Original article

A cystocele may compensate for latent stress incontinence by artificially restoring the urethral and bladder neck closure mechanisms - a critical biomechanical perspective

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Abstract: Existing hypotheses for latent stress incontinence involve kinking of the urethra by the cystocele or compression by an organ. The biomechanical hypothesis is based on dynamic transperineal ultrasound observations which showed downward stretching of the anterior vaginal wall. This was shown to stretch and narrow the urethra, restore the urethrovesical angle and tension the suburethral vagina. It is hypothesized that the key factor in restoration of continence by the extruded cystocele was the exponential increase in frictional resistance to flow caused by the urethral narrowing, inversely by the 4thpower of the decrease in radius, according to Poiseuille's Law, but also the increased tension of the urethral walls (Laplace's Law) and restoration of the distal urethral closure mechanism by the now tensioned suburethral vagina.

Keywords: Latent stress incontinence; Cystocele; Biomechanics.

INTRODUCTION

Recent studies performing concomitant stress incontinence (SI) and pelvic organ prolapse (POP) surgery have highlighted the condition of latent stress incontinence.[1] Stress incontinence is said to co-occur in up to 80% of patients with POP.[1] In a randomized trial of 600 patients in women assigned to concomitant Burch colposuspension or to no Burch colposuspension (control) along with sacrocolpopexy surgery for prolapse, after surgery, women in the control group were more likely to report bothersome symptoms of stress incontinence at 3 months than those in the Burch group who had stress incontinence (24.5 percent vs. 6.1 percent).[1]

The precise mechanism for concealment of occult stress incontinence by a vaginal prolapse is disputed. One theory is the urethral "kinking" hypothesis:[2] uterovaginal prolapse was able to prevent stress incontinence by actually obstructing ("kinking") the urethra.

Others[3] favour the traditional concept of direct compression of the urethra by the pelvic organs.

This work presents a biomechanical hypothesis based on dynamic transperineal ultrasound studies.

METHODS

Under transperineal ultrasound control, 5 patients examined in the semirecumbent sitting position were asked to strain and cough before and after using a sponge holding forceps to manually support the cystocele.[4]

Curvilinear sector scanner probes 3.75 MHz and 5.5 MHz (Capasee, Toshiba) were sited at the vaginal introitus, taking care to avoid any undue pressure on the vulva.

All testing was performed in the sitting semi-recumbent position. The patients had a comfortably full bladder. All patients lost urine on coughing with the cystocele reduced, fig1, but all were dry on coughing with the cystocele extruded. Photographs were taken on straining both before and after support of the cystocele. The ultrasound probe was positioned to observe the events at bladder neck before and after the cystocele was reduced.

OBSERVATIONS/RESULTS

As the cystocele began to extrude, the anterior vaginal wall appeared to slide downward, with apparent tensioning of the suburethral vagina and reconstitution of the urethrovesical angle, figure 2, with urethral narrowing. No kinking of the urethral tube was observed. The biomechanical processes hypothesized to restore the anatomy, figure 2 and continence, are described in figure 3.

The biomechanical hypothesis

Viewed from a biomechanical perspective, the urethra is a tube which in the continence phase, must resist the bladder emptying pressure. Figure3 explains how stretching the anterior vaginal wall downwards would also stretch and narrow the proximal urethra. Restoration of continence in the absence of urethral kinking is explained in terms of the urethra being sufficiently narrowed to resist the pressure seeking to empty the bladder. Stretching and narrowing the urethra increases the closure acting on its walls (Laplace's Law). It also increases intracavity frictional resistance to urine flow inversely by the 4th power of the radius[5] (Hagen-Poisseuille's Law). e.g., halving the radius would increase the resistance by a factor of 16.

In addition to these mechanisms, another mechanism is hypothesized, restoration of the hammock closure mechanism: stretching and tensioning the suburethral vagina enables the forward vector 'PCM', figure 3, to close the urethra from behind.[6]

Lax Connective Tissue and Diminution of the Force of Muscle Contraction

Even at rest, sufficient tension is required in the vaginal membrane to close the urethra and support the stretch receptors (N) (Figure 4) to prevent their 'firing off 'prematurely. Vaginal tension is maintained by tissue elasticity and slow-twitch pelvic muscle contraction. Connective tissue laxity 'L' in the pubourethral ligaments (PUL)(lower part, Figure 4) may cause dysfunction. As the 'trampoline analogy' shows, laxity in the ligaments (springs) or vagina (trampoline membrane) will not permit tensioning of the vagina by the muscles. The muscle forces (arrows) can only be transmitted to open or close the urethra when point 'SE' has been reached on the stress-extension curve (upper part, figure 4). With damaged connective tissue, the muscle forces (arrows) must first 'take up the slack' (L). To accommodate this, at SEL, has to be reached on the stress extension curve. As a muscle can only contract over a finite length (E), SEL cannot be reached, the muscle forces cannot close the urethra, and stress incontinence occurs. A lax vaginal

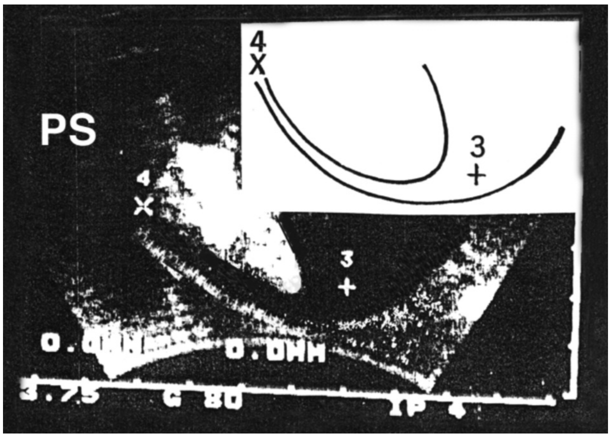

Figure 1. – Cystocele reduced Diagrammatic copy of transperineal ultrasound, patient semirecumbent. Patient leaked urine on coughing. Bladder neck funnelled on straining in a typical USI pattern: rotation of bladder with funnelling of bladder neck.

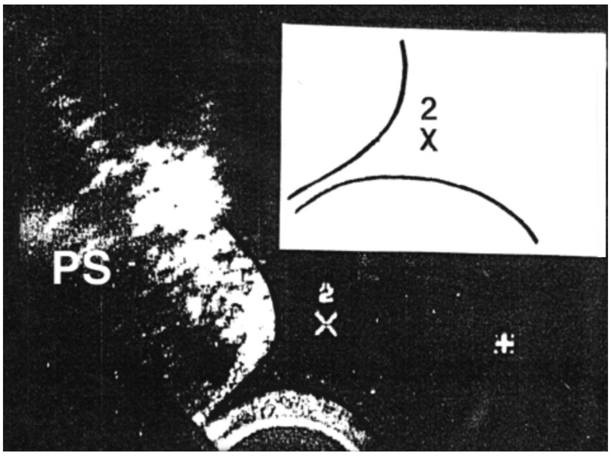

Figure 2. – Cystocele support removed. Diagrammatic copy of transperineal ultrasound, same patient as in fig1. Patient straining, cystocele descending. As the anterior wall of the cystocele descended during straining (curved arrow), the urethrovesical angle changed from funnelled to normal. Bladder neck showed closure on straining. No leakage on coughing was observed.

membrane may not adequately support the stretch receptors (N), so these may prematurely activate the micturition reflex to cause urgency.

CONCLUSIONS

Latent SI is a biomechanical consequence of cystocle.

Contributions. All authors contributed equally to the planning and writing of this paper.

Conflicts. There are no conflicts by any of the authors

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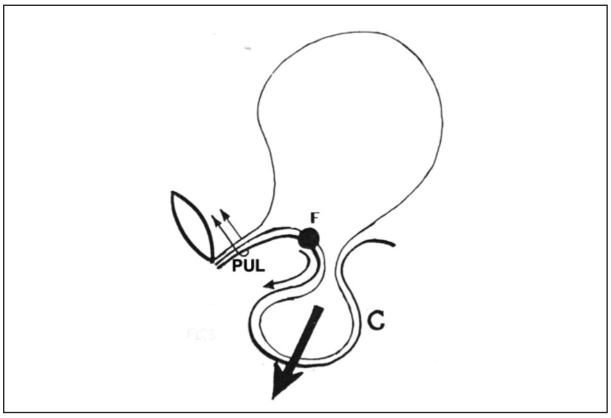

Figure 3. – Simplified Mechanism for closure in patients with latent incontinence. 'F' represents the detachment point of pubocervical fascia to anterior cervical ring. With F detached, the cystocele 'C' is free to descend (large arrow); it stretches the anterior vaginal wall and proximal urethra backwards and downwards (curved arrow). This action also stretches the loose pubourethral ligaments 'PUL', so 'C' now rotates the proximal urethra around PUL to close it. The PDF forward vectors at midurethra (twin arrows) contract to close the distal urethra.

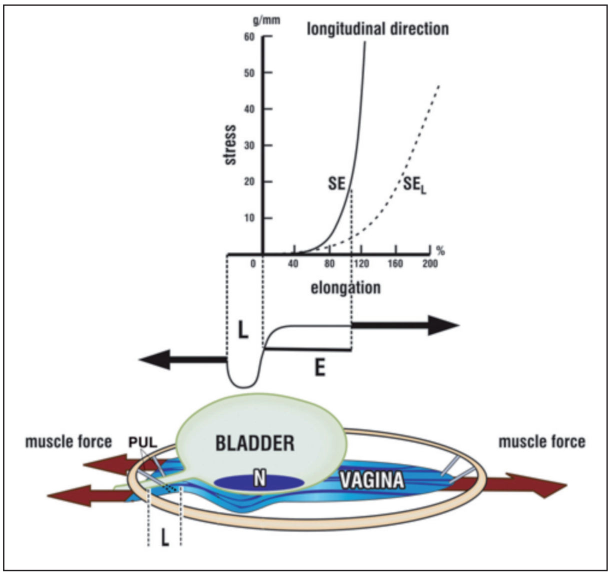

Figure 4. – Laxity in the pubourethral ligament (PUL) is indicated by broken lines 'L'. A muscle can contract only along a finite length 'E'. Some of the contraction is spent in stretching the laxity 'L', so at maximum contraction 'E', there is insufficient force remaining to reach SEL which is required to close the urethra. As the cystocele descends, it sretches out 'L", so that the muscles can now contract normally 'E' to close the urethra.
(After Yamada 1970)

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