Original Article

A Mathematical Model for Micturition Gives New Insights into Pressure Measurement and Function

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Abstract: Our objective was to analyze the factors contributing to the development of detrusor pressure during micturition in the female with reference to a mathematical model. One hundred patients with predominantly stress incontinence were investigated with micturition pressure studies. Frictional and dynamic losses were estimated at various flow rates using a mathematical model. Almost 25% of patients recorded a micturition pressure below 11 cmH₂O at peak flow (mean 23 cmH₂O, range 0-91). Large inter- and intrapatient variations in micturition pressures were recorded on retesting. The low pressures were explained by a recently described external opening mechanism, backward stretching of the vagina during micturition by the muscles of the pelvic floor. This opened out the outflow tract and created the potential for a falsely high P_{abd}. The large variability in micturition pressures on retesting was attributed to changes in urethral radius being magnified to the fourth power. It was concluded that, micturition itself, and the components for pressure generation, are complex non-linear entities which appear to be greatly modified by the external striated pelvic floor opening mechanism. Addressing anatomical defects in this mechanism may be a fruitful route of future enquiry in females with emptying problems.

Keywords: Micturition; Muscle force; Pelvic floor; Urethral resistance; Urine flow

Introduction

Voluntary micturition is said to occur by activation of the micturition reflex and relaxation of the striated muscle of the urethra and pelvic floor [1]. Quantification of urine flow, detrusor pressure, bladder work force and urethral resistance are all based on this concept.

The flow rate through an open tube is governed by the pressure difference across the tube, the tube's geometry, the surface roughness and the fluid properties. The pressure difference, $\Delta P = P_{ves} - P_0$ is the difference between the pressure of the fluid contained within the bladder, the intravesical pressure, P_{ves}, and the pressure acting at the tube exit, P_0 . The intravesical pressure is the sum of the abdominal pressure and the pressure caused by bladder muscle contraction, the 'detrusor pressure'. The intravesical pressure at a given flow rate varies according to the urethral resistance, and, in turn, with the fourth power of the radius [2]. Existing mathematical models are based on frictional resistance to flow through the urethral tube [3] or urodynamic parameters through an elastic tube [4–6]. The urethra is generally assumed to be a straight, smooth-walled circular tube carrying fully developed turbulent flow. Measured detrusor pressure and flow rate are used to determine an 'effective' urethral diameter and therefore, urethral restriction. Recent dynamic video X-ray studies have demonstrated that the urethra is not a straight tube, either at rest (Fig. 1) or during micturition (Fig. 2) [8,9]. Furthermore, contraction of specific muscles of the pelvic floor, the levator plate and longitudinal muscle of the anus, may play an important role in acting as an external mechanism for opening out the urethra during micturition [8,9]. The importance of abrupt changes in crosssection (e.g. 'funneling') of the tube shape (Fig. 2) and

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Fig. 1. Resting position of pelvic organs, normal patient. Continent patient, sitting. This is a sitting lateral X-ray in the resting position with a full bladder (B) incorporating a Foley catheter; radio-opaque dye has been injected into the Foley balloon, vagina (V), rectum (R) and levator plate (LP); superior border of LP = broken lines; the anterior diagonal line composed of small solid circles represents pubourethral ligaments (PUL), and the posterior circles the uterosacral ligaments (USL). CX = cervix; U = urethra; X = insertion point of bladder base to vagina; S = sacrum; 'rs' = ballooned area in the rectum; the intersecting white dotted horizontal and vertical lines are reference lines for organ movement drawn from fixed bony points.



Fig. 2. Micturition. With reference to Fig. 1 (same patient), the vagina (V) and bladder base appear to have been stretched open ('funneled') by a backward force (arrow); the anterior edge of LP is angulated downwards, apparently by a downward force (arrow); 'funneling' bladder neck is consistent with having been caused by (external) pelvic floor muscle forces [15]. Labeling otherwise as per Fig. 3.

contributions of frictional and dynamic components has now, for the first time, been assessed in model systems [10], based on these video observations. The aim of this study was to interpret clinical detrusor pressure readings obtained during standard testing with reference to the potential effects of an external opening mechanism, and also an experimentally validated mathematical model for dynamic and frictional losses [10].

Materials and Methods

One hundred consecutive female patients with urinary incontinence were referred for assessment. The mean age was 49 years (range 32–73), and parity 3 (range 0–7).

Urodynamic Testing

All patients were tested for bladder instability by supine filling cystometry, using methylene blue-colored saline at room temperature, infused at a rate of 100 ml/min. Gaeltec microtip transducers were calibrated before use in a water column between 5 and 50 cm in height. The transducers were inserted into the bladder and posterior vaginal fornix, then zeroed electronically. Vaginal, bladder and electronically subtracted detrusor pressures were recorded on a five channel Ormed system 5000. The filling was stopped at the point of discomfort, or after 500 ml had been infused. With the same transducers in situ, rechecked for correct positioning, the patient urinated on a commode; micturition pressures, rate and duration of flow, and the ability to voluntarily interrupt the stream were recorded. The pressures obtained at peak flow prior to interruption were noted. Pressure micturition studies were repeated on another day in 3 patients who recorded zero pressure at peak flow, in the patient with the highest recorded detrusor pressure (91 cmH₂O), and, purely for comparison purposes, in 5 other patients with detrusor pressures ranging between 8 and 20 cmH_2O .

The mathematical model used was based on a smooth-walled tube of uniform diameter carrying a fully developed flow of water, kinematic viscosity 0.01 cm^2/s and density of 1000 kg/m². The diameter of the tube was equal to the distal diameter of the urethra. The aim of the model was to study the relationship between frictional pressure drop, dynamic pressure change and the geometry of the urethra. This allowed an assessment to be made of the sensitivity of the overall pressure drop along the urethra relative to changes in the geometry.

The following formula was used:

$$\Delta P=P_{ves}-P_0=rac{8
ho Q^2Lf}{\pi^2 d^5}+rac{1}{2}
ho V^2-
ho g\Delta h$$

where the pressure difference ΔP is the difference between the intravesical pressure P_{ves} , and the pressure acting at the tube exit P_0 . Q is the volume flow rate, ρ is the fluid density, g is the acceleration due to gravity, f is the frictional factor, and Δh is the change in height of the urethra from one end to the other (approximately 2 cm). This model has been shown to provide an excellent first approximation to the pressure versus flow rate characteristic of an actual urethral tube [10]. The definitions and descriptions used here conform to the standards of the ICS (1988) [11]. Local Ethics Committee approval was obtained for the studies.

Results

Forty-six patients had urodynamically diagnosed detrusor instability, and 54 had genuine stress incontinence. The mean peak flow micturition pressure was 23 cmH₂O. Micturition pressures recorded ranged from 0 cmH₂O to 91 cmH₂O. Almost 25% of patients recorded a micturition pressure below 11 cmH₂O at peak flow,* with 9 of the 100 patients recording a subtracted detrusor pressure at 0 cmH₂O at peak flow (Fig. 3). The peak flow rates for these 9 patients were (ml/s) 42, 32, 10, 38, 20, 30, 25, 15, 25 (mean 26.4 ml/s). Bladder volumes for



Fig. 3. Zero micturition pressure at peak flow. Graphs indicate, from above down, bladder pressure, subtracted detrusor pressure, flow, abdominal pressure. Arrow indicates detrusor pressure at peak flow; 'vol. interrupt.' indicates point of voluntary interruption of flow. 'E' = the sudden rise in detrusor pressure on completion of micturition.



Fig. 4. Detrusor pressure as a function of the urethral diameter. For a tube length of 4 cm. , frictional component; – – – – –, dynamic component; — — , total.

these 9 patients ranged between 359 and 530 ml (mean 433 ml). Residual urine for this group ranged between 0 and 80 ml (mean 22 ml). Repeat testing in 3 patients with zero micturition pressures recorded pressures of 22, 20 and 24 cmH₂O, respectively, at peak flow. In the patient with 91 cmH₂O pressure, 24 cmH₂O was recorded on retesting. On repeat testing, patients with initial micturition pressures of 8, 10, 16, 20, 20 cmH₂O had pressures of 16, 50, 30, 26, and 12 cmH₂O. A sudden rise in pressure 'E' (Fig. 3), at the end of micturition was found in the majority of cases.

The mathematical model results are summarized in Fig. 4. Frictional and dynamic losses increased markedly with flow rate. Combined frictional and dynamic pressure losses at a flow rate of 50 ml/s were approximately 50 cmH₂O for a diameter of 4.7 mm, and 95 cmH₂O at 4.0 mm diameter. With reference to Fig. 4, the minimum detrusor pressure required to produce the peak flow recorded in the 9 patients who recorded zero micturition pressure, ranged between 10 and 42 cmH₂O.

Discussion

External Opening Mechanism

Video X-ray and EMG studies [8,9] suggested that the urethral tract was stretched open, much like a trapdoor (Fig. 2). This in turn was activated by contraction of the pelvic floor [8,9]. Bladder smooth muscles have characteristics that predispose to tonic contraction and urine loss, including unstable all-or-none action potentials, low-resistance pathways between cells, and modification by excitatory or inhibitory nerves. Thus the detrusor spasms during micturition [12]. This is consistent with video observations [8,9], which demonstrate a sudden uniform shrinking of the bladder until it

^{*}It is emphasized that the reading of $0 \text{ cmH}_2\text{O}$ refers only to a single point on the graph. It does not imply that the patient micturated at $0 \text{ cmH}_2\text{O}$.

empties. This gave rise to the explanation proposed elsewhere [8] for electrical silence during micturition. The water column is incompressible, so that once the ure thra has been externally opened out there is no further need for muscle contraction unless further opening is required. This, too, has been observed using intracavity EMG electrodes [8]. The contractile force of muscle is constant [13]. The detrusor pressure recorded varies entirely with resistance within the tube [14]. If there is no resistance, all the energy is converted to flow and little if any pressure is recorded [14]. A striated muscle external opening mechanism stretching an elastic urethra [8,9] would reach a peak of maximal extension. In turn, this would create maximal urethral opening, and this would be reflected in a lower detrusor pressure [14] at that point.

Pelvic Floor Contraction May Cause Inaccuracies in Micturition Pressure Measurements

Even a transient 0 cm micturition pressure recording (Fig. 3) is highly unlikely. Frictional and dynamic losses alone, at an estimated 4.7 mm diameter and flow rate of 20 ml/s, require a pressure head of 12.5 cmH₂O and approximately 45 cmH₂O at 50 ml/s. An outflow diameter of 14 mm (cross-sectional area 154 mm²) (Fig. 4) is required for frictional and dynamic forces to be negligible. A 14 mm diameter, though possible, is greater than the radiologically observed diameters of this and other studies [7]. Total pelvic floor relaxation plus gravity cannot explain the low micturition pressures recorded in this study, especially given the low residual volumes (22 ml recorded). A spherical bladder containing a urine volume of 433 ml creates a gravitational pressure equivalent to only 5.2 cmH₂O.

Comparative examination of the radiographs (Figs 1, 2) indicates that the cavities of vagina and rectum appear to be significantly stretched and narrowed during micturition, whereas the cavity of the detrusor remains unaltered. Intravaginal narrowing at the site of recording would record a falsely high p_{abd} . Alternatively, if the vaginal movement resulted in the microtransducer being exposed to a greater area initially, p_{abd} could be falsely low. Based on reference to Fig. 4, we believe that the mean 'detrusor pressure' in this study was most likely falsely low.

With reference to Fig. 2, a transducer placed in the ballooned area of the rectum, 'rs', approximately at the level of the bladder may be a more favorable position. However, even this part of the rectum appears to have been compressed during micturition. Strictly speaking, then, only a naked intraperitoneal transducer could be certain of giving an accurate p_{abd} . The well known phenomenon of micturition during straining with no apparent detrusor contraction is explained with reference to Fig. 2. Relaxation of the forward force operating during urethral closure [8,9] would instantly allow the backward and downward forces (arrows, Fig 2), to pull open the outflow tract. The intra-abdominal pressure

would then create a head of pressure to drive out the urine. Such a component appears to be present in Fig. 3. It is not possible to say whether this is voluntary abdominal straining, or secondary to the external striated muscle opening mechanism [8,9]. These concepts of external opening [8,9] have been applied clinically. Surgical repair of the ligamentous insertion points of the muscles hypothesized to activate the external opening mechanism have improved emptying both symptomatically and objectively [15,16].

A Particular Detrusor Pressure Finding is Rarely Repeatable

Other retest pressure micturition studies [17] support our findings that a particular detrusor pressure measurement is rarely repeatable. This is not surprising, given the dependence of urethral resistance, and therefore, detrusor pressure, to the inverse of the fourth power of the radius. A detrusor pressure of 24 cmH₂O obtained on repeat testing translates to a urethral diameter approximately 40% larger than that for the original 91 cm pressure.

Within the individual patient the work performed by a contracting muscle, e.g. detrusor, is constant [14,15], a concept most recently confirmed by Khullar et al. [17]. Given that the likely constancy of the stress extension curve of the only other variable, urethral tissue resistance, one would not expect 40% variation in urethral diameter in the same patient if detrusor contraction were the only factor operating to expel urine. Variations in urethral diameter are more easily explainable, however, by minor variations in recruitment of the hypothesized external striated muscle opening force [8,9] (Fig. 2). The quantum of change in the urethral diameter magnifies the detrusor pressure change to the fourth power. The stretching of the vagina and bladder base backwards and downwards (Fig. 2) is consistent with such an external opening mechanism for the urethra.

Postmicturition Pressure Rise

The sudden rise in detrusor pressure ('after contraction') on the completion of micturition, 'E' (Fig. 3) was a frequent finding in this study. It is consistent with a detrusor contracting against a urethral tube suddenly closed by elastic recoil of the vaginal hammock from the stretched position (Fig. 2) back to the resting closed position (Fig. 1) following cessation of the (external) opening forces (arrows, Fig 2). Continued contraction of the detrusor against the now closed urethra is registered as a sudden (isometric) pressure rise. Concepts which attribute distension of the outflow tract solely to detrusor contraction [4–6] cannot explain postmicturition pressure rise. Cessation of urine flow would also necessitate cessation of detrusor contraction.

Conclusion

Micturition and the components for pressure generation are complex, non-linear entities which appear to be greatly modified by the external striated pelvic floor opening mechanism. Addressing anatomical defects in this mechanism may be a fruitful route of future enquiry in females with emptying problems.

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EDITORIAL COMMENT: The authors in a previous series of articles presented a concept of an external striated muscle opening force, involving the pelvic floor muscles which assist in micturition. The theory being that a downward, backward contraction of the pelvic floor muscles opens the urethra, allowing for micturition with associated detrusor contraction. In this current study they use an experimentally validated mathematical model to support their hypothesis from the previous studies, utilizing data from micturition flow studies obtained from patients with documented lower urinary tract dysfunction. Dr Petros presents an interesting theory that, with continued evaluation of anatomical defects in the micturition mechanism, we may uncover answers to the many questions regarding female voiding dysfunction.