

# Testing the logical consistency of the Integral Theory System in a non-selected group of original articles from the November 2012 International Urogynecology Journal

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**Abstract: Aim:** To test the concepts and predictions of Integral Theory System (ITS), that pelvic prolapse and symptoms are caused by laxity in vagina / suspensory ligaments. **Methods:** A Popper's deductive method was applied. The data from published papers in the November 2012 IUJ was tested against concepts and predictions of the ITS. If these concepts and predictions could not explain that data, the ITS was considered invalid. Only descriptive papers were excluded. **Results:** Of 13 papers analysed, 12 were deemed logically consistent with ITS. Overactive Bladder (OAB) and low urethral pressure papers were attributed to loose ligament weakening of musculoelastic mechanisms, diabetic urge symptoms to greater ligament damage by macrosomia; pregnancy effects on hiatal measurements and symptoms by relaxin on collagen crossbonding; symptom improvement with vit D with positive effect on collagen metabolism and ligament strength. The ITS could not explain longer-term improvement of urge symptoms by posterior tibial nerve stimulation. **Conclusion:** There was strong logical consistency between ITS and 12/13 IUJ November 2012 papers.

**Key words:** Integral Theory System; Urinary Incontinence; Nocturia; Pelvic Organ Prolapse; Chronic Pelvic Pain; Collagen; Relaxin.

## INTRODUCTION

The Integral Theory System "ITS" is a holistic, self-contained anatomical system for diagnosis and management of pelvic organ prolapse (POP) and symptoms. It based on the Integral Theory<sup>1</sup> which in essence states that "pelvic floor symptoms and prolapse, are mainly caused by laxity in the vagina or its supporting ligaments, a result of altered collagen/elastin" (Figure 1). Management is based solely on repairing the connective tissue damage in these structures. "Repair the structure and you will repair the function". The relationship between the various prolapses, symptoms and damaged connective tissue structures in 3 zones of the vagina is summarized in figure 1. Overactive bladder and detrusor overactivity are considered to be secondary manifestations of a normal but prematurely activated micturition reflex,<sup>2</sup> in turn, caused by the same laxities.

## THE DEDUCTIVE METHOD OF KARL POPPER

According to Karl Popper, the most eminent philosopher of science of the 20<sup>th</sup> century, the key test for validity of any theory or system is that it must be able to deductively explain results from all experiments in the field in a logically consistent way. Deduction proceeds from the general to the particular.<sup>3</sup> For example, with reference to figure 1, a patient with urinary stress incontinence (USI) will have lax pubourethral ligaments (PUL). However, figure 1 indicates that urge incontinence may also be caused by PUL laxity, cystocele or a uterine prolapse. Furthermore, the ITS predicts that reinforcement of PUL with a midurethral sling may cure both stress and urge symptoms, if the urge is caused by PUL. Again with reference to figure 1, if after say a TVT, urge persists, the ITS predicts it may be corrected by repair of a cystocele or a uterine prolapse.

Fundamental to Popper's criteria, a theory can never be finally proven. It can only be taken to another stage of proof. However, it can be disapproved by one validated example. If the theory states that all swans are white and one sees a black swan, then the theory is immediately invalidated.

Our aim was to apply this acid test, Popper's deductive method, to the Integral System.

## METHODS

The concepts and predictions of the ITS were tested against all the papers published in the November 2012 issue of the International Urogynecology Journal (IUJ). To avoid bias, there was no prior examination of that issue's contents.

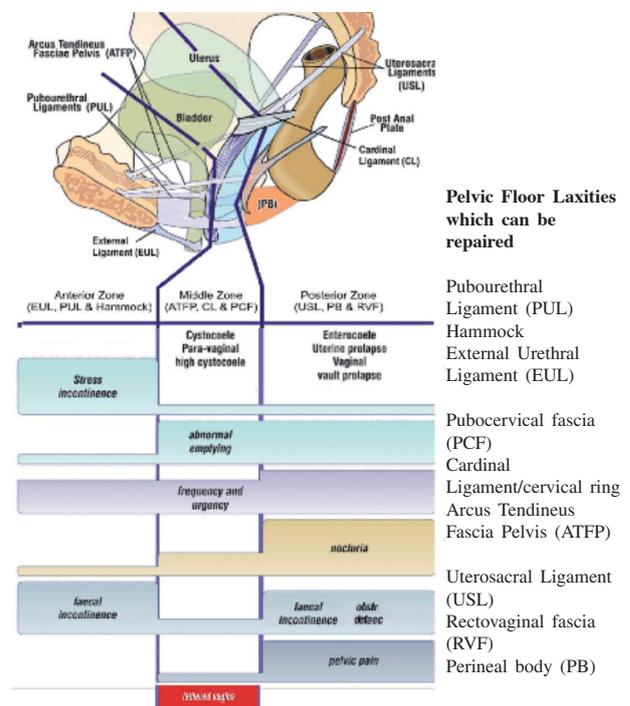


Figure 1. – The diagnostic algorithm uses symptoms to predict zone of damage. Where symptoms such as urgency occur across more than one zone, symptom grouping is used to make the diagnosis. The supporting ligaments/structures naturally divide the vagina into 3 zones: **Anterior:** External meatus to bladder neck, **Middle:** Bladder neck to cervix, **Posterior:** Cervix to perineal body. The height of the bar indicates probability of the symptom originating from damaged structures in that zone. Note that the uterus is supported partly by cardinal ligament (middle zone) and uterosacral ligament (posterior zone).

**Inclusion criteria.** We non-selectively analysed, without exception, all the papers in the November 2012 volume of IUJ to test whether the Integral System's statements and predictions were logically consistent with their data. To save space, except for key articles, all references in the text to the Integral Theory System (ITS) will refer to chapters and pages in the textbook "The Female Pelvic Floor", 3rd Ed. 2010, PEP Petros, Springer, Heidelberg<sup>4</sup> which gives a full account of the ITS ([www.integraltheory.org](http://www.integraltheory.org)).

**Exclusion criteria.** Only descriptive papers. These are listed at the end of the paper.

**How Popper's method was applied.** The key message of the IUJ papers was summarized and compared, mainly with the ITS concepts as summarized in figure 1 and published in the 3rd edition of the «Female Pelvic Floor», Petros PEP Springer Heidelberg, but also specific original papers.

#### ANALYSIS OF NOVEMBER 2012 PAPERS

The numerical order of the 13 papers tested differs to the presentation in IUJ November 2012. The 13 papers are quoted fully at the beginning of each analysis and seriatim at the end of the references section.

**Study No 1: Diamond P, Hassonah S, Alarab M, Lovatsis D, Drutz HP. The prevalence of detrusor overactivity amongst patients with symptoms of overactive bladder: a retrospective cohort study. Int Urogynecol J (2012) 23:1577-1580.**

The aim of the authors was to determine what proportion of patients presenting for urogynecologic assessment with symptoms of OAB have demonstrable DO. Of 160 patients, 93 had symptoms of frequency, nocturia and urgency (FNU); excluded from the study were 50 patients with prolapse at or beyond the hymen, and 25 with elevated *post-void residual* (PVR>100 ml).

**Preliminary analysis.** With reference to fig1, the FNU symptoms from the Diamond study were most likely caused by laxity in the posterior zone (uterosacral ligaments) and the elevated PVR from a cystocele or uterosacral ligament laxity.

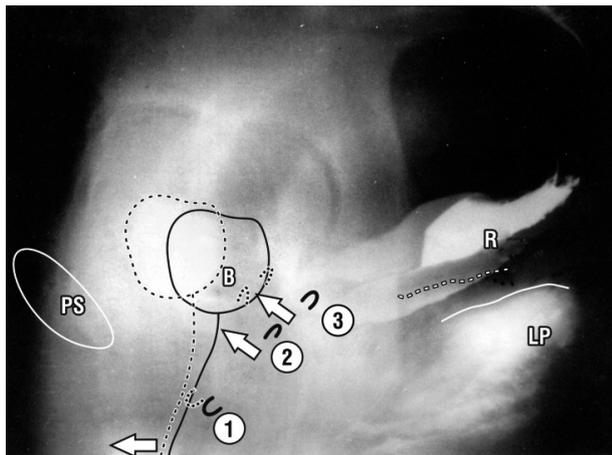


Figure 2. – Squeezing xray superimposed on resting. Rest = unbroken lines; Squeeze = broken lines. Vascular clips at midurethra "1". Bladder neck "2" and bladder base "3". Radio-opaque dye in Foley balloon, vagina, rectum and levator plate. The muscle movements during 'squeezing' are upwards and forwards. Note how levator plate (LP) and rectum (R) are lifted upwards and forwards also. The only muscle which can do this is puborectalis, which lies below LP and behind R. B = Foley balloon. Note the difference with the movements in figure 3. Because 'squeezing', the basic Kegel exercise is not the natural mechanism, it must be learnt.

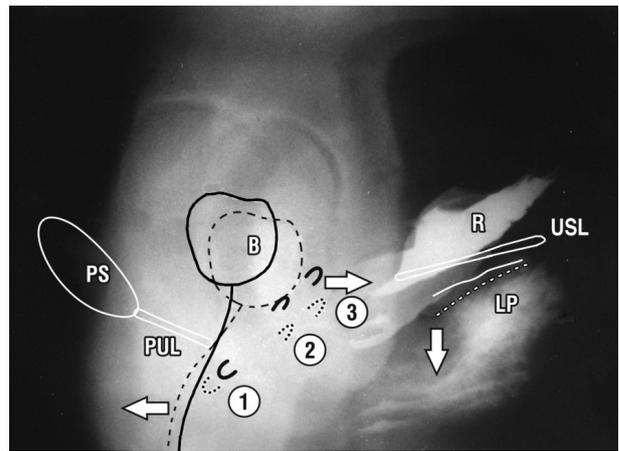


Figure 3. – Same patient and labelling as figure 2. Straining x-ray superimposed on resting. Reflex muscle movements during coughing and straining. Note how the directional movements, forwards (mid urethra '1'), backwards (bladder neck '2' and bladder base '3') all pull against PUL (pubourethral ligaments) and how the downward movements appear to be activated by downward angulation of levator plate, which pulls against (USL) uterosacral ligaments.  
Rest = unbroken lines; Strain = broken lines

Only 11 patients had DO. Diamond et al. concluded that it was mandatory to perform an assessment for urogenital atrophy, pelvic organ prolapse, and incomplete bladder emptying before initiating an anticholinergic medication, as these were common causes of OAB symptoms, frequency, urgency and nocturia. They quoted another study,<sup>5</sup> where 74.6% of patients had prolapse associated with OAB symptoms and yet another where treatment of prolapse with either pessary or surgery resulted in a significant improvement in OAB symptoms.<sup>6</sup>

**Analysis.** The data from Diamond and the other studies<sup>5,6</sup> are consistent with the Integral System's statements as summarized in the algorithm, figure 1: prolapse, urgency (OAB), nocturia and abnormal emptying are mainly caused by prolapse, or uterosacral ligament laxity. The Boer et al reference,<sup>6</sup> as quoted "where treatment of prolapse with either pessary or surgery resulted in a significant improvement in OAB symptoms", is an almost exact paraphrase of the Integral System's prediction, "Repair the structure and you will repair the function", in this case, addressing USL laxity with pessary or surgery<sup>6</sup> would also cure the OAB symptoms.

The ITS concept is that DO, urgency and nocturia are all differing expressions of a prematurely activated micturition reflex (Ch2 pp 60,61, Ch6 pp 234-267).<sup>4</sup> Urodynamically controlled data from 1997<sup>7</sup> indicate that Diamond et al. could have cured >80% of their patients with both prolapse and OAB symptoms, urge, frequency, nocturia, with a simple, overnight stay, posterior sling.

**Study No 2: Fitz FF, Resende AP, Stupp L et al. Biofeedback for the treatment of female pelvic floor muscle dysfunction: a systematic review and meta-analysis. Int Urogynecol J (2012) 23:1495-1516.**

This systematic review was beset with huge methodological issues. Nevertheless it was able to suggest that Pelvic Floor Muscle training (PFMT) with Biofeedback (BF) is not more effective than other conservative treatments for female PFM dysfunction.

**Analysis:** Only stress or mixed incontinence symptoms were addressed. The pelvic floor exercises (PFE) as described were based on Kegel exercises, "squeezing", a vol-

untary muscle contraction which pulls the bladder base, vagina and anorectum upwards and forwards (Ch2 pp33-38)<sup>4</sup>. As a learnt movement, there is no reason why biofeedback which assists timing but not quantum of contraction should enhance results. “Squeezing” (Figure 2) is a voluntary movement which needs to be learnt. It pulls all the organs forwards and upwards. It is quite different to the reflex three-directional movements which occur during coughing and straining (Figure 3): forward movement of the distal urethra and vagina and backwards/downwards movement of bladder base and proximal vagina around a competent pubourethral ligament, (Ch2 pp33-38)<sup>4</sup>. According to ITS, a more physiological method for PFE is based on a squatting regime, as this reinforces all three-directional movements, and therefore the ligaments against which they contract, (Ch5 pp219-225)<sup>4</sup>. Pad test based results indicate up to 60% improvement in 60% of patients with symptoms of USI nocturia, urgency, emptying and pelvic pain (Ch5 pp219-225)<sup>4</sup>.

**Study No 3: Editorial. Abdel-Fattah M, Rizk DE. Diabetes mellitus and female urinary incontinence: a time for change. Int Urogynecol J (2012) 23:1481-1482.**

There is a 20% increase in odds for urge, mainly urge incontinence, not associated with urinary stress incontinence (USI). Gestational diabetes may lead to macrosomia, prolonged second stage of labor, and the increased risk of instrumental delivery on the pelvic floor.

**Analysis:** The ITS explains the urgency in terms of a greater odds of damage to the uterosacral ligaments by the macrosomia than a normal sized fetus. Even in the normal patients the diameter of the fetal head is 9.4cm flexed (11.2 cm deflexed) has to traverse a pelvic inlet of only 12-13 cm. Though there is significant loosening of collagen bonds with Relaxin hormone, a macrosomic fetus will inevitable stretch all the suspensory ligaments and perineal body (Figure 1) more than a smaller fetus, (Ch2 pp48-58). Laxity in the middle and posterior groups of ligaments, may cause urgency not associated with USI. With reference to figure 1, the ITS predicts that the authors would also find a greater incidence of cystocele and uterine prolapse, the latter associated with nocturia, frequency, emptying, pelvic pain alone.

**Study No 4: Zhang YX, Xu HN, Xia ZJ and Wu B. Analysis of clinical interventional strategy for women with urinary incontinence complicated with diabetes mellitus. Int Urogynecol J (2012) 23:1572-73.**

The authors compared diabetic with non-diabetic patients and demonstrated a lower cure rate with the diabetic group. They hypothesized neurological damage or damage to the muscles.

**Analysis:** The comments on neurological damage are not inconsistent with the ITS which stipulates normal efferent and afferent nerve channels to activate organs and muscles. However, we add comments made by Abdel-Fattah and Rizk on the additional distensory damaging effects of macrosomia on the pelvic connective tissues (CT) (Ch2 pp48-58)<sup>4</sup>, but not muscle. Whether it is muscle damage or CT damage causing symptoms was directly tested in a blinded study on 50 patients the vast majority of whom had histologically proven myopathy. 89% were cured immediately with a midurethral sling,<sup>8</sup> indicating that connective tissue damage, not muscle damage was the key element in the causation of USI.

**Study No 5: Shek KL, Kruger J, Dietz HP. The effect of pregnancy on hiatal dimensions and urethral mobility: an observational study. Int Urogynecol J (2012) 23:1561-1567.**

Comparison of 3rd trimester data of the pregnant cohort with that of the non-pregnant nullipara revealed a 27% and 41% increase in hiatal area at rest and on Valsalva and an increase in segmental urethral mobility by 64% to 91% in late pregnancy. Similar results were found in patients undergoing Caesarian Section (CS).

**Analysis:** Shek and Francis’s<sup>10</sup> observations are consistent with the explanations of the ITS as regards the effects of relaxin on collagen which weakens the inter and intramolecular bonds, thereby causing prolapse and incontinence symptoms in pregnancy (Ch2 p44-46)<sup>4</sup>. Rechberger,<sup>9</sup> observed that collagen loses 95% of its strength just before delivery. This depolymerization of collagen would loosen the collagens bonds between organs and hiatus, and cause laxity in the pubourethral ligament (Ch2 p55)<sup>4</sup>, (Figure 4), allowing distension of the hiatus, rotation of bladder base and USI. The pressure of the fetus on the hiatal structures and vagina in an obstructed labour would extend the collagen structures further, explaining the authors’ statement that these changes are irreversible in patients undergoing CS. Winifred Francis reported onset of bladder symptoms during pregnancy in 1960.<sup>10</sup> This is consistent with depolymerization of collagen by Relaxin production during pregnancy, which increases vastly at 3 months when the placenta takes over productions from the corpus luteum from 3 months onwards (Ch2 44-46)<sup>4</sup>.

**Study No 6: Kapoor DS, Housami F, Swithinbank L, Drake M. Maximum urethral closure pressure in women: normative data and evaluation as a diagnostic test. Int Urogynecol J (2012) 23:1613-1618.**

The authors demonstrated that women with USI and mixed urinary incontinence (MUI) have lower maximum urethral closure pressure (MUCP) than women with detrusor overactivity incontinence (DOI) and continent women in each decade of life. MUCP decreases with age. However, MUCP failed to meet the criteria for a diagnostic test for incontinence.

**Analysis:** With reference to figure 1, urge and DOI may occur in all 3 zones, not just the anterior zone. Therefore there will be many patients with DOI who do not have low MUCP and USI. Continence and urethral pressure were shown to be unrelated in a 1997 prospective surgical study.<sup>7</sup> The cure rate for ISD patients was equivalent to those with normal urethral pressures with the midurethral sling opera-

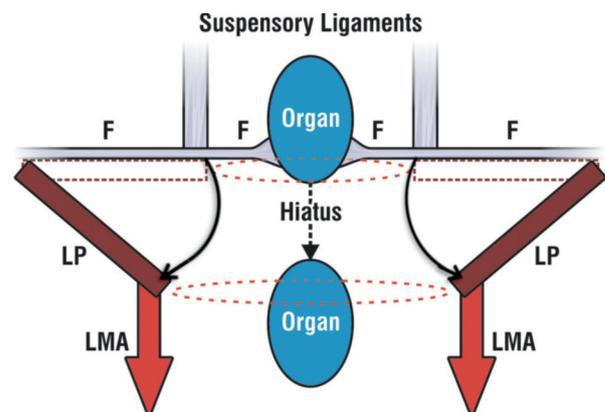


Figure 4. – Schematic coronal view, straining. During straining, the levator plate LP is angulated downwards by the Longitudinal Muscle Ani (LMA) vector, opening out the levator hiatus. If the suspensory ligaments and attached fascia (F) are weakened by Relaxin hormone, the organs are more likely to prolapse through the widened hiatus. As the elastin and collagen weaken beyond a critical mass, the prolapsed position becomes permanent as stated by Shek et al.

tion. Furthermore, cure of USI was effected even though there was no increase in MUCP post-operatively.<sup>7</sup> The ITS specifies a musculoelastic mechanism for urethral closure with distal and proximal components, both of which rely on an intact pubourethral ligament (PUL) (Ch2 pp 30-38)<sup>4</sup>. Connective tissue weakens with age. According to Gordon's Law,<sup>11</sup> laxity in PUL effectively lengthens the three-directional closure muscles, diminishing the urethral closure forces (Ch2 p50)<sup>4</sup>. The elements contributing to urethral pressure are detailed in figure 5 and Ch2 pp59-60. The important biomechanical concept for continence is that narrowing of "a" increases pressure only to the 2<sup>nd</sup> power (pressure = Force/Area), whereas it increases the intra-urethral resistance to the 4<sup>th</sup> power according to Poiseuille's Law (5<sup>th</sup> power for non-laminar flow) (Ch6 pp 227-238)<sup>4</sup>. MUCP is a static measure. Continence is dynamic. Fast-twitch contraction of the three-directional muscle forces instantaneously narrows the urethra, vastly increasing the intra-urethral resistance. This explains how patients with persistent low MUCP (<20cm H2O) post-operatively were cured with a midurethral sling.<sup>7</sup> On this basis, increased urethral resistance, not pressure, is the essential factor in continence control.

**Study No 7: Brazell HD, Claydon CS, Li J, Moore C, Dereska N, Hudson S, Swift S. Does neuromuscular blockade affect the assessment of pelvic organ prolapse? Int Urogynecol J (2012) 23:1599-1603.**

Neuromuscular blockade leads to significantly greater increases in POP-Q examination measurements compared with the office measurements, and this increase is most pronounced apically.

**Analysis:** The ITS is consistent with the explanation of the authors, "The difference noted in patients with general endotracheal anesthesia (GETA) could be explained by neuromuscular blockade causing an artificial relaxation of the levator ani (LA) muscles, which leads to a higher stage of prolapse in the OR." The vagina is densely attached to the rectum which is pulled backwards and downwards by levator plate, even in resting mode (by slow twitch muscle contraction), (Ch2 pp 33-34)<sup>4</sup> and as seen in figures 3 and 6. LA relaxation would allow the apex to prolapse downwards.

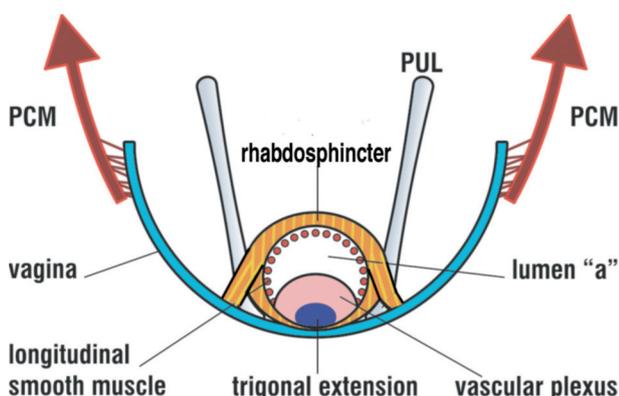


Figure 5. – The components of urethral closure. Perspective: coronal section of vagina at midurethra. The trigonal extension and vascular plexus are known as the "Cresta Urethralis"; arrows = vector closure forces (fast and slow twitch) from contraction of the anterior pubococcygeus muscle (PCM) which stretches the vagina upwards to close the urethral lumen 'a'. Contraction of the rhabdosphincter over the "Cresta Urethralis" provides a water-tight mucosal seal, and is recorded as a pressure rise on effort. Not shown is the contribution to urethral narrowing by the backward/downward vectors LP and LMA (Figure 3).

**Study No 8: Parker-Autry CY, Burgio KL, Richter HE. Vitamin D status: a review with implications for the pelvic floor. Int Urogynecol J (2012) 23:1517-1526.**

The authors of this Review present evidence to support their hypothesis that vitamin D may cause muscle weakness and lead to incontinence.

**Analysis:** The ITS makes no reference to vitamin D. From the ITS perspective, it is the loose collagenous insertion of muscle which weakens muscle contractile force, not damaged muscle per se.<sup>8</sup> It is known that decreased body collagen may be a primary factor in not only connective tissue weakness, but osteoporosis, which is related to vitamin D deficiency. The ITS may be logically consistent with the article, as collagen deficiency would cause lax muscle insertion points, and according to Gordon's Law,<sup>11</sup> muscle weakness.

**Study No 9: Daan MP, Schweitzer KJ, van der Vaart CH. Associations between subjective overactive bladder symptoms and objective parameters on bladder diary and filling cystometry. Int Urogynecol J (2012) 23:1619-1624.**

Of all four OAB symptoms the frequency symptom showed the strongest association with daytime urinary frequency, nocturia with nocturnal frequency, and urgency incontinence with incontinence episodes as measured in the bladder diary. The frequency and nocturia symptoms showed almost equal and strong associations with bladder volumes as measured in the bladder diary and by filling cystometry. The key symptom of the OAB syndrome, urgency, was either not at all or only poorly associated with objective parameters from the bladder diary and filling cystometry.

**Analysis:** The data are consistent with the ITS view of OAB symptoms: they are the cortical perception of a prematurely activated micturition reflex.<sup>2</sup> The stretch receptors at bladder base are supported by a vagina adequately tensioned by three-directional muscle forces; these muscle forces are weakened if the ligaments against which they contract are loose.<sup>11</sup> The lax vagina can no longer support the stretch receptors which fire off at a lower bladder volume, and so the patient empties her bladder more frequently during the day, "frequency" and during the night, "nocturia", (Ch2 pp 42-44; 50-51; 61-63; Ch6 234-245)<sup>4</sup>. In contrast, urgency is considered dependent on the varying sensitivity of the stretch receptors, not bladder volume per se.

**Study No 10. Chin HY, Lin KC, Wang CJ, Chiang CH, Kuo HC. Paraurethral striated muscular structures and pelvic floor muscles contribute to resting urethral closure pressure in rats. Int Urogynecol J (2012) 23:1631-1636.**

The authors injected the striated muscles alongside the urethra with botulinum toxin in an animal model. Their results showed that the paraurethral striated muscular structures contribute to urethral closure pressure in rats. These structures appeared to connect the pelvic floor muscles with urethral striated muscles to maintain adequate urethral closure pressure at rest. Contraction of the paraurethral striated muscles and pelvic floor muscles contributes about 70-80% of MUCP at rest.

**Analysis:** Assuming relevance to the human, the ITS states that the antero-medial part of m.pubococcygeus (PCM) contracts forwards against PUL to close the distal urethra (Ch2 pp32-35); (Figures 3 and 7 of this paper). The lateral parts of PCM which join behind the rectum to insert into the coccyx as "levator plate" pulls the bladder base downwards and backwards to "kink" the proximal urethra (Figure 3 and 7). These muscles are principally of the slow-

twitch variety and in stretching the urethra, they narrow it to close the space below the rhabdosphincter at the midurethra. As Pressure = Force/area, if PCM contraction weakens, the space enlarges and pressure falls.

**Study No 11. Levin PK, Wu JM, Kawasaki A, Weidner AC, Cindy L. Amundsen. The efficacy of posterior tibial nerve stimulation for the treatment of overactive bladder in women: a systematic review. Int Urogynecol J (2012) 23:1591-1597.**

The Review was beset with methodological studies. Three smaller observational studies meeting “good quality” criteria all reported statistically significant improvement in OAB symptoms, but with moderate success rates of 54-70%.

**Analysis:** The ITS explanation for surgical and non-surgical improvement/cure for OAB symptoms is based on strengthening the suspensory ligaments against which three-directional closure forces act. These in turn stretch the vaginal membrane to support the bladder base stretch receptors, which in turn prevents afferent impulses reaching the cortex as OAB symptoms.

The ITS makes no comments on neuromodulation or posterior tibial nerve stimulation (PTNS) or how these methods may or may not work. In fact, given its anatomical basis, the ITS would predict no symptom improvement for urge symptoms much beyond the immediate stimulation period. Based on Popper’s deductive criteria, the ITS predictions are inconsistent and therefore at least partly invalidated.

**Study No 12. Capes T, Stanford EJ, Romanzi L, Foma Y, Moshier E. Comparison of two classification systems for vesicovaginal fistula. Int Urogynecol J (2012) 23:1679-1685.**

The authors compared two classification systems, the Waaldijk and Goh systems which seek to predict success or failure of fistula repair. The Goh classification system was able to predict both successful closure and subsequent continence; patients with type 1 fistulae, sizes b and i, being more likely to have successful closure and continence after treatment.

**Analysis:** The ITS would predict that the Goh classification “ii”- Moderate or severe fibrosis (around fistula and/or vagina) and/or reduced vaginal length and /or bladder capacity would have a greater incidence of incontinence due to the “tethered vagina syndrome” (Ch2,p61; Ch4 179-187).<sup>4</sup> The tethered vagina syndrome was first described in 1990.<sup>1</sup> It is a “motor-type” incontinence characterized by severe scarring or tissue deficit in the bladder neck area of vagina which “tethers” the three-directional forces responsible for distal and proximal urethral closure. The more powerful posterior vectors (Figure 3), forcibly overcome the forward vector to “pull open” the bladder neck. This gives rise to the classical symptom of this condition, sudden massive loss of urine immediately on getting out of bed in the morning. Often there is no significant USI or urgency. On ultrasound, there may be little movement downwards when the patient strains. Cure of this condition consists of augmentation of the bladder neck area of the vagina with some type of skin graft, for example “skin-on Martius graft” to restore the independent three-directional organ movements seen in figure 3.

**Study No 13. RamanahR, Berger MB, Parratte BM, DeLancey JOL. Anatomy and histology of apical support: a literature review concerning cardinal and uterosacral ligaments. Int Urogynecol J (2012) 23:1483-1494.**

In this Review, the authors quote Blaisdell<sup>12</sup> who described uterosacral fibers attached to the fascia covering the

levator ani, coccygeus, and obturator muscles and presacral fascia, Campbell<sup>13</sup> who observed that fibers of the USL and CL were consistently intermingled at the cervical portion, with fibers that extended anteriorly above the internal os and posteriorly onto the proximal third of the vagina, and Butler-Manuel et al.<sup>14</sup> who found that sympathetic nerve fibers along with sensory/nociceptive nerves were relatively more abundant than parasympathetic fibers in the deep USL.

**Analysis:** Blaisdell study confirms why the proximal vagina and uterus are pulled backwards and downwards by the posterior vector forces LP/LMA, an essential part of the ITS explanation for active striated muscle closure (continence) and opening of the urethra (micturition), (Ch2 pp 30-41),<sup>4</sup> and anus (pp 64-75), and figures 6, 7, 8. Campbell’s description of cardinal ligament fibres extending over the anterior cervix and proximal 1/3 of vagina reinforce the description of the pathogenesis of high cystocele by birth-related rupture of these structures, (Ch4 p162-167).<sup>4</sup> Butler-Manuel et al.’s description of sensory/nociceptive nerves in the USL are consistent with the claims that low abdominal/pelvic pain/vulvodynia may be a referred pain from lax USLs, (Ch2 pp 108-110, Ch4 pp 195-202).<sup>4</sup>

## CONCLUSIONS

We found a strong logical consistency between the data from 12/13 of the IUJ November 2012 papers and the Integral System. The Integral System is an anatomical framework based on diagnosis and management of lax ligaments/connective tissue as the principal cause for pelvic organ prolapse and bladder, anorectal and some pain symptoms.

## DESCRIPTIVE STUDIES IUJ Nov 2012 NOT AMENABLE TO ANALYSIS

F. W. Lone, R. Thakar, A. H. Sultan, A. Stankiewicz. Accuracy of assessing Pelvic Organ Prolapse Quantification points using dynamic 2D transperineal ultrasound in women with pelvic organ prolapse. Int Urogynecol J (2012) 23:1555-1560.

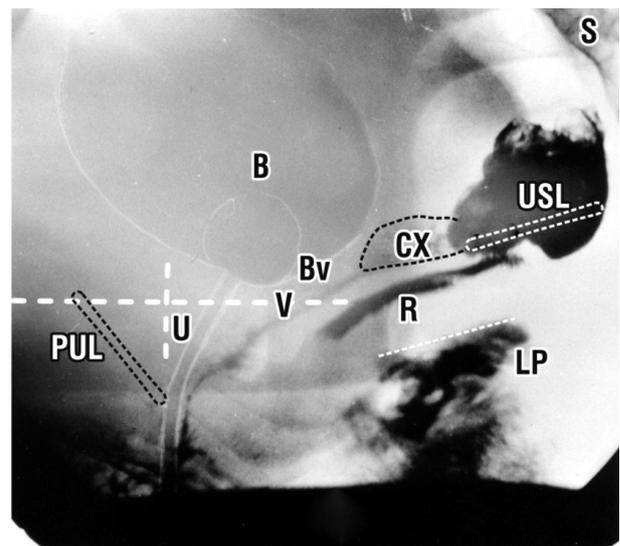


Figure 6. – Sitting lateral x-ray (resting, closed). Even at rest, slow twitch fibres of the posterior vectors (arrows, in figure 7), gently stretch the proximal urethra backwards/downwards. U = urethra; V = vagina; B = bladder; Bv = fascial attachment of bladder base to vagina; CX = cervix; LP = levator plate.

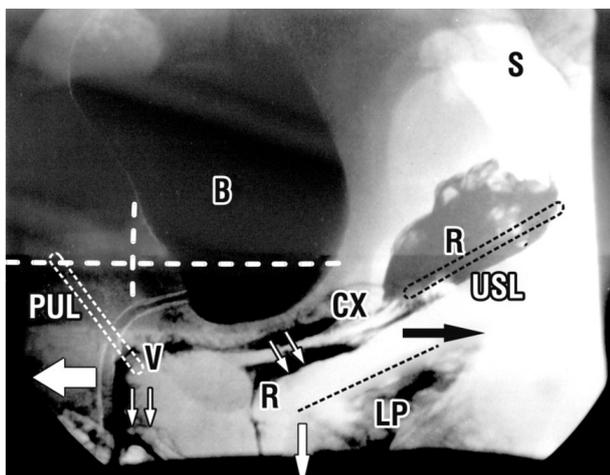


Figure 7. – Same patient and labelling as in figure 6. Urethral closure during effort (coughing or straining). Compared to figure 6, the forward muscle force (arrow) stretches the distal vagina (V) forwards against the pubourethral ligament (PUL) to close the distal urethra, ‘distal urethral closure mechanism’ (Ch2);<sup>4</sup> the backwards and downwards muscle forces (arrows), stretch and angulate the upper vagina and urethra around PUL to ‘kink’ the proximal urethra, ‘bladder neck closure mechanism’ (Ch2).<sup>4</sup> Exactly in position “R”, is the forward contraction of m. puborectalis. This stabilizes the anorectum for the posterior vectors (arrows), to stretch the rectum around “R” to form the anorectal angle and to effect anorectal closure.

Man-Jung Hung, Yi-Ting Chen, Pao-Sheng Shen, Shih-Tien Hsu, Gin-Den Chen, Esther Shih-Chu Ho. Risk factors that affect the treatment of interstitial cystitis using intravesical therapy with a dimethyl sulfoxide cocktail. *Int Urogynecol J* (2012) 23:1533-1539.

MME Lakeman, FM Zijta, J Peringa, AJ Nederveen, J Stoker, JPWR Roovers. Dynamic magnetic resonance imaging to quantify pelvic organ prolapse: reliability of assessment and correlation with clinical findings and pelvic floor symptoms. *Int Urogynecol J* (2012) 23:1547-1554.

L Cardozo, T Hall, J Ryan, CE Bitoun, I Kausar, A Darekar, A Wagg. Safety and efficacy of flexible-dose fesoterodine in British subjects with overactive bladder: insights into factors associated with dose escalation. *Int Urogynecol J* (2012) 23:1581-1590.

S Ginath, AD Garely, JS Luchs, A Shahryarnejad, CK Olivera, S Zhou, CJ Ascher-Walsh, A Condeira, ML Brodman, MD Vardy. Magnetic resonance imaging of abdominal versus vaginal prolapse surgery with mesh. *Int Urogynecol J* (2012) 23:1569-1576.

D Shveiky, AI Sokol, RE Gutman, BI Kudish, CB Iglesias. Patient goal attainment in vaginal prolapse repair with and without mesh. *Int Urogynecol J* (2012) 23:1541-1546.

#### REFERENCES

1. Petros PE & Ulmsten U. An Integral Theory of female urinary incontinence. *Acta Obstetrica et Gynecologica Scandinavica*, 1990; 153: 1-79.
2. Petros PE & Ulmsten U. Bladder instability in women: A premature activation of the micturition reflex. *Neurourology and Urodyn.* 1993; 12: 235-239.
3. Popper K R. Theories, Falsifiability, from” The Logic of Scientific Discovery” (1980), Unwin, Hyman, London, 27-146.
4. Petros PEP, The Female Pelvic Floor Function, Dysfunction and Management According to the Integral Theory, 3d Edition (2010). Ch1 Overview, 2-15; Ch2 Anatomy Function

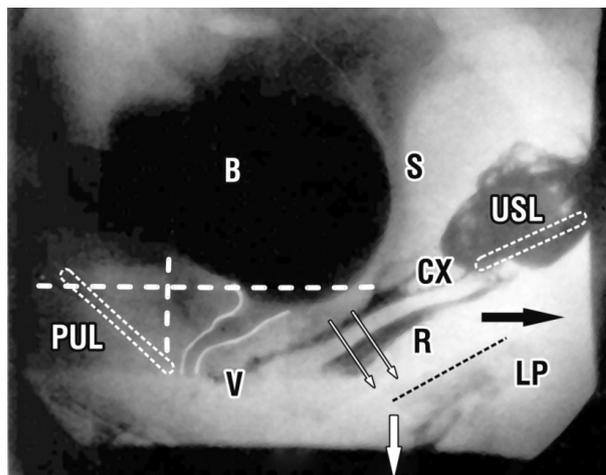


Figure 8. – Urethral opening during micturition. Same patient and labelling as in figure 6. Relaxation of m. pubococcygeus (forward arrow, figure 7) allows the posterior vectors (arrows) to stretch back the vagina and posterior urethral wall.

Dysfunction, 15-76. Ch3 Diagnosis, 77-117. Ch4 Surgery. 118-218. Ch5 Pelvic Floor Rehabilitation. New Concepts. 219-225. Ch6. Urodynamics. 226-258.

5. Digesu GA, Khuller V, Cardozo L, Salvatore S. Overactive bladder symptoms: do we need urodynamics? *Neurourol Urodyn.* 2003; 22:105-108.
6. de Boer TA, Salvatore S, Cardozo L et al. Pelvic organ prolapse and overactive bladder. *Neurourol Urodyn.* 2010; 29: 30-39.
7. Petros PE. New ambulatory surgical methods using an anatomical classification of urinary dysfunction improve stress, urge, and abnormal emptying, *Int Urogynecol J* 1997; 5: 270-278.
8. Petros PE, Swash M, Kakulas B. Stress urinary incontinence results from muscle weakness and ligamentous laxity in the pelvic floor. *Pelvipiperineology*, 2008; 27:107-109.
9. Rechberger T, Uldbjerg N, Oxlund H. Connective tissue changes in the cervix during normal pregnancy and pregnancy complicated by a cervical incompetence. *Obstets & Gynecol* 1988; 71:563-567.
10. Francis WJA. Disturbance of bladder function in relation to pregnancy. *Journal Obstets & Gynaecol British Empire*, 1960; 67:353-366.
11. Gordon AM, Huxley AF, Julian FJ. The variation in isometric tension with sarcomere length in vertebrate muscle fibres. *J Physiol.* 1966; 184:170-92.
12. Blaisdell FE. The anatomy of the sacrouterine ligaments. *Anat Rec* 1917; 12:22
13. Campbell RM. The anatomy and histology of the sacrouterine ligaments. *Am J Obstet Gynecol* 1950; 59:1-12.
14. Butler-Manuel SA, BATTERY LD, Polak JM, A'Hern R, Barton DP. Autonomic nerve trauma at radical hysterectomy: the nerve content and subtypes within the superficial and deep uterosacral ligaments. *Reprod Sci* 2008; 15:91-96.

#### REFERENCES FROM IUJ NOVEMBER 2012

15. Study No 1 Diamond P, Hassonah, S. Alarab M, Lovatsis D, Drutz HP, The prevalence of detrusor overactivity amongst patients with symptoms of overactive bladder: a retrospective cohort study, *Int Urogynecol J* 2012; 23:1577-1580.
16. Study No 2 Fitz FF, Resende AP, Stupp L, et al. Biofeedback for the treatment of female pelvic floor muscle dysfunction: a systematic review and meta-analysis *Int Urogynecol J* 2012; 23: 1495-1516.
17. Study No 3 Editorial. Abdel-Fattah M, Rizk DE. Diabetes mellitus and female urinary incontinence: a time for change. *Int Urogynecol J* 2012; 23:1481-148.
18. Study No 4 Zhang XY, Xu HN, Xia ZJ, Wu B. Analysis of clinical interventional strategy for women with urinary incontinence complicated with diabetes mellitus *Int Urogynecol J* 2012; 23:1527-153.

19. Study No 5 Shek KL, Kruger J, Dietz HP. The effect of pregnancy on hiatal dimensions and urethral mobility: an observational study, *Int Urogynecol J* 2012; 23:1561-1567.
20. Study No 6 Kapoor DS, Housami FP, Swithinbank L, Drake M. Maximum urethral closure pressure in women: normative data and evaluation as a diagnostic test *Urogynecol J*. 2012; 23:1613-1618.
21. Study No 7 Brazell HD, Claydon CS, Li J, Moore C, Dereska N, Hudson S, Swift S. Does neuromuscular blockade affect the assessment of pelvic organ prolapse? *Int Urogynecol J* 2012; 23:1599-1603.
22. Study No 8 Parker-Autry CY, Burgio KL, Richter HE. Vitamin D status: a review with implications for the pelvic floor *Int Urogynecol J* 2012; 23:1517-1526.
23. Study No 9 Daan NMP, Schweitzer KJ, van der Vaart CH. Associations between subjective overactive bladder symptoms and objective parameters on bladder diary and filling cystometry *Int Urogynecol J* 2012; 23:1619-1624.
24. Study No 10 Chin HY, Lin KC, Wang CJ, Chiang CH, Kuo HC. Paraurethral striated muscular structures and pelvic floor muscles contribute to resting urethral closure pressure in rats *Int Urogynecol J* 2012; 23:1631-1636.
25. Study No 11 Levin PI, Wu JM, Kawasaki A, Weidner AC, Amundsen CL. The efficacy of posterior tibial nerve stimulation for the treatment of overactive bladder in women: a systematic review *Int Urogynecol J* 2012; 23:1591-1597.
26. Study No 12 Capes T, Stanford EJ, Romanzi L, Foma Y, Moshier E. Comparison of two classification systems for vesicovaginal fistula, *Int Urogynecol J* 2012; 23:1679-1685.
27. Study No 13 Ramanah R, Berger MB, Parratte BM, DeLancey JOL. Anatomy and histology of apical support: a literature review concerning cardinal and uterosacral ligaments, *Int Urogynecol J* 2012; 23:1483-1494.

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