Review Article | Basic and Mechanism Sciences

A brief physiology and pathophysiology of the bladder

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Abstract: The remit of this review is confined to experimental works and publications relevant to the integral theory of female urinary incontinence (IT). Since its first publication in 1990, the IT has challenged the general view that the pathogenesis of overactive bladder (OAB) (urge, frequency, nocturia) is unknown and there is no cure. According to the IT, normal function bladder control is binary, either closed or open. Control is cortical via a peripheral feedback component: oppositely acting reflex striated pelvic muscles contract against suspensory ligaments to close the urethra for continence, open it prior to evacuation, and stretch the vagina like a trampoline to prevent excess impulses from the urothelial stretch receptors which may cause unwanted urgency at low bladder volumes (OAB). The pathogenesis of female urinary incontinence is from outside the bladder, mainly weak ligaments or vagina, due to collagen deficiency. Damage in childbirth (collagen depolymerization) and age (collagen loss) make ligaments vulnerable to damage. With weak ligaments, muscles contracting against them weaken: the muscles cannot close the urethra (manifested as stress incontinence), open it (manifested as emptying problems or retention) or stretch the vagina to prevent the urothelial stretch receptors firing off prematurely (manifested as urge incontinence). Weak pubourethral ligaments can cause stress urinary incontinence (SUI), or SUI plus urge (mixed incontinence). Weak uterosacral ligaments (USLs) can cause urge, frequency, nocturia and emptying difficulties. Treatment consisting of surgical/non-surgical strengthening of ligaments can cure or improve SUI, OAB, and emptying dysfunctions. In summary, bladder control is from outside the bladder, binary, with cortical and peripheral components. A small change in definition, from "overactive" to "overactivated" is consistent with this concept, retains the acronym "OAB", and opens the door to probability of cure and a massive increase in research endeavours.

Keywords: Binary control; pathophysiology; urethra; ligaments; pelvic muscles

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Introduction

The key points of the article are summarized in the video abstract (Video S1).

Foreword by Professor Axel Ingelman-Sundberg, Karolinska Institutet 1990:

"*To me it has always been obvious, that, in general, the reason behind female urinary incontinence has to be looked for outside the bladder, that is, in the structures supporting the urethra and bladder neck—specifically ligaments, pelvic floor muscles and vagina. If symptoms of urinary incontinence arise from a dysfunctional anatomy in the aforementioned structures, then function should come with restoration of anatomy.*"

The remit of this review is confined to experimental works and publications relevant to the integral theory of female urinary incontinence (IT). Since its first publication in 1990, the IT has challenged the general view that the pathogenesis of overactive bladder (OAB) (urge, frequency, nocturia) is unknown and there is no cure. The key discovery in the 1990 integral theory was that the control of bladder function was not from the bladder itself, but from structures outside it, by three oppositely acting reflex striated pelvic muscle forces (1) (*Figure 1A*, large arrows) (see Video S2). These muscles contracted against suspensory ligaments, anterior pubourethral ligament (PUL) and posterior uterosacral ligament (USL), to close the urethra for continence, open it for evacuation, and to stretch the vagina like a trampoline to prevent excess impulses from the urothelial stretch receptors which may cause unwanted urgency (OAB). The remit of this review is based on integral theory publications including (but not confined to) references (1-18) (https://obgyn.onlinelibrary.wiley.com/toc/16000412/1990/69/S153).

Highlight box

Key findings

- Control of the bladder is not from the bladder itself, but from ligaments and muscles outside of it, with ligaments most vulnerable to damage because of collagen changes at birth and after menopause.

What is known and what is new?

- Overactive bladder (OAB) and emptying problems are said to have unknown pathogenesis and no cure.
- Bladder control is binary, either closed or open, cortically controlled, peripherally activated, by pelvic muscles contracting against suspensory ligaments to close urethra (continence), open it, and to stretch the vagina to prevent premature micturition (urgency).
- Pathogenesis of female urinary incontinence is mainly from outside the bladder, from weak or loose ligaments and in special circumstances, from nerve or muscle damage.
- OAB is a prematurely activated but otherwise normal micturition.
- Weak ligaments are an important cause of SUI, urgency (OAB), and emptying dysfunctions [underactive bladder (UAB)].

What is the implication, and what should change now?

- OAB, UAB, and SUI are potentially surgically curable by ligament repair. "Repair the structure (ligament) and you will restore the function".

The binary model for normal bladder function

With reference to *Figure1A*, the binary control works like an electric switch to control two different reflexes, either closed or open (micturition) (2) (see Videos S2,S3). These reflexes are mutually exclusive, with cortical and peripheral components for each function. Muscle spindles work via a reflexly coordinated feedback system to maintain the balance of forces which tension the vagina (*Figure 1B*). Closure is the dominant reflex.

Control of urgency

The opposite contractions of the pubococcygeus muscle (PCM), and the levator plate (LP) and conjoint longitudinal muscle of the anus (LMA) tension the vagina to support the urothelial stretch receptors "N" from below (*Figure 1A*). Tensioning stretches the vaginal collagen in the manner of a trampoline to support the hydrostatic pressure exerted by the urine column on "N" (see Video S4). This reflex action prevents "N" firing off the afferent emptying impulses which activate the micturition reflex to empty, felt as "an urge to go" by the patient (1).

Urethral closure on effort

With reference to *Figure 1A*, the PCM pulls the distal vagina forwards against the PUL to close the urethra from behind; the LP stretches the proximal vagina and urethra backwards against the PUL to tension them; the LP contracts down against the USL to rotate the now tensioned bladder base around the arc of Gilvernet to close the urethra at the bladder neck (1) (see Video S2).

Micturition

With reference to *Figure 1A* (1) the PCM relaxes; the LP/LMA pull open the posterior wall of the urethra (white broken lines) to reduce resistance to urine flow from the

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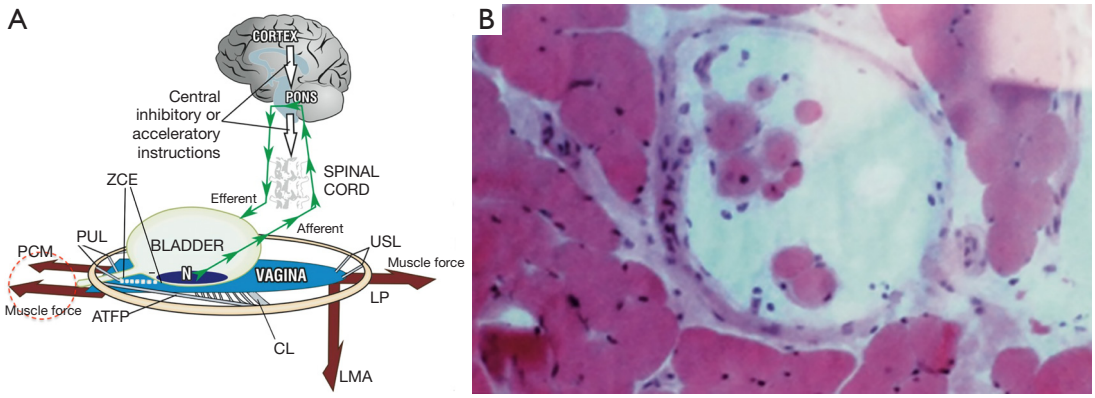

Figure 1 Mechanics of bladder control (reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd ed. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright). (A) Binary circuit for control of bladder function. Cortical control of urge: (large white arrows) can directly block afferent urge impulses (small green arrows) which activate micturition. Peripheral control of urge: the cortex activates the three directional muscles to prevent OAB by stretching the vagina like a trampoline to support the stretch receptors "N" from below. For micturition, the "PCM" relaxes (red broken circle); the "LP/LMA" can now pull open the urethra to facilitate micturition (white broken lines) by exponentially lowering the resistance to flow. Ligaments: USL, uterosacral; PUL, pubourethral; CL, cardinal; ATFP, arcus tendineus fascia pelvis. Muscles: PCM, pubococcygeus; LP, levator plate; LMA, conjoint longitudinal muscle of the anus. ZCE is the "zone of critical elasticity", which acts like an elastic hinge to allow the forward and backward muscle vectors (large arrows) to operate independently of each other. (B) Muscle spindle. The muscle spindle was found in the anterior portion of the pubococcygeus muscle. Vaginal tension in (A) supporting the urothelial stretch receptors N is reflexly controlled by the cortex. The muscle spindles adjust the length of the muscle to tension the vagina. OAB, overactive bladder.

contracting detrusor. Active opening by external muscle forces exponentially lowers the resistance to urine flow to enable evacuation (inversely by the 4th power of the radius "Poiseuille's Law") (see Video S3).

Pelvic muscle or ligaments, which is the main cause of bladder dysfunction?

The integral theory paradigm (ITP)'s main focus is loose or weak ligaments caused by altered collagen (1). Pathogenesis can be congenital, pregnancy/childbirth related (*Figure 2*) or menopausal (collagen breakdown/excretion). Clearly muscle damage must be a factor in pathogenesis. However, the high cure rates for pelvic symptoms attained following ligament repair (3) confirm the integral theory's view that ligament damage is the main cause. A further validation of this view was a blinded biopsy study of 47 women undergoing a midurethral sling operation for stress incontinence; 44 women had gross histological muscle damage, yet 89% were cured of SUI the next day (4).

Pathogenesis within the binary model

With reference to *Figure 1A*, any abnormality in the binary control circuit can affect the micturition reflex to cause retention or OAB. Neurological lesions such as stroke (cortex), spinal cord injury, multiple sclerosis (MS) in the afferent circuit, can cause retention. MS in the efferent nerve circuits can affect peripheral control to cause urge. Local lesions such as inflammation or a tumor near "N" can increase afferent impulses to cause OAB and incontinence.

Urge as a prematurely activated uncontrolled normal micturition

A urodynamically controlled experiment demonstrated what was then known as "detrusor instability" (now "DO") was equivalent to a prematurely activated micturition (5). The pattern of urine loss was identical to that seen in normal micturition: (I) sensation of urge; (II) fall in proximal urethral pressure; (III) rise in detrusor pressure; (IV) urine loss.

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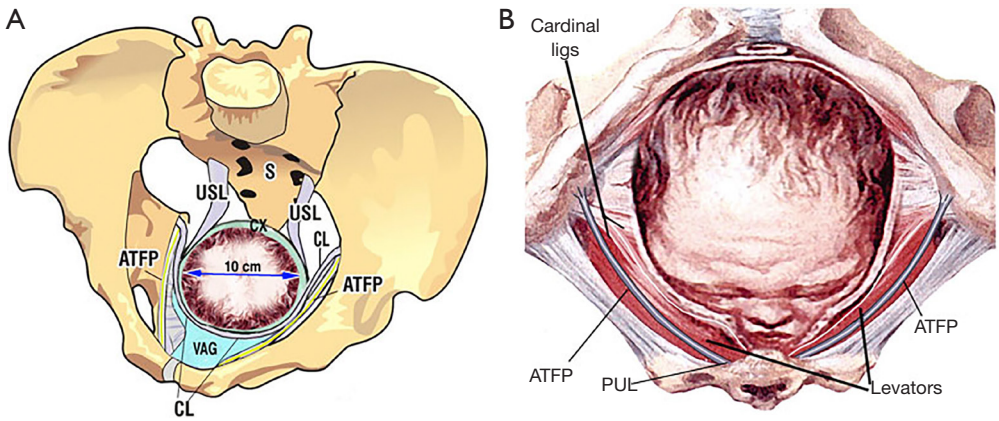

Figure 2 Pathogenesis of pelvic organ prolapse (reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd ed. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright). The head stretches or tears proximal parts of the structures suspending the organs. Ligaments may also be congenitally loose or weakened by post-menopausal breakdown of collagen. (A) A head at 10-cm full dilatation, may overstretch the cardinal ligament "CL", uterosacral ligaments "USL", and tear the vaginal attachment "VAG" to the cervix to cause cystocele and uterine/apical prolapse. (B) At the outlet, the pubourethral ligaments "PUL" levator attachments to the symphysis, may be overstretched and the perineal bodies pushed laterally, so the rectum pushes through into the vagina to cause a rectocele. The levator attachment to the symphysis may also be torn, stretched or dislocated. ATFP, arcus tendineus fascia pelvis; S, sacrum.

OAB (urge, frequency, nocturia)

With reference to *Figure 3*, weakness in either the PUL or USL will weaken the muscles which contract against them. Weakened PCM and LP/LMA muscles cannot adequately tension the vagina to support "N" to prevent the afferent emptying impulses reaching the cortex which are interpreted as "urge to go". Beyond a critical point the micturition reflex temporarily takes control, the system becomes unstable, and swings between "open" and "closed", which is the key characteristic of OAB and urodynamic "DO" ("detrusor overactivity") (6). See also https://doi.org/10.1002/nau.24990 (2).

Emptying: urinary retention

With reference to *Figure 3*, weak USLs weaken the contractile force of the LP and LMA muscles which contract against them. The weakened LP/LMA cannot open the urethra prior to micturition. Consequently, the detrusor contracts against a relatively unopened urethra against high urethral resistance, which the cortex interprets as "obstruction", which is what it is (7,8).

The pathogenesis of nocturia

A loose USL cannot prevent the proximal vagina and bladder base from being pulled down by gravity "G" (*Figure 4*). The downward stretching pulls on the stretch receptors "N" which activate the micturition reflex which the cortex interprets as "urge". A tampon can sometimes alleviate nocturia, if it can sufficiently support the weakened USLs.

Surgical cure of lower urinary tract symptoms (LUTS)

Following the first report of cure of SUI and urge by the prototype midurethral sling operation in the 1990 integral theory (1), a principal focus of the ITP has been surgical cure of prolapse and LUTS by ligament repair and to explain how restoring collagen to a damaged ligament could cure such a wide range of LUTS.

How surgery cures nocturia and urgency

With reference to *Figure 4*, reinforcing the USLs with a

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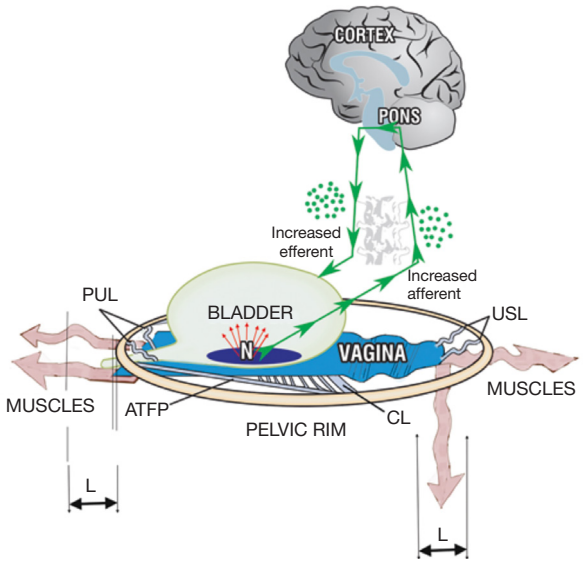

Figure 3 The anatomy of urge incontinence (reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd ed. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright). 3D image of bladder in "open mode" (micturition). Urge incontinence as a premature activation of an otherwise normal micturition reflex: loose suspensory ligaments (PUL, USL), are unable to suspend the vagina adequately, so it becomes loose (wavy lines). The muscles which insert into the loose ligaments "L" lengthen; their contractile force weakens (wavy arrows); they cannot stretch the vagina sufficiently to support the stretch receptors "N"; "N" fire off increased afferent (emptying) impulses at a low bladder volume and this is perceived by the cortex as urgency. If the afferents are sufficient to activate the micturition reflex, the posterior urethral wall is opened out (funnels); the efferent impulses from the cortex activate the detrusor to contract, and the patient may uncontrollably lose urine (urge incontinence). The wavy arrows emphasize weakened muscle forces. PUL, pubourethral ligament; USL, uterosacral ligament; CL, cardinal ligament.

sling prevents bladder base traction by gravity "G" and so prevents the stretch receptors "N" firing off to cause urge. However, the peripheral musculoelastic control mechanism cannot counter bladder instability caused by damage to the cortical inhibitory circuits, say by MS, or excessive stimulation of "N" by tumor or inflammation.

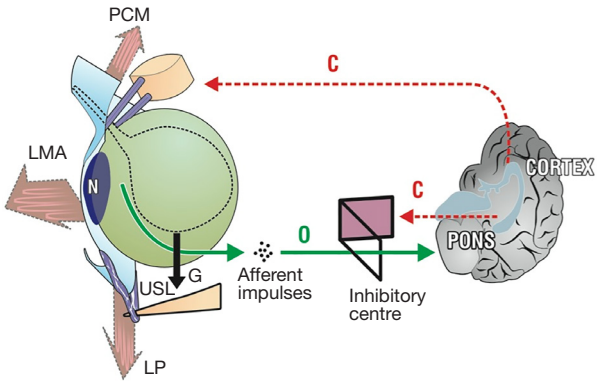

Figure 4 The anatomy of nocturia (reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd ed. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright). Pelvic muscles (large arrows) are relaxed. As the bladder (broken outline) fills, it is distended downwards by gravity "G". If the uterosacral ligaments "USL" are weak, it continues to descend until the stretch receptors "N" are stimulated, activating the micturition reflex once the cortical closure reflex "C" has been overcome. O, opening; PCM, pubococcygeus muscle; LP, levator plate; LMA, conjoint longitudinal muscle of the anus.

These conditions need to be excluded before proceeding to surgical correction.

The anatomy of continence and stress urinary incontinence (SUI)

The mechanics of continence and SUI are described in *Figure 5*. Direct proof of the importance of a firm midurethral anchoring point is control of SUI with hemostat support at the midurethra, evident in Video S5.

Ultrasound proofs of weak PUL as cause of SUI

Figure 6 is a transperineal ultrasound of a woman with SUI. At rest (*Figure 6A*) the urethra is closed. In *Figure 6B* (strain) the PUL extends. The anterior and posterior vaginal walls (a&p) are stretched backwards/downwards to open out the posterior wall of the urethra along its length. In *Figure 6C* (midurethral anchor) a hemostat (white arrow) inserted behind the symphysis as in Video S2, mechanically supports

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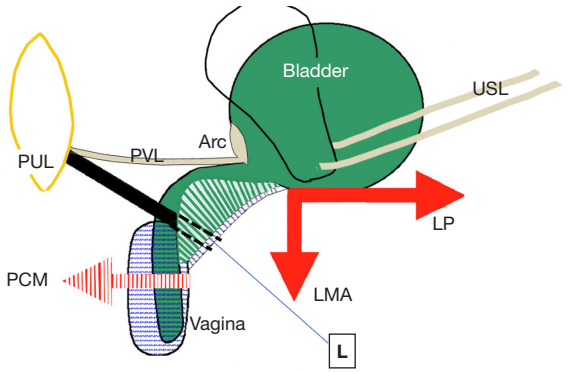

Figure 5 Schematic anatomy of continence and stress incontinence (reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd ed. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright). Normal closure: a normal PUL does not lengthen. The "PCM" contracts forward to close the distal urethra. The "LP" stretches the vagina, PVL, and proximal urethra backwards; the "LMA" rotates the bladder base around the arc of Gilvernet to close (kink) the bladder neck. Stress incontinence: a weak PUL lengthens on effort "L". The LP/LMA contract against the PUL to pull open the posterior urethral wall. Urine is lost on effort. The geometry of stress incontinence and normal micturition is almost identical; the urethra is pulled open (funnels) by posterior muscle forces (*Figure 6*). PUL, pubourethral ligament; PVL, pubovesical ligament; USL, uterosacral ligament; LP, levator plate; LMA, conjoint longitudinal muscle of the anus; PCM, pubococcygeus muscle.

the weakened PUL, and temporarily restores urethral closure along its length and normal urethrovesical geometry on straining. The anterior and posterior vaginal walls (a&p) are tensioned; the urethra is closed at the bladder neck and distally.

Explaining surgical and non-surgical cure/improvement of OAB

"*The reason behind female urinary incontinence has to be looked for outside the bladder, that is, in the structures supporting the urethra and bladder neck, specifically, ligaments, pelvic floor muscles and vagina*" (1). Ligaments are the structures most vulnerable to deterioration because of changes in their main structural component, collagen, caused by labour, delivery and age (1). Since the early 1990s, surgeons who follow the ITP have been reporting high cure rates for SUI, OAB, urinary retention and chronic pelvic pain, by repair of the suspensory ligaments of the pelvis, principally the PUL and USL (9-18).

Conclusions

Bladder control is binary, with cortical and peripheral components. A small change in definition from "overactive" to "overactivated" is consistent with this concept, retains the acronym "OAB", and opens the door to a massive increase

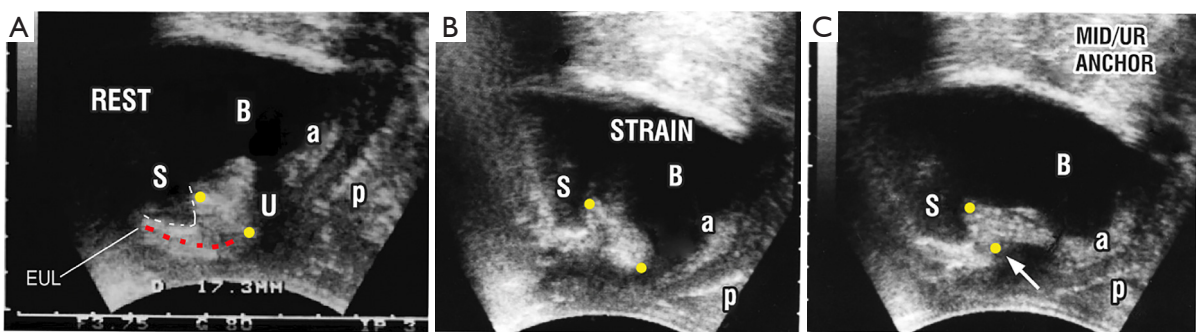

Figure 6 Transperineal ultrasound of a woman with SUI (reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd ed. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright). At rest (A), during straining (B) and right frame, with a hemostat supporting the pubourethral ligament at midurethra. Note restoration of anatomy by midurethral anchoring by the hemostat test (C, white arrow). The two yellow circles mark the length of the pubourethral ligament extending from behind the lower border of the symphysis to the midurethra; red broken lines mark the distal urethra. S, symphysis; U, urethra; B, bladder; a&p, the anterior and posterior walls of the vagina; EUL, external urethral ligament; SUI, stress urinary incontinence.

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in research endeavours.

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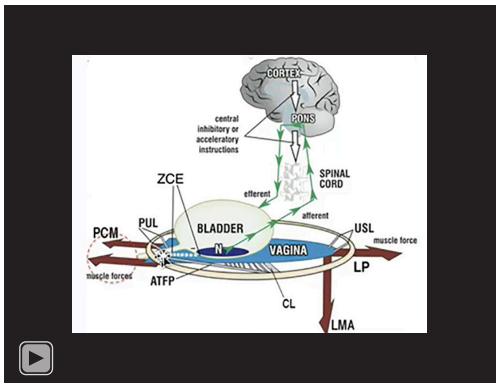
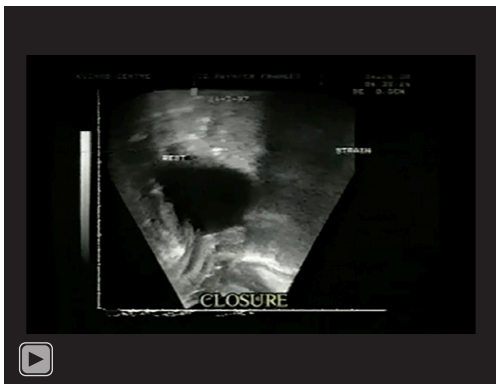
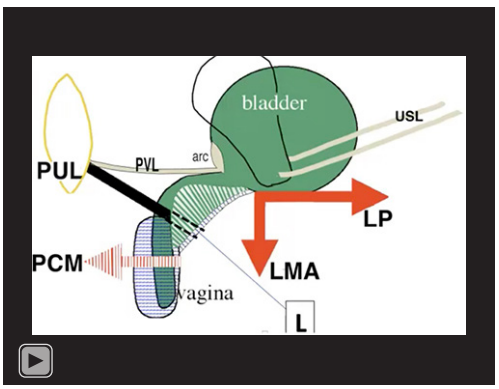
Supplementary



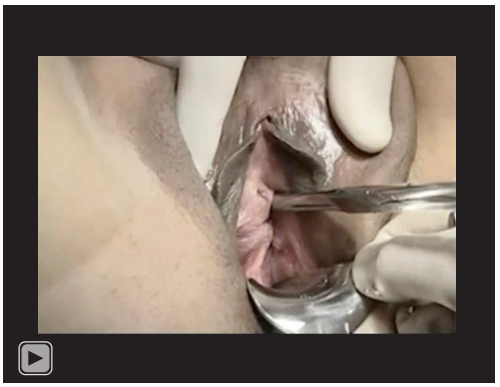
Video S1 Video abstract.



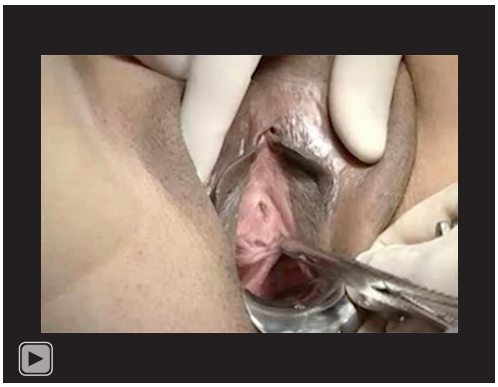
Video S2 Ultrasound demonstrates the three oppositely acting reflex muscle forces which close the urethra distally and at bladder neck.



Video S3 Video X-ray micturition. Note how the downward angulation of the levator plate opens out the posterior urethral wall. Also, how the detrusor spasms to empty.



Video S4 How stretching a glove (the vagina and urothelial stretch receptors) by opposite forces reduces the weight impacting on the glove (vagina, and urothelial stretch receptors).



Video S5 Control of urine loss on coughing by mechanical support of the pubourethral ligament with a hemostat. By permission from Professor Paolo Palma.

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