



Initial observation of the urodynamic pressure flow study — characteristics of bladder-neck or prostate median lobe dynamics during micturition

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ABSTRACT

Purpose: We present our observations of a urodynamic pressure flow study graph pattern that may be associated with bladder neck hypertrophy or prostate median lobe enlargement.

Materials and Methods: We report 23 male patients with signs and symptoms of lower urinary tract dysfunction who underwent urodynamic testing with pressure flow study analysis. We recognized an unusual pressure flow plot curve and found that these patients had cystoscopic evidence for bladder neck hypertrophy or cystoscopic or ultrasound evidence for prostate median lobe enlargement.

Results: The patients here demonstrated showed a pattern of increasing detrusor pressure on the pressure flow study — graph phase after maximum of flowrate, where a decreasing pressure pattern is expected. We contemplated that this pattern may show how bladder outflow dynamics is affected by the anatomical features of these patients and how patients may have symptoms of LUT dysfunction or significant residual urine despite a relatively good maximum flow rate.

Conclusion: We present a not previously described variation of the pressure flow study graph pattern common in men with prostatic hyperplasia, in a series of patients with median lobe prostatic hyperplasia and or hypertrophy of the bladder neck.

1. Introduction

A urodynamic pressure flow study (PFS) is gold standard for the diagnosis and grading of bladder outflow obstruction BOO. Detrusor pressure at maximum flow ($P_{\text{det}}Q_{\text{max}}$) is commonly used in clinical practice [1] and the ICS bladder outlet obstruction index(BOOI) [1] is based on $P_{\text{det}}Q_{\text{max}}$ and Q_{max} . A PFS graph or -plot (or loop), showing pressure versus flow of the entire voiding in a continuous X-Y graph, provides additional information about the voiding process [1–6], and is recommended as the ICS-standard, in addition to the time based graph, since 1998 [7]. In the example figure of this ICS standard, the PFS-plot is shown with pressure on the vertical axis and flowrate on the horizontal axis [7]. The distensible collapsible tube hydrodynamics — paradigm [2,7,8] is principal for clinical interpretation of pressure and flow dynamics during voiding and predicts the archetypical PFS loop of a voiding [1,3,6]. The paradigm includes that ($p_{\text{det.at}}$) Q_{max} is a measure of distension of the flow controlling zone in the bladder outflow tract and that the lowest pressure that maintains flow is a measure of collapsibility [8]. In the physiology of voiding the detrusor pressure opens –distends– the bladder outlet to its maximum at Q_{max} following on to $p_{\text{det.max}}$. After Q_{max} there is normally a balance between pressure and flow loop as is explained in the earliest clinical manuscript of Abrams and Griffiths in 1979 [6] and in the ICS-standard [7].

the balance here mentioned, is seen in the gradual and synchronous decline of pressure and flowrate after the moment of Q_{max} . Complete collapse of the bladder outflow controlling zone occurs eventually, at the termination of flow, or at the moment that the detrusor pressure decreased to below the outlet pressure [4]. The period between maximal distension (Q_{max}) and complete collapse (the second part of the voiding) is regarded a ‘balanced’ and ‘passive’ phase during a normal voiding whether bladder outflow obstruction is present or not [2].

Earlier studies have shown however that pressure and flow are not always perfectly balanced throughout the entire voiding in every patient [3–5,9]. Neurogenic dyssynergia is a typical example, however a variety in slope and curvature of the second part of the PFS graph, when compared to the physiological urethral resistance relation, [3,4] may also be present in patients without LUT-relevant neurology [3–5,10]. Especially two earlier reports have observed that the collapse-pressure is not always representing a passive balance between pressure and flowrate after Q_{max} and noted this pressure to be relatively high, which has been discussed as dys-elasticity (to associate with dys-synergia) as a variant of collapsibility [5,10].

This study reports a cohort of men in which we found a PFS loop that deviated from the loop pattern depicted in the earlier report and the ICS-standard, which we considered ‘unusual’ and not as expected

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for normal (BPH) outflow tract collapse. We will explain this later in the manuscript and first replicate what we consider contemporary knowledge about bladder-neck hypertrophy (BNh) and prostate median or middle lobe enlargement (PMLe).

In general: Symptoms of lower urinary tract dysfunction (LUTS) in men are very often caused by the challenges that prostate enlargement poses to the LUT. With or without prostate enlargement, BNh as well as PMLe may play a role in male LUT dysfunction leading to LUTS.

The bladder-neck, when cystoscopically appearing as a (citation from [11]: ‘It is not necessary to go over the pathologic anatomy or the symptoms except to state that there is in all a...) ...bar, a tight collar or a firm, hard fibrous non-dilatable ring’, is used as the explanation for male lower urinary tract (LUT) symptoms (LUTS) already since very early descriptions [11,12]. Surgical interventions are considered effective on the basis of plausibility and empirical knowledge and e.g., the Hugh Hampton Young cold punch operation to cut the bladder-neck, has been the first specific technique to cure ‘voiding symptoms and or retention’, associated with BNh in men [13]. Alpha-blockers have become an addition in the therapeutic armamentarium of symptomatic BNh, without very much specific evidence, and bladder-neck (electrocautery) incision has replaced cold punch nowadays [14]. Further clarification of the pathophysiology postulated a century ago, is however unavailable and objective grading and or staging of the dysfunction caused by BNh or PMLe is not possible. Apart from cystoscopic diagnosis, cystography has been evaluated for these patients however without a clear-cut conclusion regarding the pathophysiology and without relevance to demonstrate the dynamic aspects of the condition during the course of voiding [15,16].

The diagnosis of a PMLe protruding in the bladder [17] is deemed relevant as a proxy for bladder outflow obstruction (BOO) [18]. Resection of the prostate median lobe is done with TURP [19] and proved to be feasible also with laser resection [20,21]. Clinical studies have, on the other hand, concluded that alpha blocking therapy is less effective in men with significant PMLe [22,23]. Surgical methods to preserve normal ejaculation, avoiding bladder-neck or median lobe damage or resection are emerging for ‘male LUTS’ [24] and specific, objective diagnosis of the relevance of the bladder-neck or the prostate median lobe as a cause for the symptoms in an individual patient may therefore become more relevant. Awareness of BNh or PMLe dynamics during voiding in a patient with LUTS could be of help for the individualization of the management plan. BNh or PMLe-resection is associated with improved flowrate and reduction of symptoms in the predominance of the patients. However better diagnosis and knowledge of the individual’s pathophysiology may also lead to more precise selection of contemporary management possibilities [24].

BNh as well as PMLe have stood the test of time as being important anatomical features with or without prostate enlargement associated with symptomatic dysfunction of the LUT. Both BNh and PMLe are visible with cystoscopy, cystography and or with (trans rectal) ultrasound but these observations are observer dependent and little standardized although intravesical protrusion can be measured on ultrasound images [17].

Although there is agreement on the anatomical features of BNh and PMLe the pathophysiology of the voiding dysfunction is still imprecisely understood. Bladder outlet (urethral lumen) function during voiding is a feature with four dimensions when (voiding) time is added to length and 2 dimensional cross-sectional area and any imaging would only show 2 or 3 of these. It is difficult to make reliable and truthful images of outflow dynamics with or without bladder-neck ‘extra’ dynamics during the process of voiding because imaging always misses at least one dimension. The relevance of BNh or PMLe for BOO or outflow dynamics can therefore only be studied in an unreliable way by imaging. Consequently, a standard for the objective diagnosis or the grading of BNh or PMLe-BOO does not exist and specific standard terms for this dysfunction are lacking. Although higher grade of BOO and or detrusor overactivity and or PMLe were reported to be associated

with bladder trabeculation, cystoscopy was not very specific in grading of the BPH-BOO [25] and it is never shown that the cystoscopic appearance of BNh or PMLe by itself is very specific to diagnose the LUT voiding dysfunction or to quantify BOO grade.

The patients that we report here are ‘found’, and clustered here because of their unusual PFS-plot appearance. We have considered that the anatomical features that were subsequently also found in these patients may be responsible for the urodynamic-pathophysiologic-unusual PFS-loop.

2. Study design, materials, and methods

The patients were not prospectively recruited for this study, but only clustered, based on alertness to unusual PFS patterns. The indication for urodynamics in all these patients has (only) been based on our local routine clinical standard, that is in accordance with our national urologists’ guideline. In our teaching – university – hospital we perform cystometry and PFS analysis in all elderly male patients referred with LUTS resistant to pragmatic initial therapy. Our standard includes also that male patients with LUTS have a transrectal prostate ultrasound and furthermore some, with a somewhat higher incidence (lower threshold) than recommended in the (international) guidelines, have a cystoscopy.

The included patients underwent ICS-standard transurethral double lumen F8 catheter urodynamic pressure subtraction study with 35–50 ml/min fill rate, fluid filled tubing with external pressure transducers – cystometry and room temperature water until strong, not uncomfortable, desire to void as per good urodynamic practice [26] in agreement with the newer edition [27]. PFS was permitted in standing 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 monitor balanced response of pressures. Postvoid residual urine (PVR) was measured via the urodynamic catheter after PFS. Most of the patients had (not catheterised) uroflowmetry and PVR preceding the urodynamic study. All patients completed an IPSS with bother score.

We report 23 selected patients with an unusual PFS loop pattern. This pattern-type will be explained in the results session.

3. Results

This group of male patients were mean age 62 year (range 32–71) with an average IPSS score of 18 points (range 6–36 points) and average IPSS bother score of 4 (2–6 points). Prostate volume was thirty-one grams (range 20–79 grams). Mean free uroflowmetry Q_{max} was 16.0 mL/s (range 8–31.7 mL/s) with a 291 mL voided (range 135–660 mL) and average PVR 118 mL (0–660). The mean URA (urethral resistance parameter) [8] was 25 cmH₂O and the mean LinPURR obstruction grade [28] was 2. Two patients only, had BOO when standard ICS grading; BOOI >40 and or URA >28 cmH₂O and or LinPURR-grade >2 was applied.

A common observation however, in all patients here included, has been an upward deflection of the PFS graph-curve in the second phase of micturition (where in a typical – obstructed – voiding a downward curve is expected). See Fig. 1. This is not reported earlier and therefore we will specifically discuss its relevance here below. In comparison with the expected or ‘typical’ urethral resistance relation (Fig. 1a) the pressure has been more than 20cmH₂O too high in a large part of the curve in all the patients here described.

After noticing these 23 patients with an unusual PFS-loop pattern we searched for other, potentially coexisting features of these patients. We then found that in twelve of these patients an elevated bladder-neck was observed with outpatient cystoscopy. Two patients had an earlier TURP and a cystoscopically diagnosed elevated or contracted bladder-neck. In eleven patients the ultrasound showed a protruding median lobe, observed also by cystoscopy in two patients.

Further post hoc observation:

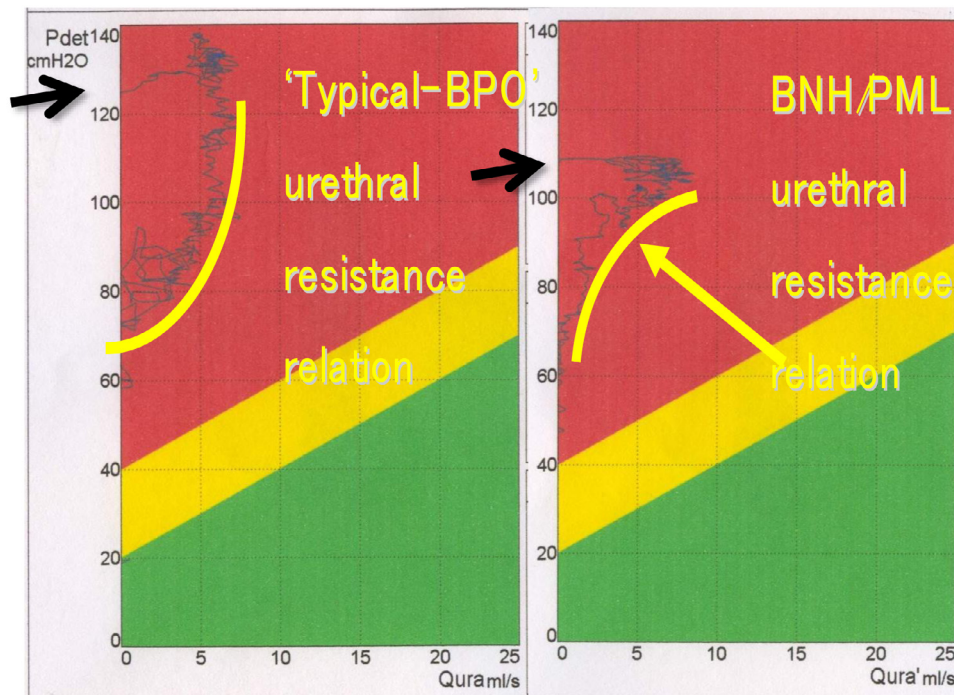


Fig. 1. The left side picture shows obstructed voiding in a P/Q graph: the left hand side picture is the usual or 'typical' pattern of BOO seen with prostate enlargement; flowrate begins at high pressure (see the arrow) and declines with a convex downward curve-pattern until the minimum pressure that produces flow. The right hand side shows that subsequent to Q_{max} the pressure is relatively high and the curve 'kinks-in' with a concave upward pattern. This demonstrates the relatively increasing grade of BOO after Q_{max} which is not the usual pattern as is described in references 2, 4, 6 and 7.

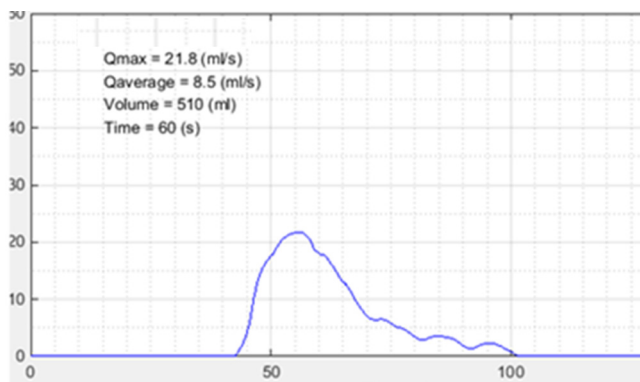


Fig. 2. The flowrate here demonstrated shows a good Q_{max} and a prolonged tail end; associated with BNH in this patient.

While analysing this set of patients we also noticed that we could also recognize the characteristic voiding disturbance in the free uroflowrate, and we give an example in Fig. 2. A prolonged – tail-end of the flowrate pattern was visible in the majority (14/23) of these patients however not all (other) uroflow tests were of an adequate volume or were reported to be representative.

4. Interpretation of results

These patients had a relatively large number of bothersome symptoms and PVR but also on average absent to moderate grade of BOO as diagnosed with the standard PFS analysis and a relatively high Q_{max} . The curve of the PFS plot, showing increasing pressure (with decreasing flowrate) in the second phase of micturition, is unusual and reveals an increase of bladder outflow resistance in the voiding phase after Q_{max} .

To further explain: Fig. 1 left side represents the normal (=most frequent and most physiological [3,6,7,9]) pressure and flow sequence

during –obstructed- voiding; the loop turns clockwise in time [3,7]. The detrusor pressure rises after the voluntary initiation of voiding and at a given moment (arrow at 124cmH₂O) the pressure is high enough to open the outlet (the flow controlling zone [2], usually the prostate, is pushed open) the flow begins and rate increases until maximum Q_{max} . From that moment there is a fixed balance (curve over-projected) between the by the detrusor voiding contraction generated pressure in the bladder and the urethral resistance. The collapsing pressure is a measure of BOO [5–8,29].

In the patients that we demonstrate here the pressure-flow equilibrium of the micturition phase after Q_{max} is disturbed and the outflow resistance is relatively increasing instead of 'steady' [4] after Q_{max} . The bending to the higher pressure lower flow region, subsequent to Q_{max} as Fig. 2 shows, is sign of that increasing outflow resistance. The overall difference in curve shape following Q_{max} is indicated schematically.

We selected/collected the men with an unusual PFS-loop pattern that deviated more than 20cmH₂O, which is larger than the test–test variation in male PFS [30], but nevertheless arbitrary. We have not evaluated-included patients with PFS loops that had lesser deviation. Subsequently we found an anatomical feature, that could be a plausible reason for the deviation from the usual loop. All men had a PMLE or BNh found independently from (and before) the urodynamic tests.

We postulate, based on these observations how the bladder-neck and or the prostate median lobe play a role in the pathophysiology of the dysfunction. Following on to the moment of Q_{max} , bladder-neck or the PML kink-in, and increase the outflow resistance because of the changing bladder shape during voiding. A contracting and emptying bladder is a shrinking sphere and in the patients with PMLE and or BNh, the initially funnelled outlet may become relatively more obstructive because of the 'additional' (more than usual, without PML or BNh) diminishing of vesical outflow tract circumference. The lack of continuation in bladder outlet funneling as was probably observed earlier [31] and this, or a disturbed circular length elasticity of the bladder base and outlet cause the here denoted type of BNh obstruction and the typical PFS pattern. Capsizing of the – intravesical– PML towards

the lumen of the outlet, in association with reduction of intravesical volume may be an explanation for our observations. Increasing BOO after Q_{\max} , in the second phase of the voiding, may cause PVR and symptoms despite relatively normal Q_{\max} and relatively low grade of BOO (based on $P_{\det} Q_{\max}$).

This is an initial retrospective case-cohort; it is not possible to calculate the sensitivity or the specificity of these initial observations. That these 23 patients were found in a consecutive series of 120 male patients that had urodynamic testing may give an indication. At present we are unable to conclude whether it is possible to discriminate between BNH or PMLe solely based on PFS patterns. Nevertheless, we regard this series can become an advance in the knowledge of the pathophysiology of the voiding of male patients, with or without prostate enlargement, with BNH and or with PMLe. We even postulate also that these observations may become relevant to further understand female –possible– prolapse associated voiding dysfunction [32].

Certainly, we agree that these observations should be prospectively and independently confirmed and the sensitivity as well as the specificity of this ‘unusual’ PFS pattern should be further studied. Other series with adequate data can be retrospectively studied or better, prospective series would allow calculation of the sensitivity and specificity of the PFS pattern for the anatomical features if the imaging is done separate and blinded for the urodynamic testing and analysis. Nevertheless, we now present these urodynamic observations for the first time with our observation that all these patients with this unusual PFS-plot pattern, also had noticeable PMLe or BNH.

5. Conclusion

We postulate that the pathophysiology of lower urinary tract dysfunction caused by bladder-neck hypertrophy or prostate median lobe enlargement can be explained with detailed analysis of the ICS standard pressure flow study graph and plot. Bladder neck hypertrophy and or enlarged prostate median lobe can cause a recognizable and face valid pressure flow study pattern that may become helpful in the grading and the staging of this specific male lower urinary tract dysfunction. We also present a flowrate pattern that may be(come) useful as an initial marker for the relevance of prostate median lobe or bladder neck hypertrophy as a cause of the individual men’s symptoms of dysfunction.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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