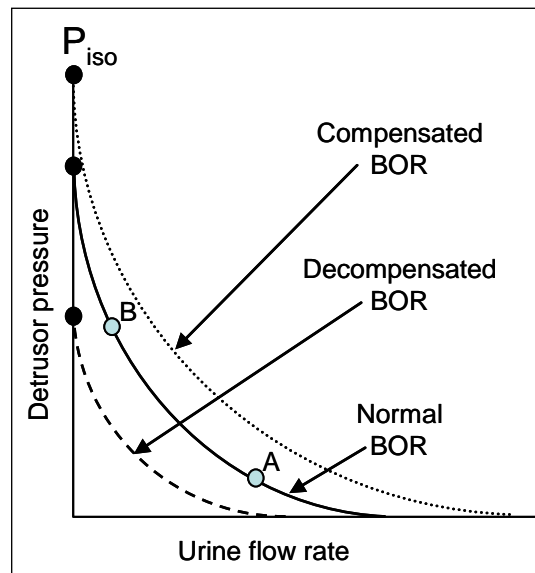


## CHANGES IN BLADDER CONTRACTILITY AFTER TRANSURETHRAL RESECTION OF THE PROSTATE

### Hypothesis / aims of study

The Hill equation describes the relationship between the force of muscle contraction and the velocity of muscle shortening. Translated to the bladder, this equation relates the detrusor pressure to the urine flow rate, which is called the bladder output relation (BOR) [1].

At zero flow rate, the BOR intercepts the y-axis at a pressure value which is called the isovolumetric bladder pressure ( $P_{iso}$ ). The  $P_{iso}$  is a measure for the bladder contractility and can be determined using the condom catheter method. During unobstructed voiding, the urine flow rate and detrusor pressure values are within normal limits (fig 1, position A). During progressive development of obstruction by prostate enlargement, voiding changes to a lower urine flow rate combined with a higher detrusor pressure (fig 1, position B). At this stage the bladder contractility has not changed, i.e.  $P_{iso}$  is the same. If the bladder compensates by an increase of the contractility as a consequence of hypertrophy of the detrusor muscle, the BOR moves towards the upper right corner, giving a higher  $P_{iso}$  (fig 1, dotted line). If decompensation occurs and bladder contractility decreases, BOR moves towards the lower left corner and  $P_{iso}$  is lower (fig 1, dashed line). We used the condom catheter method to study maximum urine flow rate and bladder contractility before and after trans urethral resection of the prostate (TURP), to establish whether the bladder was compensated or decompensated.



**Figure 1** Bladder output relation (BOR) of a normal (solid), compensated (dotted) and decompensated (dashed) bladder [1]

### Study design, materials and methods

Twenty-one male patients (age between 42 and 82) with lower urinary tract symptoms (LUTS) underwent a non-invasive investigation before and approximately 3 months after TURP. Inclusion criteria were: age  $\geq 18$  years, maximum free flow rate  $\geq 5$  ml/sec, mentally and physically able to visit the outpatient clinic, signed informed consent. If the patient was unable to urinate in the standing position, had had previous surgery or congenital disease of the lower urinary tract or heart failure, he was excluded.

The non-invasive investigation consisted of voiding into a uroflowmeter to determine the maximum free flow rate ( $Q_{max}$ , ml/s) and voiding through a condom catheter to determine the isovolumetric bladder pressure. During voiding through the condom, the urine flow was repeatedly interrupted to measure the pressure in the condom, the maximum of which ( $P_{cond,max}$ , cmH<sub>2</sub>O) reflects the isovolumetric bladder pressure [2].

Data were presented as median (InterQuartile Range, IQR). Differences before and after TURP were tested using the non-parametric Wilcoxon signed Rank (WR) test. Differences between groups of patients were tested using ANOVA with Bonferroni correction.

### Results

Overall, the patients ( $n=21$ ) voided with a  $Q_{max}$  of 7.9 (5.4) ml/s before TURP and had a  $P_{cond,max}$  of 102 (71) cmH<sub>2</sub>O. After TURP, the median  $Q_{max}$  was considerably higher (24.3 (14.9) ml/s,  $p<0.05$ ), whereas the  $P_{cond,max}$  was relatively unchanged (99 (69) cmH<sub>2</sub>O,  $p=0.61$ ).

In the majority of patients ( $n=11$ ), the increase in  $Q_{max}$  after TURP was accompanied by a decrease in  $P_{cond,max}$  (table 1, group 1). In 8 patients the increase in  $Q_{max}$  was combined with an increase in  $P_{cond,max}$  after TURP (group 2). In 2 patients the  $Q_{max}$  was not improved postoperatively and  $P_{cond,max}$  had decreased by 26 and 29 cmH<sub>2</sub>O, respectively. This was, however, not significant because of the small number of patients.

The parameter values were not significantly different between these groups before TURP. After TURP, group 2 had a significantly higher  $Q_{max}$  than group 3 and a significantly higher  $P_{cond,max}$  than both other groups.

**Table 1** Max free flow rate ( $Q_{max}$ ) and bladder contractility ( $P_{cond,max}$ ) before and after TURP

	$Q_{max}$ (ml/s)		$P_{cond,max}$ (cmH <sub>2</sub> O)	
	Pre TURP	Post TURP	Pre TURP	Post TURP
Group 1 Q↑ P↓ (n=11)	6.4 (5.1)	23.7 (11.8)*	110 (75)	95 (39)*
Group 2 Q↑ P↑ (n=8)	8.4 (10.7)	28.9 (9.0)*	99 (70)	137 (56)*
Group 3 Q≈ P↓ (n=2)	10.4 (0.9)	10.3 (0.8)	80 (46)	52 (48)

Q=urine flow rate; P=condom pressure; \* p<0.05 pre TURP vs Post TURP

#### Interpretation of results

Removal of an obstruction by TURP decreases urethral resistance and consequently increases maximum flow rate, as was seen in 90% of our patients. Based on the different BORs in figure 1 we hypothesize 3 different scenarios. Bladder contractility could be comparable before and after TURP, i.e. recovery from B to A in figure 1. The second possibility is a decrease in bladder contractility after TURP, i.e. recovery from compensated BOR to normal BOR. And thirdly, there could be an increase in bladder contractility after TURP, i.e. recovery of decompensated BOR to normal BOR.

In our data, most patients had a small but statistically significant decrease in bladder contractility, after removal of the obstruction and an increase in maximum flow rate. We hypothesize that these patients had a compensated bladder before TURP. Postoperatively, the bladder does not have to compensate anymore and adapts to the new non-obstructed situation, i.e. to normal BOR with a lower  $P_{iso}$ . Thus, compensatory hypertrophy of the bladder detrusor was reversible after removal of the urethral obstruction [3].

In the other group of patients, the bladder contractility increased after TURP. We hypothesize that these patients were decompensated and the increase in  $P_{iso}$  indicates recovery of the bladder contractility. The possible damage to the bladder as a consequence of the long-lasting increased urethral resistance (decompensation of the bladder) was obviously reversible in these patients. Such reversible contractile dysfunction is also seen in the "stunned myocardium", in which normalization of the blood flow is accompanied by reversible myocardial contractile dysfunction that will recover spontaneously. Analogously, we hypothesize that removal of the obstruction will normalise urine flow accompanied by spontaneous recovery of the decreased bladder contractility. We measured bladder contractility after 3 months, during which the bladder could have recovered to normal contractility levels.

The strong increase in  $P_{iso}$  after TURP in group 2 could be explained by the influence of the urethrovesical reflex, which is responsible for the maintenance of the detrusor contraction in the normal bladder. The 3-4 fold increase in urine flow after TURP could overstimulate the afferent nerves to the bladder and consequently increase contractility. In group 1, this urethrovesical reflex is opposed by the decrease in  $P_{iso}$  because of the shift from compensated BOR to normal BOR.

Two patients showed no recovery in flow rate at all. If lowering urethral resistance by TURP has no effect on the maximum flow rate, then apparently the urethra was not obstructed. The voiding dysfunction probably had another origin, i.e. disease of the bladder wall.

#### Concluding message

Generally, maximum urine flow rate considerably increased after TURP. This was mostly accompanied by a decrease, and sometimes by an increase in bladder contractility. These two observations may be related to adaptation from compensated BOR to normal BOR or spontaneous recovery of reversible bladder damage (stunning), respectively.

In two patients maximum flow rate was unchanged after TURP. Probably, other underlying pathology caused the voiding problems.

#### References

1. World J Urol. 13:30-33 (1995)
2. Neurourol Urodyn. 18:455-475 (1999)
3. J Urol. 158:89-93 (1997)

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