therapeutic plasma levels (3) is a potent competitive antagonist on the α_{1A} /α_{1L} adrenoceptor-mediated contractile responses in human prostatic adenoma.

References

1. Br.J.Urol. 74: 572-578, 1994.
2. Br.J.Pharmacol.Ther. 14: 48-58, 1959.
3. Eur.J.Clin.Pharmacol. 37: 53-58, 1989.

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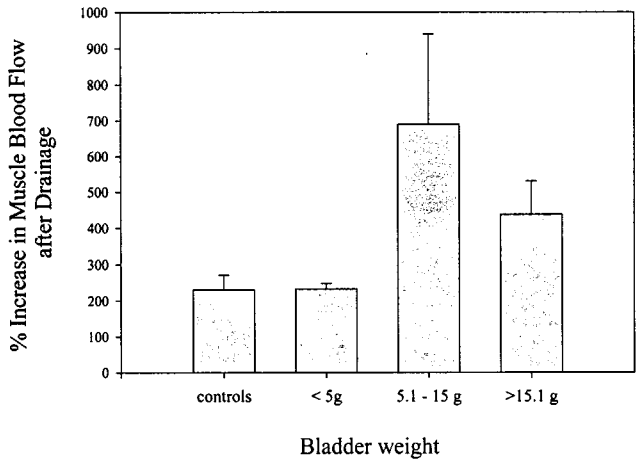
Title (type in CAPITAL LETTERS, leave one blank line before the text):

EFFECTS OF BLADDER CATHETERIZATION AND EMPTYING ON BLOOD FLOW AFTER OUTLET OBSTRUCTION

Aims of Study: Previous studies have shown that catheterization and drainage of the urinary bladder results in a significant nitric oxide-induced increase of blood flow to the bladder. It was also shown that long term obstruction causes a significant decrease in blood flow to the bladder. The purpose of this study was to determine the effects of catheterization and drainage on blood flow after 4 weeks of partial outlet obstruction.

Methods: 15 New Zealand White rabbits received a partial outlet obstruction by standard methods. After 4 weeks the rabbits were anaesthetized and blood flow to the bladder muscle and mucosa were determined by a standardized fluorescent microsphere technique. After transurethral catheterization and complete drainage of the bladder the blood flow was determined again. The same procedure was performed in 5 unobstructed control animals. The bladders were assingned to groups based on bladder weight and were named compensated (<5.1g), intermediately compensated (5.1.-15g) and decompensated (>15.1g).

Results: 4 weeks of partial bladder outlet obstruction caused a significant decrease of blood flow to the bladder muscle (without changes in the blood flow to mucosa) and a increases of bladder weight and residual urine. After drainage of the bladder the blood flow increased 6 fold in the obstructed group and 2.5 fold in the control animals (Figure). Within the obstructed animal group the increase was highest in the intermediate group. The severely decompensated bladders (weight > 15g) showed a limited ability to increase blood flow after drainage.



Conclusion: The overall decreased blood flow to the bladder smooth muscle appears to be an etiological factor in bladder contractile dysfunction secondary to partial outlet obstruction. The ability to increase blood flow after emptying the bladder could be an important determinant of compensation in the early phase of outlet obstruction. Loss of the ability to increase the blood flow after emptying might be the turning point from reversible to irreversible damage of the bladder due to outlet obstruction.

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BLADDER DYSFUNCTION IN ACUTE URINARY RETENTION AND SUBSEQUENT CATHETERIZATION

AIMS OF STUDY: Previously we have reported that ischemia-reperfusion causes bladder dysfunction, and that free radicals including nitric oxide play an important role on ischemia-reperfusion injury in the bladder.[1,2] It is well known that bladder ischemia is caused by urinary retention, but bladder dysfunction due to acute urinary retention is still unclear. In this study, we attempted to investigate bladder function, blood flow and vesical pressure in acute urinary retention and subsequent catheterization in rat urinary bladder. Furthermore, we attempted to measure malonaldehyde (MDA) and 4-hydroxyalkenals (4-HNE) as makers of lipid peroxidation in the bladder.[3]

METHODS: Eight weeks male Wistar rats were used in this study. Rat penile urethra was clamped with a small clip and cystostomy was performed to infuse 3ml of saline (infusion speed 24 ml/hour) to induce acute urinary retention. Thirty minutes after the induction of urinary retention, cystostomy was opened to make the bladder empty. In functional studies, contractile responses to carbachol and 100 mM KCl were measured in these conditions (before (A), 3ml of urinary retention (B), 3ml of urinary retention exposed 30 minutes (C), and subsequent 30 minutes after catheterization (D). Moreover, in vivo real-time monitoring of blood flow and vesical pressure were measured in the bladder with a laser Doppler flowmeter and cystometography, respectively. MDA and 4-HNE were measured by colorimetric assay in these groups (A-D). Statistical analysis of the differences between groups was performed using analysis of variance and the multiple comparison Fisher's test. $P < 0.05$ was regarded as the level of significance.

RESULT: Data of functional study and MDA, and 4-HNE are shown in the TABLE. In functional study, Emax values of carbachol to bladder in A, B, C and D groups were 11.8 $\pm$ 1.3, 11.9 $\pm$ 1.7, 9.8 $\pm$ 0.8, and 6.9 $\pm$ 0.7 g/mm^2, respectively (TABLE). Contractile response to 100mM KCl showed in the same manner as Emax values of carbachol. In real-time monitoring of blood flow and vesical pressure, acute urinary retention significantly decreased blood flow and increased vesical pressure, and subsequent catheterization increased blood flow and decreased vesical pressure in the bladder. The concentrations of MDA and 4-HNE in the bladder in the group D were significantly higher than that in groups A, B, and C (TABLE). Our data indicated that Emax value in the group D is siginificantly lower than the other groups, and lipid peroxidation in the group D is significantly higher than the other groups.