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Author(s)
A.Schroder[1,2], P.Chichester[3], B.A.Kogan[2], J Lieb[2], and R.M.Levin[2,3,4]

Institution, city, country
Department of Urology, Johannes Gutenberg-University, Mainz, Germany[1], Albany Medical College, Albany, NY[2], Albany College of Pharmacy, Albany, NY[3], Stratton VA Medical Center, Albany, NY[4],USA

Title (type in CAPITAL LETTERS, leave one blank line before the text)
EFFECT OF CHRONIC BLADDER OUTLET OBSTRUCTION ON THE BLOOD FLOW OF THE RABBIT URINARY BLADDER

Aims of Study: Previous studies have shown that the initial reaction of the rabbit urinary bladder to partial bladder outlet obstruction is an increase of blood flow at 1 day and a return to baseline blood flow at 1 week. Both mucosal and muscle blood flow followed this pattern, but mucosal blood flow was always 4-5 fold greater. In this study, we examined the effects of 4-weeks of outlet obstruction on the bladder blood flow and correlated it with the severity of bladder contractile dysfunction.

Methods. Fifteen New Zealand White rabbits underwent a partial outlet obstruction by standard methods. After 4 weeks the rabbits were anaesthetized and blood flow to the muscle and mucosa were determined by a standardized fluorescent microsphere technique. After completion of the blood flow procedure the intravesical pressure was determined by suprapubic puncture with a 18 gauge needle connected to a pressure transducer. After opening of the abdomen (and relief of the abdominal pressure) a control measurement of the bladder pressure was done. A section of each detrusor was used for in vitro contractility studies. Contractile responses to field stimulation, carbachol, ATP, and KCl were determined. A section of each detrusor tissue was fixed in formalin and used for determination of smooth muscle volume fraction.

Results: Four weeks of partial bladder outlet obstruction caused a significant and variable increase in bladder weight and a decrease of blood flow to the bladder muscle without changes in the blood flow to mucosa (Table 1). There was a clear correlation between the severity of contractile dysfunction, bladder weight and the magnitude of the decrease of blood flow in muscle The contractile responses to all frequencies of field stimulation were increased significantly in the obstructed bladders with a weight < 5g , but were reduced significantly in the bladders > 5.1g, which were considered decompensated. Bladders >15g showed a significantly reduced contractile response to carbachol and KCl The smooth muscle (SM) volume fraction remained stable at ~40%.

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Table 1 **Effects of chronic outlet obstruction on blood flow**

	Bladder Weight (g)	Urine Volume (ml)	Blood flow Muscle (ml/g/min)	Blood flow Mucosa (ml/g/min)	Volume Fraction Smooth Muscle (%)
Control	2.48 ± 0.24 (1.6 – 3 1.g)	30 ± 3 8 22-40 ml	0.08 ± 0.01	0.27 ± 0 04	45 ± 1.5
Obstructed	9.68 ± 1.6 (3.5 – 22 1g)	127 ± 30 (20-402 ml)	0.04 ± 0.01	0 32 ± 0 05	40 2 ± 2.0

Values represent mean ± SEM of 5 (control) and 15 (obstructed) animals Numbers in parameters indicate range.

There was no significant change in intravesical pressure with increasing urine volume After opening the abdomen wall there was significant decrease in intravesical pressure

Conclusion Bladder decompensation was associated with reduced blood flow to bladder smooth muscle. The decreased blood flow to the decompensated bladder muscle is not caused by an increase of intravesical pressure due to the higher amount of urine. From these studies, we hypothesize that decreased blood flow to the bladder smooth muscle is an etiological factor in bladder contractile dysfunction (bladder decompensation) secondary to partial outlet obstruction.