfusion group. Treatment with 30 mg/kg of L-NAME and L-NMMA significantly increased the contractile response to carbachol compared to the I-R group without L-NAME or L-NMMA measured seven days after ischemia-reperfusion induction. In the C.CMG studies, 30 minutes ischemia significantly decreased the Pdet, and subsequent reperfusion slightly recovered the Pdet in the rat. The Pdet from 30 minutes ischemia-30 minutes reperfusion rats receiving treatment with L-NAME (30 mg/kg) returned to basel revel and, was significantly recovered compared to that from the 30 minutes ischemia rats.

Conclusion: 1) Ischemia induced by clamping of the rat abdominal aorta caused reduction in contractile responses to carbachol of the bladder dome, and subsequent reperfusion caused additional damage to smooth muscle judged by functional study; 2) In contrast to these findings, *in vivo* study Pdet in 30 minutes ischemia rats was

significantly lower than that in controls, and subsequent reperfusion slightly recovered the Pdet; 3) Ischemia-reperfusion injury was prevented by treatment with NO inhibitors *in vivo* and *in vitro* studies.

TABLE 1

	Emax, gm/mm^2	% Control	ED_{50}, μM
Control	11.1 ± 1.3	100.0	1.9 ± 0.3
I	8.8 ± 0.7	80.0	2.4 ± 0.3[c]
I-R	4.5 ± 0.6[a]	40.5[a]	4.8 ± 0.9[a]
+ L-NAME,30 mg/kg	6.6 ± 0.4[a]	59.4[a]	4.5 ± 0.9[a]
I-R (1Week)	7.9 ± 0.7[b]	71.2[b]	0.9 ± 0.1
+ L-NAME (30 mg/kg)	9.0 ± 0.8	81.1	1.3 ± 0.2
+ L-NMMA (30 mg/kg)	9.2 ± 0.6	82.9	1.1 ± 0.2

Data are shown as mean ± S.E.M. of 6-8 separate determinations in each group. a) significantly different from any other group. b) significantly different from control and I-R groups. c) significantly different from ischemia-reperfusion groups. $p < 0.05$ is level of significance.

TABLE 2

	Pdet, cmH_2O	Bladder Capacity, ml
Basel	34.4 ± 1.8*	0.53 ± 0.09
I	29.5 ± 0.7	0.80 ± 0.18#
I-R	32.0 ± 1.6	0.63 ± 0.15
Basel (L-NAME, 30 mg/kg)	35.0 ± 1.9*	0.64 ± 0.15
I (L-NAME, 30 mg/kg)	28.2 ± 1.0	1.01 ± 0.12#
I-R (L-NAME, 30 mg/kg)	33.7 ± 2.7*	1.10 ± 0.14#

Data are shown as mean ± S.E.M. of 6-8 separate determinations in each group. *) significantly different from I group. #) significantly different from Basel group. $p < 0.05$ is level of significance.

References: 1)Life Sci. 62: PL149,1998. 2) Neurourol. Urodyn. 17: 409, 1998. 3) J Pharmacol Meth 15:157, 1986

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92

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VALSALVA AND COUGH URETHRAL PRESSURES : INTERCHANGEABLE OR COMPARABLE?

Aims of study: Valsalva and cough leak point pressures have been used to evaluate the outcome of continence surgery and identify low pressure urethras. The Valsalva leak point pressure (LPP) relies on the woman gradually increasing her intra-abdominal pressure until urinary leakage is seen whereas the cough LPP relies on the woman producing graded coughs of increasing pressure until she leaks. Cough LPP's are higher than valsalva LPP's in the same woman but this may be due to the method of increasing intra-abdominal pressure. A standardised technique does make the valsalva LPP reproducible and the technique has been found to be reproducible and correlate with urethral pressure profilometry. The aim of this study is to evaluate whether the mechanism of incontinence during a cough or Valsalva are the same by measuring the pressure transmitted to the urethra.

Method: Symptomatic women underwent urodynamics. At the end of the test a fluid filled rectal line was left in place and urethral pressure profilometry was carried out. Urethral pressure profilometry was performed using a 7F catheter with 2 solid state microtip transducers 4 cm apart. The technique of Hilton and Stanton(1) was used. The pressure along the urethra was then measured at three points, the proximal urethra, at the point of maximal urethral closure pressure and the distal urethra during a cough and a Valsalva manoeuvre. The intra-abdominal pressure was kept the same for both manoeuvres by simultaneously recording pressures through the rectal pressure catheter.

The urethral and intra-abdominal pressure rises during both manoeuvres were then compared using the technique of Altman and Bland (2).