Factors determining the amount of residual urine in men with bladder outlet obstruction: Could it be a predictor for bladder contractility?

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Abstract Objective: To determine from urodynamic data what causes an increased postvoid residual urine volume (PVR) in men with bladder outlet obstruction (BOO), urethral resistance or bladder failure, and to determine how to predict bladder contractility from the PVR.

Patients and methods: We analysed retrospectively the pressure-flow studies (PFS) of 90 men with BOO. Nine patients could not void and the remaining 81 were divided into three groups, i.e. A (30 men, PVR < 100 mL), B (30 men, PVR 100–450 mL) and C (21 men, PVR > 450 mL). The division was made according to a receiver operating characteristic curve, showing that using a threshold PVR of 450 mL had the best sensitivity and specificity for detecting the start of bladder failure.

Results: The filling phase showed an increase in bladder capacity with the increase in PVR and a significantly lower incidence of detrusor overactivity in group C. The voiding phase showed a significant decrease in voided volume and maximum urinary flow rate ($Q_{\text{max}}$) as the PVR increased, while the urethral resistance factor (URF) increased from group A to B to C. The detrusor pressure at $Q_{\text{max}}$ ($P_{\text{det}Q_{\text{max}}}$) and opening pressure were significantly higher in group B, which had the highest bladder contractility index (BCI) and longest duration of contraction. Group C had the lowest BCI and the lowest $P_{\text{det}Q_{\text{max}}}$.

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Conclusions: In men with BOO, PVR results from increasing outlet resistance at the start and up to a PVR of 450 mL, where the bladder reaches its maximum compensation. At volumes of >450 mL, both the outlet resistance and bladder failure are working together, leading to detrusor decompensation.

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prostatic enlargement in 76 (prostate volume 55–90 mL) and bladder-neck obstruction in 14. Cystoscopy of these 14 patients showed a narrow and high bladder neck that was pliable, without contracture or fibrosis, suggesting a bladder neck dysfunction. Clinically, obstructive urinary symptoms were more common in groups A and B than C (mean IPSS 22, 28 and 13, respectively) with a statistically significant difference ($P < 0.001$). Overflow incontinence and nocturnal wetting were more apparent in group C than groups A and B (10/21, 0/30 and 2/30 patients, respectively, $P < 0.001$). Renal impairment and bilateral hydronephrosis (assessed by ultrasonography) were also more prevalent in group C than in groups A and B (mean serum creatinine levels of 2.3, 1 and 0.9 mg/dL, respectively; $P < 0.001$).

Analysis of the results of the filling phase of the urodynamic studies showed that the bladder capacity increased with the increase in the PVR among the three groups. Assessing the mean (SD) PVR in the three groups compared with the mean (SD) bladder capacity, the PVR was <20%, 20–70% and >70% of the bladder capacity in groups A, B and C, respectively.

The detrusor compliance was also low in all groups but with no statistically significant difference. Detrusor overactivity was significantly lower in group C than in groups A and B ($P = 0.048$; Table 1).

In the voiding phase, the voided volume and $Q_{\text{max}}$ decreased significantly as the PVR increased ($P < 0.001$ and 0.015, respectively; Table 1). The urethral resistance increased from group A to B to C, as shown by the significantly greater URF in group B than in group A ($P = 0.005$) and the further increase in group C above both groups A and B ($P < 0.001$ and 0.096, respectively; Table 1).

The $P_{\text{det}}Q_{\text{max}}$ was significantly high in group B, followed by group A then group C ($P = 0.028$ and 0.018, respectively). The effect on the opening pressure was similar, being significantly higher in group B than group A and group C ($P = 0.01$ and 0.04, respectively; Table 1). Group C had the lowest BCI, with a statistically significant difference from both groups A and B ($P < 0.001$). However, group B had a higher BCI than both groups A and C ($P < 0.001$). Also, group B had the longest duration of contraction, with statistically significant difference from group A ($P < 0.02$) but not from group C ($P = 0.4$; Table 1).

Discussion

The precise definition of the detrusor compensatory response to BOO is still controversial [11,12]. To date there is no agreement among urologists about the exact time at which detrusor compensation reaches its maximum limit before the bladder starts to fail [13,14]. We attempted to find a urodynamic explanation for the natural progress of chronic retention in men.

In the present study men with BOO were divided into one of three groups, with group A having obstructive symptoms with an obstructed voiding pattern on PFS but with an insignificant PVR. Group B had a significant PVR and an obstructed PFS pattern but still had good contractility. In group C the contractility was weakened, resulting in a greater PVR with an obstructed PFS pattern.

We tried to determine the cause of the progressive accumulation of the PVR in these patients; is it an increased outlet resistance, or bladder failure, or both. In group B the bladder contractility was good and even higher than that in group A, and the only factor responsible for the PVR was the increased outlet resistance. However, group C had a continuous increase in the urethral resistance with a concomitant decrease in the contractility, implying that both factors contributed to a greater PVR.

Most of the urodynamic variables showed that group B had the best contractility amongst the three groups, as shown by the highest $P_{\text{det}}Q_{\text{max}}$, the highest BCI and the longest duration of contraction. However, group C had the lowest contractility amongst the three groups, as shown by the lowest $P_{\text{det}}Q_{\text{max}}$, lowest BCI and, interestingly, the lowest incidence of detrusor overactivity (which requires a working detrusor muscle).

The ROC curve and scatter blots were very helpful for determining the point at which bladder compensation reaches its maximum limit before the bladder contractility changes from good to weak. The ROC curve showed that a PVR threshold of 450 mL had the best sensitivity and specificity to detect this change (Fig. 1). This was confirmed by scatter blots, where most of the data from patients with good contractility (BCI ≥ 100) were in the area with a PVR of <450 mL (Fig. 2).
There is still a debate about the use of the PVR as a predictor of acute urinary retention (AUR) in patients with BPH or after TURP. Some authors believe that the PVR is not a strong predictor of AUR [15,16], while others report that men were 3.6 times more likely to have a recurrence of AUR after TURP if they had a preoperative PVR of \( P \geq 500 \text{ mL} \) [17]. The present study supports the second opinion, because in this group of patients the bladder contractility is very weak, raising the possibility of postoperative AUR.

In conclusion, in men with BOO, the PVR results from an increasing outlet resistance at the start and up to a PVR of 450 mL, where the bladder reaches its maximum compensation and power of contractility. With a PVR of \( P > 450 \text{ mL} \) both the outlet resistance and bladder failure operate together, leading to detrusor decompensation.

![Figure 2](image-url)
Conflict of interest

None.

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