# Letter to the editor

Dear Editors,
I write to comment on the Levin hypothesis

1. Levin M The role of detrusor rigidity in the lower urinary tract dysfunction. Hypothesis Pelviperineology 2018; 37: 70-73

As a pelvic floor surgeon with a major interest in bladder and anorectal function, I found Dr Levin's hypothesis[1] most interesting. His hypothesis starts from the known, symptoms of bladder emptying and urgency increase with age. He provides evidence of fibrosis within the detrusor smooth muscles then proceeds seamlessly to elaborate his hypothesis. The hypothesis is an admirable example of deductive logic. However, it fails on one major point, it cannot explain reported surgical cures of overactive 'OAB'[2] and underactive bladder 'UAB'[3-4]. Furthermore it fails to mention the external mechanisms which open and close the urethra, figs1&2. The reason why UAB has a slow flow (at least in females) can be attributed to the effect of this external mechanism on the urethral tube which, by narrowing or expanding the urethra, exponentially affects urine flow[4] figs 3&4, in an inverse relationship with the 4th power of the radius, Poiseuille's Law[5].
The bladder is a receptacle with only one role, to empty. Electrical transmission is smooth muscle to smooth muscle[6] and the bladder empties by spasm. This is evident on viewing any video of micturition.
Control of evacuation is by muscle forces acting at the urethral outlet tube, in turn controlled by closure or micturition reflexes[7]. The diameter of the urethra is varied by external muscle forces: it is narrowed for closure, or expanded just prior to evacuation, figs 1,2, 4. These muscles forces are external to the urethra. They rapidly alter the urethral diameter and therefore, the resistance to flow, exponentially[5]. Almost all bladder dysfunctions can be explained by the effect of the closure or micturition reflexes

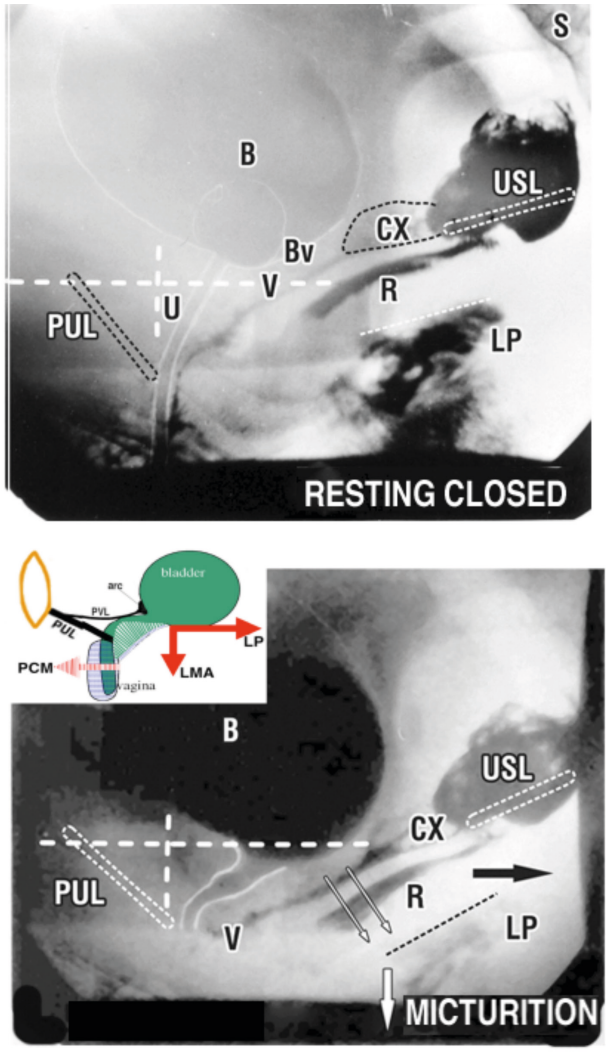


Fig 1 Anatomical changes in bladder when the micturition reflex is dominant
*Upper XR - Resting closed* Directional slow twitch muscle fibres maintain urethral closure distally and at bladder neck. Bv=ligamentous attachment of bladder base to anterior vaginal wall (V). R=rectum; LP=levator plate; CX=cervix; USL=uterosacral ligament; PUL=pubourethral ligament' U=urethra
*Lower XR - Micturition* The forward PCM vector (insert) relaxes. The backward LP/LMA vectors stretch the vagina backwards and downwards against USL to open out the posterior urethral wall. The pubovesical ligament attachment to the arc of Gilvernet (PVL, insert figure) prevents the anterior bladder wall prolapsing into the outflow tract. Note downward angulation of LP.

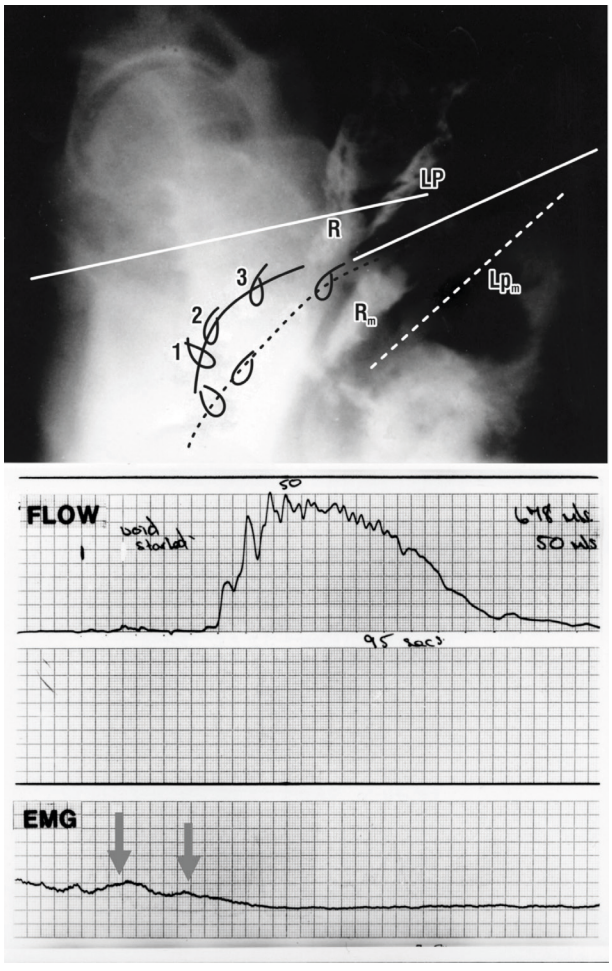


Fig. 2 Normal micturition in the female
*Upper image* Micturition xray (broken lines) superimposed on resting xray. Subscript "m" denotes position of organ during micturition. R=rectum; LP=levator plate. Clips have been applied to the midurethra '1', bladder neck '2' and bladder base '3'. Note downward/backward displacement of the clips indicating stretching open of the posterior urethral wall.
*Lower image* EMG activity (arrows) commences prior to the start of urine flow.

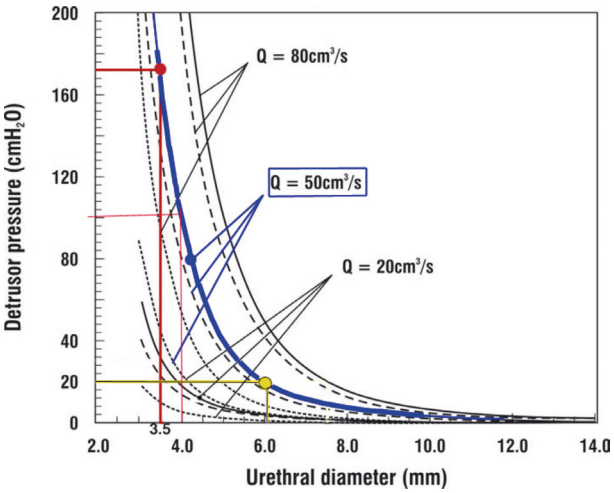


Fig 3 Exponential nature of urine flow is related to urethral diameter
For a flow rate of 50ml/sec (thick blue line), opening the urethral diameter from 3.5mm to 4 mm reduces the head of pressure required by detrusor to expel the urine from 172 cm H2O to 100 cm H2O). Expanding to 6mm (yellow lines), reduces the head of pressure to 20cm H2O. The blue line is the total urethral resistance to flow. The broken lines are dynamic and frictional flow components.

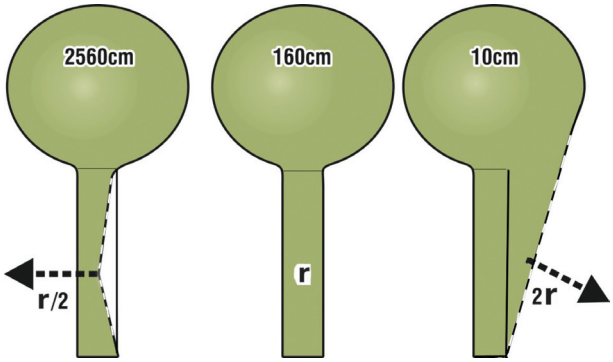


Fig 4 How external muscle forces alter urethral tube diameter to facilitate closure or evacuation
The arrows indicate how muscle forces can externally alter urethral diameter. The 160cm pressure needed to empty through resting urethral radius 'r' is a nominal figure. Pressure needed to empty following active closure to (r/2) or opening to (2r) is calculated by applying Poiseuille's Law, whereby internal resistance to flow varies inversely by the 4th power of the radius. For example, if the urethral tube can be stretched open to twice the radius (2r) during micturition, the pressure needed to evacuate the bladder falls by a factor of 16 (2x2x2x2), from 160cm to 10cm H2O. Broken lines indicate change in diameter by muscle forces (arrows).

on the urethral tube and its effect on urine flow[7]. The posterior opening forces (arrows, fig1), contract against the uterosacral ligaments (USL). If USLs are loose, the posterior muscle forces (arrows) weaken; the vagina cannot be stretched to support the bladder base stretch receptors. These may fire off prematurely to activate the micturition reflex to cause urge and urine loss. Nor does it take much urethral narrowing to cause emptying difficulties. The margin is very small. For example, inability of the posterior vectors to open the urethra because of lax USLs from 3.5 to 4mm (a very small amount), fig 3, almost doubles the head of pressure required by bladder to expel the urine, from 100cm H2O to 172 cm H2O. Repair of USLs has been shown to cure both OAB[1] and both OAB and UAB[2-3]. I agree with Dr Levin about age-related causation of OAB and UAB. However, the cause from the perspective of the Integral Theory[7] is collagen loss in the uterosacral ligaments, which is repaired by the new collagen generated by the posterior sling tapes[2-4].

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