251

Rosier P F W M[1]
1. *Department of Urology University Medical Centre Utrecht*

INTRA- PROSTATIC PROTRUSION SHOWS A TYPICAL URODYNAMIC PATTERN ON PRESSURE FLOW ANALYSIS.

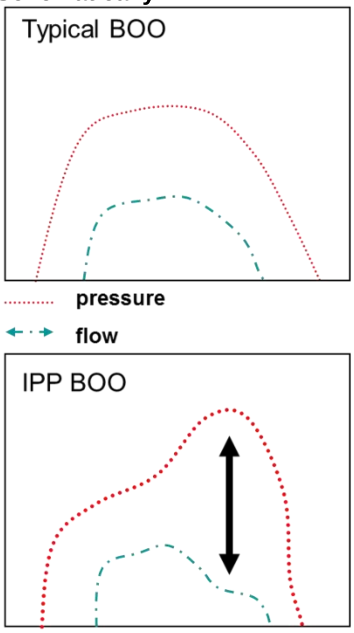
<u>Hypothesis / aims of study</u>
Prostate enlargement may cause symptoms of lower urinary tract dysfunction in male patients. Intravesical protrusion of the prostate middle lobe (IPP) has been reported by various research groups as a specific type of prostate enlargement, relevant for management. Reports suggest that patients with IPP do respond to a lesser extent on alpha blocking therapy and recent single centre studies and expert opinions suggest that these patients could specifically benefit from surgery.
The pathophysiology of the voiding dysfunction related to IPP is however poorly understood. A ‘ball valve’ obstruction type is suggested in some manuscripts, based on hypothesis or on cystoscopic appearance. IPP may be recognized on trans- rectal or trans- abdominal ultrasound, but the observation does not explain why IPP leads to failure of prostate (alpha- blocking) relaxing treatment.
Pressure flow analysis can be applied for the diagnosis and grading of bladder outlet obstruction and the detrusor pressure at maximum flow ($P_{det}atQ_{max}$) has shown relevance in clinical practice. The ICS obstruction number (ICS-OBS) is based on $P_{det}atQ_{max}$.
A pressure flow (P/Q) graph or -plot, showing the pressure and flow relation of the complete voiding however, provides 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_{det}at)Q_{max} is a measure of distensibility or ‘flow controlling zone’. Usually bladder outlet distension is maximal at the moment of Q_{max}. After Q_{max} the pressure and flow (and detrusor and outlet) are normally in balance and collapse of the bladder outlet is seen at the termination of flow.
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 P/Q-graph observations in patients with IPP as a step towards better understanding of voiding dynamics in these patients and have the aim to elucidate pathophysiology IPP dynamics and explain the relative resistance to pharmacotherapy of these patients.

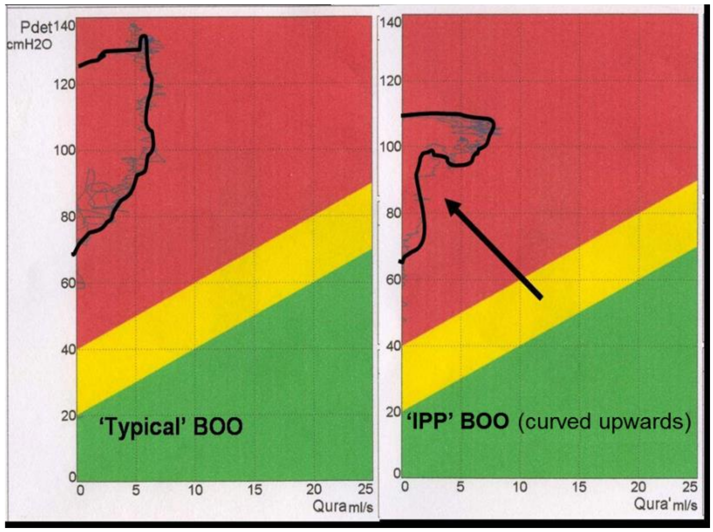
<u>Study design, materials and methods</u>: We have prospectively collected patients with IPP that underwent invasive urodynamic testing as part of our clinical routine. The patients had trans rectal ultrasound of the prostate and uroflowmetry preceding the urodynamic study. All patients completed the International Prostate Symptom and bother Score (IPSS).
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 room temperature saline cystometry until strong (but not very uncomfortable) desire to void. Voiding was permitted in the preferred position with weight transducer uroflowmetry 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.

<u>Result</u>
Mean IPSS score of this group of 23 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 uroflowmetry Q_{max} was 16.0 ml/s (range 8-31.7 ml/s) with a volume voided 291 ml (range 135-660) and average post void residual 118 ml (0-660). Average Urethral resistance parameter (URA) was 25 cmH_2O and average Schäfer obstruction grade was 2. Only two patients had bladder outlet obstruction on the basis of contemporary agreed limits; BOOI >40; URA > 28 cmH_2O and or Schäfer grade >2. Common finding in all these patients was however an upward deflection of the pressure flow curve in the second phase of micturition (where in a typical –obstructed- voiding a downward curve is expected).

See figures: left: typical pressure flow, time based pressure and flow are parallel; IPP pressure and flow deviate –towards increase of outlet resistance- after Q_{max} observable (right side) in an upward curve on the pressure low graph.

Schematically **Real study**





Interpretation of results

The upward curvature of the P/Q plot -second –lower pressure- phase of micturition indicates that (protruding) prostate middle lobe is causing an increase in bladder outlet 'resistance' after Q_{max}.

This upward second flow phase curve can therefore be regarded the as the urodynamic pressure flow analysis evidence for the until now hypothetical 'ball valve' obstruction effect.

This selected group of patients had on average moderate or no BOO, based on the commonly agreed limits, a relatively good Q_{max} but a relatively large amount of residual urine and many symptoms with much bother.

This study provides the urodynamic evidence that he prostate middle lobe plays a role in voiding dysfunction following the moment of Q_{max} as a 'capsizing' or turning over' of the –intravesical- prostate middle lobe towards the lumen of the outlet. Outlet dynamics and increasing grade of 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 $P_{det}atQ_{max}$).

Concluding message

Intravesical prostate protrusion causes a 'typical' P/Q urodynamic pattern that provides a fundamental explanation for ineffective voiding in a proportion of patients with relatively high maximum flow rates. The dynamic middle lobe 'ball valve' effect may also explain relative resistance to pharmacological management in some of the male patients with LUTS. Detailed observation of pressure flow curve has explained pathophysiology of lower urinary tract dysfunction caused by prostate middle lobe enlargement and intravesical protrusion.

References

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

Disclosures

Funding: none **Clinical Trial:** No **Subjects:** HUMAN **Ethics not Req'd:** Evaluation of standard medical management **Helsinki:** Yes **Informed Consent:** No