EXPERIMENTAL EVIDENCE FOR THE BLADDER CONTRACTILITY MODELS OF SCHAFER AND GRIFFITHS

Hypothesis / aims of study
Bladder contractility – the ability of the bladder muscle to generate and maintain the pressure for voiding – is an important feature of urodynamic diagnosis. It is insufficient simply to measure voiding bladder pressure, because this depends heavily on urine flow rate. The maximum contraction pressure can be established only against a closed outlet (isometric conditions); when flow is underway, the pressure inevitably drops. This phenomenon was described for skeletal muscle by the Hill equation, but is equally applicable to the smooth muscle of the bladder (detrusor).

Two well-used models predict the relation between flow rate and bladder pressure. In the Schafer nomogram (1) straight lines delimit very weak, weak, normal and strong contractility. Derek Griffiths (2) derived a model from the Hill equation as applied to a spherical bladder. Here, contractility is expressed as the Watts Factor (WF); pressure has a complex relationship with flow which involves bladder volume. This is potentially the more accurate model but is more difficult to apply in clinical practice.

Experimental evidence to support either model has been scarce, because it requires measurement of bladder pressure and flow rate for a range of different outlet conditions. During normal voiding, flow rate is relatively fixed until the bladder relaxes at the end. By presenting a variable outlet resistance, linear inflation of a penile cuff provides the means to vary the flow rate while the bladder maintains its contraction. Using this method, the aim of the present study was to provide experimental evidence to support the existing theoretical models for bladder contractility.

Study design, materials and methods
We studied 117 men being investigated for lower urinary tract symptoms. Each underwent voiding cystometry according to the ICS guidelines for good urodynamic practice. The bladder was filled to cystometric capacity, and the subject was instructed to void. When voiding was established, a penile cuff was inflated at 10 cmH₂O s⁻¹ until flow was interrupted (3). The cuff was then deflated, and the process repeated until there was no resumption of flow. Flow rate, vesical pressure, abdominal pressure and (by electronic subtraction) detrusor pressure were recorded continuously during the entire study.

47 men were unobstructed, 38 equivocal and 32 unobstructed according to the provisional ICS nomogram. For each subject separately, we plotted the relation of detrusor pressure with flow rate. Where a patient had more than one cuff inflation, we used the one with the highest initial flow rate. We then divided the graph into rectangles of 1ml s⁻¹ x 5 cm H₂O, and calculated the mean path of the pressure-flow curve for each rectangle (Figure 1). The results are shown in Figure 2.

Figure 1. The pressure-flow relation for 117 men with lower urinary tract symptoms. We show three traces that follow the typical pattern, with the remaining 114 in grey. The mean pressure-flow relation was calculated for each of the 960 rectangles separately.
As predicted by the theory, there was an inverse relation between flow rate and bladder pressure. As flow reduced by inflating the cuff (moving right to left on the graphs), the bladder pressure increased. The general shape of the observed relation was particularly similar to that predicted by Griffiths. At low flow rates (the left of the graph), the observed increase was similar to that predicted by theory. However at higher flow rates, the pressure rise observed in our experiments was less than predicted.

Concluding message
The theoretical models of Schafer and Griffiths predict a rising bladder pressure as flow is progressively obstructed. Our experimental data show marked similarities to the theory, but with some discrepancy between the predicted and observed pressure rises. We hope these preliminary results will stimulate further debate on the physiology of the obstructed bladder.

References

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