

TOWARDS DIAGNOSIS OF, BLADDER-NECK OR PROSTATE MIDDLE LOBE, 'DYNAMIC BLADDER OUTLET OBSTRUCTION' DURING VOIDING.

Hypothesis / aims of study

Bladder-neck hypertrophy and prostate middle lobe enlargement can cause symptoms of lower urinary tract dysfunction in male patients. However, the pathophysiology of this dysfunction is poorly understood. Surgical or other interventions on the basis of empirical knowledge are sometimes 'effective', but without understanding of the physiology of the bladder outlet dysfunction in these patients all approaches remain pragmatic and little scientific.

Bladder-neck hypertrophy (BNH), bladder-neck obstruction and prostate middle lobe (PML) enlargement are currently diagnosed on the basis of cystoscopy and-or video-voiding-cystography (with or without urodynamics) and-or with (transrectal) ultrasound. All these methods have the disadvantage of subjective interpretation of two-dimensional imaging. Bladder outlet function during voiding is, however, a dynamic feature with three, and when 'time' is included, four dimensions. A method of objective diagnosis of bladder-neck function during voiding has, apparently, been difficult to obtain and the relevance of an enlarged PML, in terms of bladder outlet obstruction or -dynamics has remained unveiled. Consequently, a gold standard for the diagnosis of BNH or PML – 'dysfunction' or -'obstruction' is lacking.

Pressure flow analysis can be applied for the diagnosis and grading of bladder outlet obstruction and the detrusor pressure at maximum flow ($P_{\text{det}} \text{ at } Q_{\text{max}}$) has shown relevance in the clinical practice for elderly male patients with prostatic enlargement. The ICS obstruction number (ICS-OBS) is based on $P_{\text{det}} \text{ at } Q_{\text{max}}$. A pressure flow (P/Q) graph or -plot, showing the pressure and flow relation of the entire voiding however, can provide additional information about the voiding process. The 'laws' of distensible collapsible tube hydrodynamics are helpful in clinical interpretation of pressure and flow dynamics during voiding.^{1,2} Minimum pressure required to ensure flow is a measure of collapsibility and $(P_{\text{det}} \text{ at } Q_{\text{max}})$ is a measure of distensibility or 'flow controlling zone'. Typically bladder outlet distension is maximal at the moment of Q_{max} . Subsequent to Q_{max} the pressure and the flow (and detrusor and bladder outlet) are in equilibrium and the termination of flow is associated with collapse of the bladder outlet. Previous studies have shown that pressure and flow are however not perfectly balanced throughout the entire voiding in every patient.^{2,3} Some have demonstrated variety in slope and curvature, when compared to the 'standard' and 'static' passive urethral resistance relation.³

We present our P/Q-plot observations in patients with BNH and-or noticeable PML enlargement. The diagnoses of PNL enlargement or BNH, as well as the indication for urodynamic investigation, were based on clinical -routine- arguments. This is a retrospective case-cohort series of patients without comparator; neither the sensitivity nor the specificity of our findings can precisely be computed. We regard this cohort study however, as a step towards better understanding of voiding dynamics in patients with BNH or PML enlargement.

Study design, materials and methods

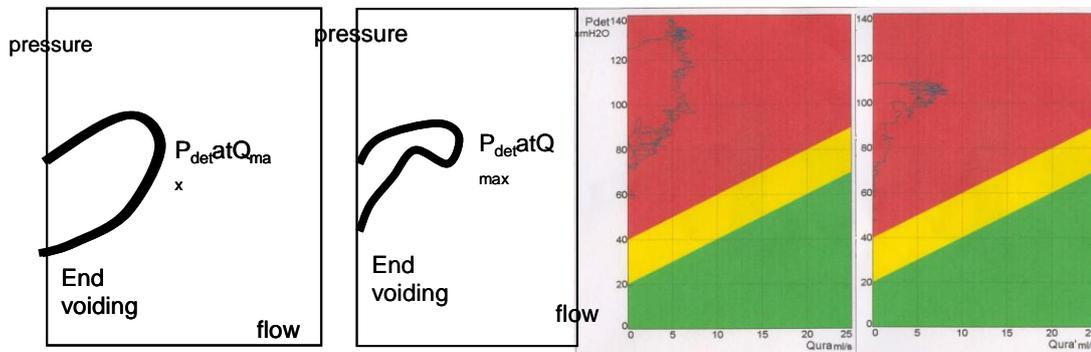
We have collected 23 patients with 'clinical' evidence of BNH or PML. Twelve of these had a 'high' bladder-neck ('BNH'), observed during outpatient cystoscopy. In eleven patients a 'protruding PML' was observed, by cystoscopy and-or by transrectal ultrasound. Two patients had a history of TURP and a remaining 'high' bladder-neck. The majority of the patients had transrectal ultrasound of the prostate and (uncatheterised) uroflowmetry preceding the urodynamic study. All patients completed the International Prostate Symptoms and bother Score. Patients were without relevant neurologic abnormalities or urinary tract infection.

The patients included in this cohort have had (ICS-) standard transurethral (F8 dual lumen catheter) urodynamic investigation with 25-50 ml/min fill, external fluid pressure transducers - subtraction cystometry and room temperature water until strong (but not very uncomfortable) desire to void. Voiding was permitted in the sitting position with weight transducer flowmetry and curves were corrected for the (short) time delay between pressure and flow. Cough tests were performed during cystometry and after voiding to control for equilibrated pressure response and consequently, adequate catheter position. Post void residual was measured via the transurethral catheter. What we present here is 23 *selected* patients that showed urodynamic evidence of 'dynamic bladder outlet obstruction'. (A not ICS standardized term that will be explained in the interpretation of results session.)

Results

Mean IPSS score of this group of male patients (mean 62 year, range 32-71) was 18 points (range 6-36 points) with average 4 (2-6 points) on IPSS bother question. Mean prostate volume was 31 grams (range 20-79 grams). Mean free flowmetry Q_{max} was 16.0 ml/s (range 8-31.7 ml/s) with a volume voided 291 ml (range 135-660) and average postvoid residual 118 ml (0-660). Average urethral resistance parameter URA was 25 cmH₂O and average Schäfer obstruction grade was 2. Only two patients had bladder outlet obstruction according to the current limits (BOO; URA >28 cmH₂O and Schäfer grade >2). Common observation in all these patients was however an upward deflection of the pressure flow curve in the second phase of voiding (where, in a typical voiding with BOO, a downward curve is expected). See figure 1 and 2 and figure 3.

	Schematically		Real study
Typical BOO	Bladder-neck BOO	Typical BOO	Bladder-neck BOO



Interpretation of results

The upward curvature of the P/Q plot -second (lower pressure-) phase of voiding indicates that there is a secondary increase in bladder outlet 'resistance' subsequently to Q_{max} . We speculate that the BNH or PML are causing this 'dynamic bladder outlet obstruction' in these patients, however, prospective and systematic comparison to a gold standard to evaluate the sensitivity and the specificity of these observations is needed.

This selected group of patients had on average moderate or no BOO on the basis of contemporary limits, a relatively good Q_{max} but a relatively large amount of residual urine and many symptoms with much bother. Probably the bladder-neck and-or a PML play a role in voiding dysfunction following the moment of Q_{max} as a result of bladder architecture during voiding. A contracted bladder is a 'shrinking' sphere and in these patients the outlet may become relatively more obstructive as a consequence of the diminishing (intravesical) volume and diminishing outlet lumen in combination with a relative lack of 'length stretching' of the bladder base. Likewise, 'capsizing' or 'turning over' of the -intravesical- PML towards the lumen of the outlet as a result of the diminishing bladder circumference is an explanation for our observations. 'Dynamic bladder outlet obstruction' (in the second phase of the voiding) might cause residual urine and symptoms despite good Q_{max} and relatively low grade of BOO (based on $P_{detatQ_{max}}$).

Concluding message

Detailed observation of pressure flow curve can explain pathophysiology of lower urinary tract dysfunction caused by BNH or PML enlargement. BNH and or enlarged PML cause a urodynamic P/Q pattern that is potentially sensitive and specific enough to be applicable in clinical practice.

References

1. Griffiths: Brit Jour Urol 1973
2. Schafer: In: Hinman1983
3. Spångberg: Neurourol Urodyn 1989

<i>Specify source of funding or grant</i>	NONE
<i>Is this a clinical trial?</i>	No
<i>What were the subjects in the study?</i>	NONE