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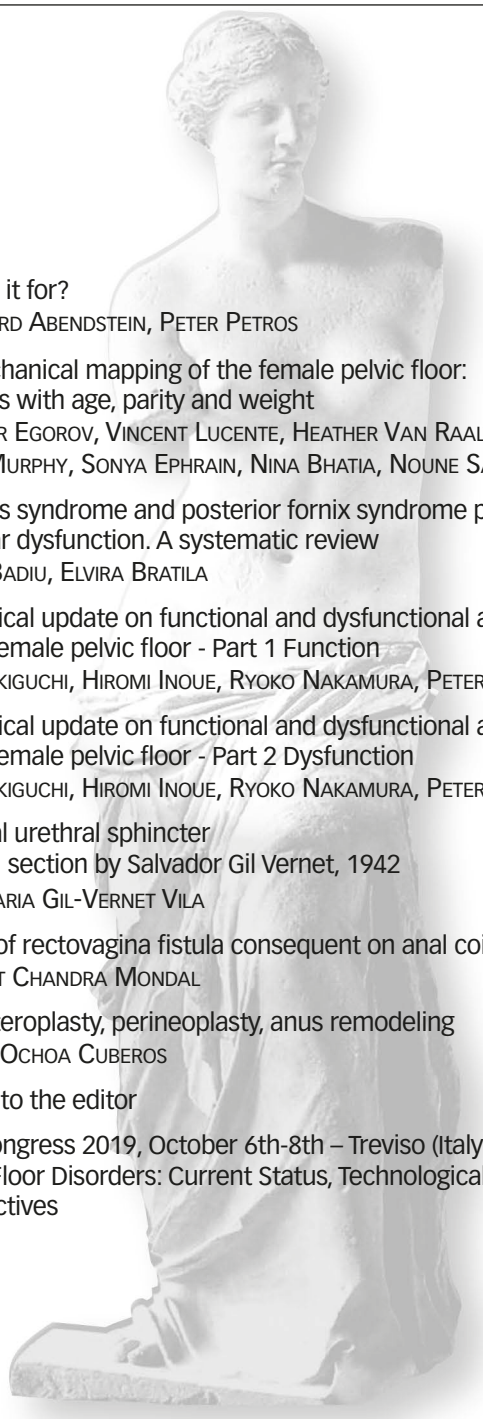

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e-mail: benito.ferraro@sanita.padova.it - luisa.marcato@sanita.padova.it
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Editorial

What is it for?

BURGHARD ABENDSTEIN, PETER PETROS

[1] *FA für Gynäkologie, Geburtshilfe und Chirurgie, Akademisches Lehrkrankenhaus, Feldkirch, Austria*
[2] *University of NSW Professorial Dept of Surgery, St Vincent's Hospital Sydney*

The Urologist/Anatomist Salvador Gil Vernet (1892 – 1987) made many outstanding contributions to anatomical knowledge and urological surgery. He was greatly admired by his fellow urologist, the Canadian-American Charles Brenton Huggins, Nobel Laureate. Gil Vernet's famous statement was that an answer was required to the question of *"what is it for?"*.

The question *"what is it for?"*

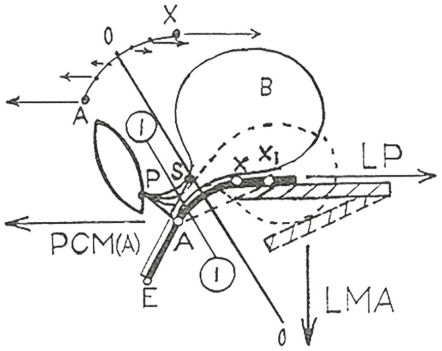
The authors never cease to be astounded at the answers received to our question, "How does the midurethral sling (MUS) work?" The standard reply is "It reinforces the pubourethral ligament"(PUL). A frequent reply to the next question, "What is the mechanism of urethral closure" is typically answered by "Intraabdominal pressure transmission". Usually there is no answer to subsequent questions, "How does PUL reconstruction restore the closure mechanisms?" Or "How does the MUS restore intraabdominal pressure transmission" or "Have you read the original studies which led to the MUS?"

How MUS works- original descriptions

The mechanicss of urethral closure (Fig. 1) is an original 1990 figure which explained the mechanism for urethral closure. It is based on the urodynamic, dynamic ultrasound, xray and prototype MUS surgical studies described in 79 pages of the 1990 ACTA Obstetrica et Gynecologica Scandinavica[1]. These studies scientifically underpinned the prototype MUS and the Integral Theory on which the MUS is based[1]. The key discoveries from the experiments detailed in [1] which led to the midurethral sling were: 3 directional muscle forces contract against competent pubourethral ligaments (PUL) to close the distal urethra and bladder neck, fig1; that loose pubourethral ligaments cause urinary stress incontinence (USI); that collagen damage causes loose ligaments; that a precisely inserted tape creates new collagen to repair the PUL. All subsequent surgeries which use tapes for ligament repair, whether for USI, prolapse or other symptoms are based on this concept, repair of collagen damage to the ligaments[2].

The mechanics of urinary stress incontinence (USI). In the original description for USI causation[1], a loose PUL, fig1, cannot support the vagina (heavy black line) or urethra. On effort, the pubourethral ligament 'P-A' elongates and the system enters "open mode', exactly the same as what occurs during micturition when PCM(A) relaxes[1]; the posterior vectors LP/LMA funnel bladder base/urethra between X_1 (bladder base) and A (insertion point of PUL) and urine is lost. In the prototype operation, the pubourethral ligament length P-A was restored by the MUS tape, which also restored the two closure mechanisms, distal and bladder neck[1].

Independent validation of the mechanics of continence restoration This mechanism for restoration, first demonstrated clinically, with dynamic ultrasound and xrays in 1990, was independently validated by a well documented 3D ultrasound study by Wen et al in 2018, 28 years later[3]. Wen et al. confirmed several predictions of the original 1990 Integral Theory[1]: 3 directional vector forces acting around point 0-0; that point P-A (pubourethral ligament) 'PUL' lengthens with USI; that P-A is shortened in patients cured of USI and remains lengthened in those not cured of USI. None of the findings from the original studies, nor Wen et al's confirmation thereof, nor the following video demonstration of the distal and proximal closure mechansims by a 'virtual' or 'simulated' operation can be explained by pressure transmission theories. (see video 'virtual op.' https://youtu.be/0UZuJtajCQU). The video shows partial control with midurethral support, but requires folding of the suburethral vagina for complete continence.



1990 diagram urethral closure copy
Urethral closure mechanism as described in 1990 (1) - Original diagram. I-I is the resting position of bladder neck. The heavy black line represents the vagina. During effort PCM (A) the anterior portion of m.pubococcygeus pulls the distal vagina A-E forwards against the pubourethral ligament 'P-A' to close distal urethra from behind and firmly anchor the distal urethra. Levator plate (LP) pulls backwards against P-A extending the the upper vagina from X to X1 and bladder neck to 0-0. X is the vesico-vaginal ligamentous attachment of bladder base to the anterior vaginal wall immediately below the cervix. P-S is the pubovesical ligament which inserts into 'S', a fibro-muscular thickening in the lower anterior wall of bladder 'B' known as the 'pre-cervical arc of Gil Vernet'. LMA (conjoint longitudinal muscle of the anus) contracts downwards against the cardinal/uterosacral ligaments to pull down on the anterior border of LP. This pulls down X1 and bladder around the arc of Gilvernet 'S', to effect closure at bladder neck (O-O) much like kinking a hose.

A final comment from Salvador Gil Vernet :*"Precise, almost mathematical knowledge of anatomy is a highly fertile source of surgical applications, suggesting new techniques and helping perfect and simplify existing surgical methods, making them less mutilating and more benign and, in short, raising surgery to the rank of true science."*

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Correspondence to:
[1]Burghard Abendstein MD PhD, burghard.abendstein@vlkh.net1

Original article

Biomechanical mapping of the female pelvic floor: changes with age, parity and weight

VLADIMIR EGOROV[1], VINCENT LUCENTE[2], HEATHER VAN RAALTE[3], MILES MURPHY[2], SONYA EPHRAIN[2], NINA BHATIA[3], NOUNE SARVAZYAN[1]

[1] *Artann Laboratories, Trenton, United States*
[2] *The Institute for Female Pelvic Medicine & Reconstructive Surgery, Allentown, United States*
[3] *Princeton Urogynecology, Princeton, United States*
Contact email: vegorov@artannlabs.com

Abstract: Quantitative biomechanical characterization of pelvic supportive structures and functions in vivo is thought to provide insight into the pathophysiology of pelvic floor disorders including pelvic organ prolapse (POP). An innovative approach - vaginal tactile imaging - allows biomechanical mapping of the female pelvic floor to quantify tissue elasticity, pelvic support, and pelvic muscle functions. The objective of this study is to explore an extended set of 52 biomechanical parameters to characterize pelvic floor changes with age, parity, and subject weight for normal pelvic floor conditions. 42 subjects with normal pelvic conditions (no POP, no stress urinary incontinence) were included in the data analysis from an observational, case-controlled study. The Vaginal Tactile Imager (VTI) was used with an analytical software package to automatically calculate 52 biomechanical parameters for 8 VTI test procedures (probe insertion, elevation, rotation, Val-salva maneuver, voluntary muscle contractions in 2 planes, relaxation, and reflex contraction). The ranges, mean values, and standard deviations for all 52 VTI parameters were established. 12 VTI parameters were identified as statistically sen-sitive ($p < 0.05$; t-test) to the subject age; 9 parameters were identified as statistically sensitive ($p < 0.05$; t-test) to the sub-ject parity; no sensitivity was found to subject weight. Among the 12 parameters sensitive to women's age, 6 parameters show changes (decrease) in tissue elasticity and 6 parameters show weakness in pelvic muscle functions with age. Among the 9 parameters sensitive to parity, 5 parameters show changes (decrease) in tissue elasticity and 4 parameters show weakness in pelvic muscle functions after giving birth. The biomechanical mapping of the female pelvic floor with the VTI provides a unique set of parameters characterizing pelvic changes with age and parity. These objectively measurable biomechanical transformations of pelvic tissues, support structures, and functions may be used in future research and practical applications.

Keywords: Biomechanical mapping; Female pelvic floor; Aging, Parity, Tactile imaging; Tissue elasticity, Pelvic sup-port, Pelvic function, Elastography,

INTRODUCTION

Many pelvic floor disorders including POP, stress urinary incontinence (SUI), sexual dysfunction, congenital anomalies, and others are clearly manifested in the mechanical properties of pelvic organs [1–4]. Therefore, biomechanical mapping of the response to applied pressure or loads within the pelvic floor opens up new possibilities in biomechanical assessment and monitoring of pelvic floor conditions. The newly developed vaginal tactile imaging technique allows biomechanical mapping of the female pelvic floor including assessment of tissue elasticity, pelvic support, and pelvic muscle functions in high definition [5, 6].

Previously, we reported the intra- and inter-observer reproducibility of vaginal tactile imaging [7] and proposed interpretation of biomechanical mapping of the female pelvic floor [8]. The new mechanistic parameters were introduced for assessment of the vaginal [9] and pelvic floor conditions [10].

The objective of this study is to identify an extended set of Vaginal Tactile Imager (VTI) parameters for biomechanical mapping of the female pelvic floor, to establish parameter ranges, and to explore their sensitivity to age, parity and patient weight for normal pelvic floor conditions.

MATERIALS AND METHODS

Definitions

Tactile Imaging is a medical imaging modality translating the sense of touch into a digital image [9]. The tactile image is a function of $P(x, y, z)$, where P is the pressure on soft tissue surface under applied deformation and x, y and z are the coordinates where P was measured. The tactile image is a pressure map on which the direction of tissue deformation must be specified.

Functional Tactile Imaging translates muscle activity into dynamic pressure pattern $P(x, y, t)$ for an area of interest, where t is time and x and y are coordinates where pressure P was measured. It may include: (a) muscle voluntary contraction, (b) involuntary reflex contraction, (c) involuntary relaxation, and (d) specific maneuvers.

Biomechanical Mapping = Tactile Imaging + Functional Tactile Imaging

A tactile imaging probe has a pressure sensor array mounted on its face that acts similar to human fingers during a clinical examination, deforming the soft tissue and detecting the resulting changes in the pressure pattern on the surface. The sensor head is moved over the surface of the tissue to be studied, and the pressure response is evaluated at multiple locations along the tissue. The results are used to generate 2D/3D images showing pressure distribution over the area of the tissue under study.

Generally, an inverse problem solution for tactile image $P(x, y, z)$ would allow the reconstruction of tissue elasticity distribution (E) as a function of the same coordinates $E(x, y, z)$. Unfortunately, the inverse problem solution is hardly possible for most real objects because it is a non-linear and ill-posed problem. However, the tactile image $P(x, y, z)$ per se reveals tissue or organ anatomy and elasticity distribution because it maintains the stress-strain relationship for deformed tissue [11, 12]. Thus the spatial gradients $\partial P(x, y, z)/\partial x$, $\partial P(x, y, z)/\partial y$, and $\partial P(x, y, z)/\partial z$ can be used in practice for soft tissue elasticity mapping, despite structural and anatomical variations [6].

Vaginal Tactile Imager

The VTI, model 2S (Advanced Tactile Imaging, Inc., NJ), was used in all test procedures. The VTI probe, as shown

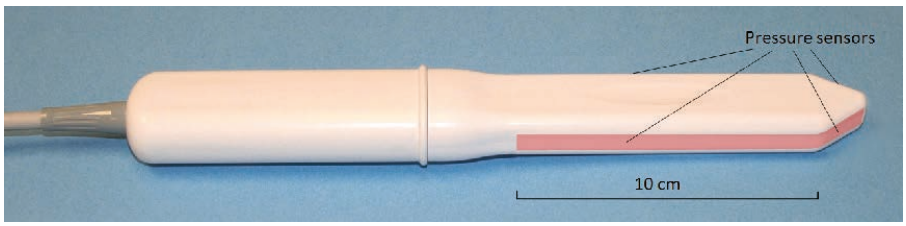

Figure 1. - Vaginal Probe. Pressure sensors are aligned on the outer surfaces of the probe (highlighted in the image).

in Figure 1, is equipped with 96 pressure (tactile) sensors spaced at 2.5 mm consecutively on both sides of the probe, an orientation sensor, and temperature controllers to provide the probe temperature close to a human body before the examination. During the clinical procedure, the probe is used to acquire pressure responses from two opposite vaginal walls along the vagina. The VTI data are sampled from the probe sensors and displayed on the VTI monitor in real time. The resulting pressure maps (tactile images) of the vagina integrate all the acquired pressure and positioning data for each of the pressure sensing elements. Additionally, the VTI records the dynamic contraction for pelvic floor muscles with resolution of 1 mm. A lubricating jelly is used for patient comfort and to provide reproducible boundary/contact conditions with deformed tissues.

This VTI probe allows 3 - 15 mm tissue deformation at the probe insertion (Tests 1), 20 - 45 mm tissue deformation at the probe elevation (Test 2), 5 - 7 mm deformation at the probe rotation (Test 3) and recording of dynamic responses at pelvic muscle contractions (Tests 4 - 8). The probe maneuvers in Tests 1 - 3 allow accumulation of multiple pressure patterns from the tissue surface to compose an integrated tactile image for the investigated area using a proprietary image composition algorithm similar to the imaging of the prostate and breast [11, 12]. The spatial gradients $\partial P\,(x, y) / \partial y$ for anterior and posterior compartments are calculated within the acquired tactile images in Tests 1 and 2; *y*-coordinate is directed orthogonally from the vaginal channel, x-coordinate is located on the vaginal channel. The VTI software includes data analysis tools and reporting functions. It visualizes the anatomy, pressure maps, and calculates (automatically) 52 VTI parameters for eight test procedures. The VTI examination procedure consists of eight tests: 1) probe insertion, 2) elevation, 3) rotation, and 4) Valsalva maneuver, 5) voluntary muscle contraction, 6) voluntary muscle contraction (left *versus* right side), 6) involuntary relaxation, and 8) reflex muscle contraction (cough). Tests 1 - 5 and 7 - 8 provide data for anterior/posterior compartments; test 7 provides data for left/right sides.

The VTI absolute measurement accuracy is as follows: ±0.2 kPa within 10 kPa range, ±0.5 kPa at 25 kPa, ±1.0 kPa at 60 kPa. The VTI relative pressure measurement accuracy lies in the range between ±0.05 kPa to ±0.1 kPa. The VTI pressure measurement resolution is 0.001 kPa. The VTI absolute measurement accuracy for probe orientation is ±0.5 degree and ±0.1°C for measuring the temperature inside the probe on the surface of the pressure sensors. The VTI probe was calibrated immediately before every subject examination; it was cleaned and disinfected between the patients.

Biomechanical Mapping Parameters

Table 1 lists 52 biomechanical parameters being calculated for every participating subject based on VTI data recorded in tests 1 - 8. Anatomical assignment of the targeting/contributing pelvic structures into the specified parameters is based on published data [1, 3, 4, 13-15].

Population Description

42 subjects with normal pelvic conditions (no POP, no SUI) were inlcuded in the data analysis from multi-site ob-

Table 1. VTI Biomechanical Parameters.

No.	VTI Test	Parameters Abbreviation	Units	Parameter Description	Parameter Interpretation	Parameter Class	Targeting/Contributing Pelvic Structures
1	1	Fmax	N	Maximum value of force measured during the VTI probe insertion [9]	Maximum resistance of anterior *vs* posterior widening; tissue elasticity at specified location (capability to resist to applied deformation)	Maximum vaginal tissue elasticity at specified location	Tissues behind the anterior and posterior vaginal walls at 3-15 mm depth
2	1	Work	mJ	Work completed during the probe insertion (Work = Force × Displacement) [9]	Integral resistance of vaginal tissue (anterior and posterior) along the probe insertion	Average vaginal tissue elasticity	Tissues behind the anterior and posterior vaginal walls at 3-15 mm depth
3	1	Gmax_a	kPa/mm	Maximum value of anterior gradient (change of pressure per anterior wall displacement in orthogonal direction to the vaginal channel)	Maximum value of tissue elasticity in anterior compartment behind the vaginal at specified location	Maximum value of anterior tissue elasticity	Tissues/structures in anterior compartment at 10-15 mm depth
4	1	Gmax_p	kPa/mm	Maximum value of posterior gradient (change of pressure per posterior wall displacement in orthogonal direction to the vaginal channel)	Maximum value of tissue elasticity in posterior compartment behind the vaginal at specified location	Maximum value of posterior tissue elasticity	Tissues/structures in anterior compartment at 10-15 mm depth
5	1	Pmax_a	kPa	Maximum value of pressure per anterior wall along the vagina	Maximum resistance of anterior tissue to vaginal wall deformation	Anterior tissue elasticity	Tissues/structures in anterior compartment
6	1	Pmax_p	kPa	Maximum value of pressure per posterior wall along the vagina	Maximum resistance of posterior tissue to vaginal wall deformation	Posterior tissue elasticity	Tissues/structures in posterior compartment
7	2	P1max_a	kPa	Maximum pressure at the area of pubic bone (anterior, A1 in Figure 2)	Proximity of pubic bone to vaginal wall and perineal body strength	Anatomic aspects and tissue elasticity	Tissues between vagina and pubic bone; perineal body
8	2	P2max_a	kPa	Maximum pressure at the area of urethra (anterior, A2 in Figure 2)	Elasticity/mobility of urethra	Anatomic aspects and tissue elasticity	Urethra and surrounding tissues
9	2	P3max_a	kPa	Maximum pressure at the cervix area (anterior, A3 in Figure 2)	Mobility of uterus and conditions of uterosacral and cardinal ligaments	Pelvic floor support	Uterosacral and cardinal ligaments
10	2	P1max_p	kPa	Maximum pressure at the perineal body (posterioir, see P1 in Figure 2)	Pressure feedback of Level III support	Pelvic floor support	Puboperineal, puborectal muscles

11	2	P2max_p	kPa	Maximum pressure at middle third of vagina (posterioir, see P2 in Figure 2)	Pressure feedback of Level II support	Pelvic floor support	Pubovaginal, puboanal muscles
12	2	P3max_p	kPa	Maximum pressure at upper third of vagina (posterioir, see P3 in Figure 2)	Pressure feedback of Level I support	Pelvic floor support	Iliococcygeal muscle, levator plate
13	2	G1max_a	kPa/mm	Maximum gradient at the area of pubic bone (anterior, see A1 in Figure 2)	Vaginal elasticity at pubic bone area	Anterior tissue elasticity	Tissues between vagina and pubic bone; perineal body
14	2	G2max_a	kPa/mm	Maximum gradient at the area of urethra (anterior, see A2 in Figure 2)	Mobility and elasticity of urethra	Urethral tissue elasticity	Urethra and surrounding tissues
15	2	G3max_a	kPa/mm	Maximum gradient at the cervix area (anterior, see A3 in Figure 2)	Conditions of uterosacral and cardinal ligaments	Pelvic floor support	Uterosacral and cardinal ligaments
16	2	G1max_p	kPa/mm	Maximum gradient at the perineal body (posterioir, see P1 in Figure 2)	Strength of Level III support (tissue deformation up to 25 mm)	Pelvic floor support	Puboperineal, puborectal muscles
17	2	G2max_p	kPa/mm	Maximum gradient at middle third of vagina (posterioir, see P2 in Figure 2)	Strength of Level II support (tissue deformation up to 35 mm)	Pelvic floor support	Pubovaginal, puboanal muscles
18	2	G3max_p	kPa/mm	Maximum gradient at upper third of vagina (posterioir, see P3 in Figure 2)	Strength of Level I support (tissue deformation up to 45 mm)	Pelvic floor support	Iliococcygeal muscle, levator plate
19	3	Pmax	kPa	Maximum pressure at vaginal walls deformation by 7 mm [9]	Hard tissue or tight vagina	Vaginal tissue elasticity	Tissues behind the vaginal walls at 5-7 mm depth
20	3	Fap	N	Force applied by anterior and posterior compartments to the probe [9].	Integral strength of anterior and posterior compartments	Vaginal tightening	Tissues behind anterior/ posterior vaginal walls.
21	3	Fs	N	Force applied by entire left and right sides of vagina to the probe [9].	Integral strength of left and right sides of vagina	Vaginal tightening	Vaginal right/left walls and tissues behind them.
22	3	P1_l	kPa	Pressure response from a selected location (irregularity 1) at left side (see S1 in Figure 2)	Hard tissue on left vaginal wall	Irregularity on vaginal wall	Tissue/muscle behind the vaginal walls on left side.
23	3	P2_l	kPa	Pressure response from a selected location (irregularity 2) at left side (see S2 in Figure 2)	Hard tissue on left vaginal wall	Irregularity on vaginal wall	Tissue/muscle behind the vaginal walls on left side.
24	3	P3_r	kPa	Pressure response from a selected location (irregularity 3) at right side (see S1 in Figure 2)	Hard tissue on right vaginal wall	Irregularity on vaginal wall	Tissue/muscle behind the vaginal walls on right side.
25	4	dF_a	N	Integral force change in anterior compartment at Valsalva maneuver	Pelvic function* at Valsalva maneuver	Pelvic function	Multiple pelvic muscle*
26	4	dPmax_a	kPa	Maximum pressure change in anterior compartment at Valsalva maneuver.	Pelvic function* at Valsalva maneuver	Pelvic function	Multiple pelvic muscle*
27	4	dL_a	mm	Displacement of the maximum pressure peak in anterior compartment	Mobility of anterior structures* Valsalva maneuver	Pelvic function	Urethra, pubovaginal muscle; ligaments*
28	4	dF_p	N	Integral force change in posterior compartment at Valsalva maneuver	Pelvic function* at Valsalva maneuver	Pelvic function	Multiple pelvic muscle*
29	4	dPmax_p	kPa	Maximum pressure change in posterior compartment at Valsalva maneuver.	Pelvic function* at Valsalva maneuver	Pelvic function	Multiple pelvic muscle*
30	4	dL_p	mm	Displacement of the maximum pressure peak in posterior compartment	Mobility of posterioir structures* Valsalva maneuver	Pelvic function	Anorectal, puborectal, pubovaginal muscles; ligaments*
31	5	dF_a	N	Integral force change in anterior compartment at voluntary muscle contraction	Integral contraction strength of pelvic muscles along the vagina	Pelvic function	Puboperineal, puborectal, pubovaginal and ilicoccygeal muscles; uretra
32	5	dPmax_a	kPa	Maximum pressure change in anterior compartment at voluntary muscle contraction	Contraction strength of specified pelvic muscles	Pelvic function	Puboperineal, puborectal and pubovaginal muscles
33	5	Pmax_a	kPa	Maximum pressure value in anterior compartment at voluntary muscle contraction.	Static and dynamic peak support of the pelvic floor	Pelvic function	Puboperineal and puborectal muscles*
34	5	dF_p	N	Integral force change in posterior compartment at voluntary muscle contraction	Integral contraction strength of pelvic muscles along the vagina	Pelvic function	Puboperineal, puborectal, pubovaginal and ilicoccygeal muscles
35	5	dPmax_p	kPa	Maximum pressure change in posterior compartment at voluntary muscle contraction	Contraction strength of pelvic muscles at specified location	Pelvic function	Puboperineal, puborectal and pubovaginal muscles
36	5	Pmax_p	kPa	Maximum pressure value in posterior compartment at voluntary muscle contraction.	Static and dynamic peak support of the pelvic floor	Pelvic function	Puboperineal and puborectal muscles*
37	6	dF_r	N	Integral force change in right side at voluntary muscle contraction	Integral contraction strength of pelvic muscles along the vagina	Pelvic function	Puboperineal, puborectal, and pubovaginal muscles
38	6	dPmax_r	kPa	Maximum pressure change in right side at voluntary muscle contraction	Contraction strength of specific pelvic muscle	Pelvic function	Puboperineal or puborectal or pubovaginal muscles
39	6	Pmaxa_r	kPa	Maximum pressure value in right side at voluntary muscle contraction	Specified pelvic muscle contractive capability and integrity	Pelvic function	Puboperineal or puborectal muscles
40	6	dF_l	N	Integral force change in left side at voluntary muscle contraction	Integral contraction strength of pelvic muscles along the vagina	Pelvic function	Puboperineal, puborectal, and pubovaginal muscles
41	6	dPmax_l	kPa	Maximum pressure change in left side at voluntary muscle contraction	Contraction strength of specific pelvic muscle	Pelvic function	Puboperineal or puborectal or pubovaginal muscles
42	6	Pmaxa_l	kPa	Maximum pressure value in left side at voluntary muscle contraction	Specified pelvic muscle contractive capability and integrity	Pelvic function	Puboperineal or puborectal muscles
43	7	dPdt_a	kPa/s	Anterior absolute pressure change per second for maximum pressure at involuntary relaxation	Innervation status of specified pelvic muscles	Innervations status	Levator ani muscles
44	7	dpcdt_a	%/s	Anterior relative pressure change per second for maximum pressure at involuntary relaxation	Innervation status of specified pelvic muscles	Innervations status	Levator ani muscles
45	7	dPdt_p	kPa/s	Posterior absolute pressure change per second for maximum pressure at involuntary relaxation	Innervation status of specified pelvic muscles	Innervations status	Levator ani muscles
46	7	dpcdt_p	%/s	Posterior relative pressure change per second for maximum pressure at involuntary relaxation	Innervation status of specified pelvic muscles	Innervations status	Levator ani muscles

47	8	dF_a	N	Integral force change in anterior compartment at reflex pelvic muscle contraction (cough)	Integral pelvic function* at reflex muscle contraction	Pelvic function	Multiple pelvic muscle*
48	8	dPmax_a	kPa	Maximum pressure change in anterior compartment at reflex pelvic muscle contraction (cough).	Contraction strength of specified pelvic muscles	Pelvic function	Multiple pelvic muscle*
49	8	dL_a	mm	Displacement of the maximum pressure peak in anterior compartment	Mobility of anterior structures* at reflex muscle contraction	Pelvic function	Urethra, pubovaginal muscle; ligaments*
50	8	dF_p	N	Integral force change in posterior compartment at reflex pelvic muscle contraction (cough)	Integral pelvic function* at reflex muscle contraction	Pelvic function	Multiple pelvic muscle*
51	8	dPmax_p	kPa	Maximum pressure change in posterior compartment at reflex pelvic muscle contraction (cough).	Contraction strength of specified pelvic muscles	Pelvic function	Multiple pelvic muscle*
52	8	dL_p	mm	Displacement of the maximum pressure peak in posterior compartment	Mobility of anterior structures* at reflex muscle contraction	Pelvic function	Anorectal, puborectal and pubovaginal muscles; ligaments*

** requires further interpretation*

servational, case-controlled study (clinical trial identifier NCT02294383). The subject age, height, weight, and parity distribution data are present in Table 2. Prior to the VTI examination, a standard physical examination was performed, including a bimanual pelvic examination and Pelvic Organ Prolapse Quantification (POP-Q) [16]. None of the analyzed subjects had a prior history of pelvic floor surgery. The clinical protocol was approved by the Institutional Review Board and all women provided written informed consent to be enrolled into the study. This clinical research was done in compliance with the Health Insurance Portability and Accountability Act. The VTI examination data for eight Tests were obtained and recorded at the time of the scheduled routine urogynecologic visits.

Total study workflow comprised of the following steps: (1) Recruiting women who routinely undergo vaginal examination as a part of their diagnostic treatment of concerned areas; (2) Acquisition of clinical diagnostic information related to the studied cases by standard clinical means; (3) Performing a VTI examination in lithotomic position; (4) Analyzing VTI data and assessment of the VTI parameters for pelvic floor characterization.

Statistical Analysis

52 biomechanical parameters were calculated automatically per each of the 42 analyzed VTI examinations or cases (one VTI examination per each subjects). In some rare cases the parameter calculation required a manual correction of the anatomical location where the parameters must be calculated. Unpaired *t*-tests between two subject groups with thresholds by age, parity or subject weight were completed per parameter to determine whether the parameter showed dependence on the age, parity or subject weight. For visual evaluation of the analyzed clinical data distributions we used notched boxplots [17] showing a confidence interval for the median value (central horizontal line), 25% and 75% quartiles. The spacing between the different parts of the box helps to compare variance. The boxplot also determines skewness (asymmetry) and outlier (cross). The intersection or divergence of confidence intervals for two patient samples is a visual analog of the *t*-test. The MATLAB (MathWorks, MA) statistical functions were used for the data analysis. The MATLAB (MathWorks, MA) statistical functions were used for the data analysis.

Figure 2 shows the locations of the measured VTI parameters for test 2 and 3 in mid-sagittal plane of the female pelvic floor.

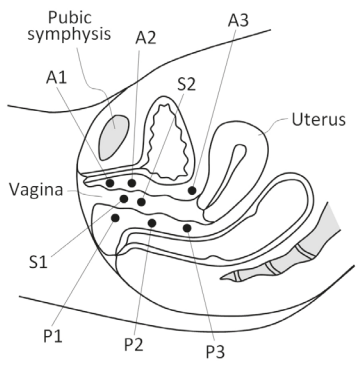

Figure 2. - Locations of the VTI parameters within the pelvic floor. A1-A3 are in anterior compartment (Test 2), P1-P3 in posterior compartment (Test 2), and S1, S2 are in lateral compartments (left and right sides, Test 3).

RESULTS

To illustrate the approach and location used in calculating the biomechanical parameters, the VTI examination data for all eight tests, as they observed by an operator in real time, are displayed in Figures 4 - 11 (see Supplementary material). Table 2 displays the calculated statistics (hypothesis testing outcome *H*- and *p*-value) for two age groups with threshold of 52 y.o. Average values for 52 biomechanical parameters and standard deviations (SD) for both groups are presented. Table 4 presents the calculated statistics (hypothesis testing outcome *H*- and *p*-value) for nulliparous *versus* parous (Parity > 0) woment, two parity groups average values and standard deviations for 52 biomechanical parameters.

Calculated statistics (hypothesis testing outcome *H*- and *p*-value) for two weight groups with threshold of 69 kg demonstrated no sensitivity to women weight; $H = 0$ and $p > 0.316$ for all 52 biomechanical parameters.

The *t*-tests for the age groups demonstrate that 12 out of 52 parameters have statistically significant differences between the groups and that these parameters have the potential to be used for identification of age-related changes in the female pelvic floor (see Table 2). The analysed groups, statistically, have the same subject height, weight, and parity distributions ($H = 0$, $p>0.05$). The *t*-tests for the parity groups demonstrate that 9 out of 52 parameters have statistically significant differences between the groups and that these parameters have the potential to be used for the identification of parity changes in the female pelvic floor (see Table 3). The analysed groups also statistically have the same subject height, weight and age distributions ($H = 0$, $p>0.05$). The *t*-tests for the weight groups demonstrate that o out of 52 parameters have statistically significant differences between the groups; nothing changes with women's weight (see Table 3). The analysed groups thus statistically have the same subject height, age and parity distributions ($H = 0$, $p>0.05$).

Figure 3 displays the boxplots for select parameters for the age groups presented in Table 2 (panels A - D) and for parity groups presented in Table 3 (panels E - H).

Table 2. Biomechanical Parameters for two age groups: Group 1 of 26 subjects ≤52 y.o., Group 2 of 16 subjects >52 y.o.

		H	*p*	Units	Average Group 1	Average Group 2	SD Group 1	SD Group 2
	Height →	0	0.315	cm	163.1	159.3	10.1	14.3
	Weight →	0	0.177	kg	60.4	80.8	6.8	6.1
	Age →	1	**1×10^{-12}**	**y.o**	**40.7**	**68.3**	8.3	9.0
	Parity (P) →	0	0.117	-	1.23	1.75	0.99	1.06
Parameters number ↓	**Test ↓**							
1	1	1	0.016	N	1.46	0.90	0.80	0.48
2	1	1	0.009	mJ	49.23	31.14	23.41	15.79
3	1	0	0.103	kPa/mm	2.82	1.67	2.65	0.84
4	1	0	0.196	kPa/mm	1.74	1.29	1.21	0.78
5	1	0	0.263	kPa	43.10	33.48	29.90	20.24
6	1	1	0.047	kPa	26.07	17.07	16.27	8.15
7	2	0	0.490	kPa	26.96	30.33	14.04	17.02
8	2	0	0.534	kPa	12.44	10.89	8.21	6.98
9	2	0	0.648	kPa	8.96	7.78	6.70	10.03
10	2	0	0.077	kPa	15.86	10.45	11.19	5.14
11	2	0	0.914	kPa	9.45	9.68	7.23	5.03
12	2	0	0.198	kPa	8.14	4.99	9.22	3.24
13	2	0	0.488	kPa/mm	1.74	2.12	1.74	1.54
14	2	0	0.794	kPa/mm	0.76	0.83	0.76	0.99
15	2	0	0.610	kPa/mm	0.62	0.51	0.58	0.81
16	2	0	0.057	kPa/mm	0.93	0.39	1.06	0.40
17	2	0	0.763	kPa/mm	0.40	0.43	0.29	0.32
18	2	0	0.107	kPa/mm	0.56	0.25	0.74	0.18
19	3	0	0.562	kPa	33.27	30.37	15.42	15.99
20	3	0	0.120	N	4.40	3.45	2.14	1.34
21	3	0	0.073	N	1.37	0.90	0.90	0.60
22	3	1	0.003	kPa	11.43	5.60	7.10	2.02
23	3	1	0.012	kPa	5.87	3.42	3.52	1.55
24	3	1	0.001	kPa	12.29	5.91	6.93	2.48
25	4	0	0.128	N	1.44	1.02	0.94	0.53
26	4	0	0.717	kPa	11.28	9.90	12.38	9.07
27	4	0	0.558	mm	2.31	1.28	4.68	5.47
28	4	0	0.094	N	1.46	0.94	1.06	0.56
29	4	0	0.350	kPa	7.73	5.76	7.02	4.78
30	4	0	0.299	mm	1.44	3.44	4.46	6.49
31	5	0	0.248	N	1.71	1.35	1.09	0.79
32	5	0	0.124	kPa	25.15	17.58	16.43	12.79
33	5	0	0.259	kPa	43.52	36.54	20.97	15.76
34	5	0	0.070	N	2.11	1.39	1.36	0.96
35	5	1	0.024	kPa	16.45	9.66	10.58	5.98
36	5	1	0.009	kPa	26.06	17.39	11.59	6.51
37	6	0	0.056	N	1.03	0.63	0.62	0.59
38	6	1	0.019	kPa	9.70	5.04	6.33	4.80
39	6	1	0.004	kPa	16.62	8.99	8.41	6.16
40	6	0	0.110	N	1.01	0.64	0.70	0.67
41	6	1	0.031	kPa	8.61	4.71	5.57	4.80
42	6	1	0.002	kPa	15.81	7.86	8.48	5.51
43	7	0	0.501	kPa/s	-1.45	-1.08	1.82	1.37
44	7	0	0.618	%/s	-3.36	-2.76	3.79	3.31
45	7	0	0.195	kPa/s	-1.27	-0.68	1.60	0.87
46	7	0	0.785	%/s	-4.26	-3.91	4.24	3.37
47	8	0	0.138	N	2.59	1.83	1.44	1.33
48	8	0	0.990	kPa	13.91	13.97	14.27	16.30
49	8	0	0.530	mm	6.99	5.91	3.91	5.66
50	8	0	0.062	N	2.68	1.69	1.42	1.46
51	8	0	0.096	kPa	13.55	8.66	7.94	8.01
52	8	0	0.723	mm	4.01	3.19	6.03	6.90

Table 3. Biomechanical Parameters for two groups: Group 1 of 11 nulliparous subjects, Group 2 of 31 subjects with parity ≥1.

		H	*p*	Units	Aver Group 1	Aver Group 2	SD Group 1	SD Group 2
Height →		0	0.843	cm	161.1	161.9	13.8	11.3
Weight →		0	0.587	kg	147.5	152.6	25.1	27.2
Age →		0	0.185	y.o	45.6	53.1	15.2	16.1
Parity (P) →		1	**1×10^{-11}**	-	**0.0**	**1.9**	0.0	0.7
Parameters number ↓	**Test ↓**							
1	1	1	0.034	N	1.65	1.10	1.08	0.53
2	1	0	0.141	mJ	50.95	39.29	29.43	19.08
3	1	0	0.072	kPa/mm	3.41	2.01	3.18	1.66
4	1	0	0.651	kPa/mm	1.69	1.52	0.61	1.21
5	1	0	0.347	kPa	46.04	37.09	36.63	22.61
6	1	0	0.291	kPa	26.61	21.24	11.45	15.14
7	2	0	0.497	kPa	25.54	29.20	5.00	17.35
8	2	0	0.427	kPa	10.24	12.42	6.22	8.20
9	2	0	0.511	kPa	7.12	9.00	4.79	8.92
10	2	0	0.321	kPa	16.31	12.91	9.10	9.82
11	2	0	0.826	kPa	9.16	9.67	6.88	6.35
12	2	0	0.397	kPa	8.64	6.34	12.46	5.11
13	2	1	0.020	kPa/mm	0.90	2.23	0.44	1.79
14	2	0	0.100	kPa/mm	0.43	0.92	0.44	0.92
15	2	0	0.343	kPa/mm	0.41	0.63	0.28	0.76
16	2	0	0.579	kPa/mm	0.86	0.68	0.73	0.96
17	2	0	0.221	kPa/mm	0.51	0.38	0.38	0.26
18	2	0	0.197	kPa/mm	0.64	0.37	1.03	0.36
19	3	0	0.464	kPa	35.15	31.10	15.42	15.65
20	3	0	0.332	N	4.52	3.86	2.00	1.89
21	3	0	0.061	N	1.59	1.05	0.81	0.80
22	3	0	0.144	kPa	11.63	8.35	5.98	6.36
23	3	1	0.006	kPa	7.09	4.17	4.16	2.32
24	3	1	0.042	kPa	13.22	8.67	6.16	6.19
25	4	0	0.218	N	1.55	1.14	1.14	0.66
26	4	0	0.891	kPa	11.10	10.48	13.25	10.25
27	4	0	0.284	mm	3.51	1.31	5.04	4.99
28	4	0	0.505	N	1.40	1.16	1.23	0.78
29	4	0	0.960	kPa	6.90	6.77	7.11	5.86
30	4	0	0.541	mm	3.45	2.06	4.69	5.79
31	5	0	0.216	N	1.89	1.46	1.51	0.73
32	5	1	0.047	kPa	30.17	19.46	21.62	11.79
33	5	0	0.064	kPa	50.08	37.58	22.65	17.14
34	5	0	0.313	N	2.17	1.72	1.74	1.06
35	5	0	0.360	kPa	16.17	13.04	12.72	8.36
36	5	0	0.322	kPa	25.55	21.76	12.30	10.18
37	6	1	0.034	N	1.27	0.74	0.87	0.51
38	6	0	0.096	kPa	10.88	6.80	7.34	5.55
39	6	0	0.093	kPa	17.71	12.11	7.65	8.25
40	6	1	0.008	N	1.42	0.69	1.13	0.45
41	6	1	0.044	kPa	10.39	5.97	7.05	4.76
42	6	0	0.072	kPa	17.03	11.09	8.73	7.82
43	7	0	0.975	kPa/s	-1.31	-1.29	1.55	1.68
44	7	0	0.993	%/s	-3.11	-3.10	3.22	3.70
45	7	0	0.868	kPa/s	-0.94	-1.03	0.80	1.47
46	7	0	0.666	%/s	-4.64	-3.96	3.65	3.94
47	8	0	0.406	N	2.63	2.13	1.15	1.51
48	8	0	0.153	kPa	20.51	11.74	8.12	16.14
49	8	0	0.796	mm	6.90	6.39	3.20	5.16
50	8	0	0.437	N	2.61	2.12	1.31	1.56
51	8	0	0.404	kPa	13.55	10.70	6.20	8.78
52	8	1	0.049	mm	7.44	2.39	9.71	4.32

DISCUSSION

The results of this research agree with previously reported data [4-10]; however, the current analysis includes the biggest VTI parameter set ever considered. 12 of 52 biomechanical parameters are identified as having statistically significant sensitivity to the women's age (see Tables 2). Their average changes are from 49.9% to 107.8% (78.2% in average). 9 of 52 biomechanical parameters are identified as having statistically significant sensitivity to women's parity (see Tables 3). Their average changes are from 49.6% to 211% (83.2% in average). These changes with age and parity clearly outperform possible deviations related to VTI intra- and inter-operator variability which were found on an average of ±15.1% (intra-observer error) and ±18.4 (inter-observer error) [7]. These reproducibility errors have value and sign intrinsically by a chance, but we have identified statistically systematic parameter changes by age and parity. No parameter changes were found with the women weight.

Let's consider the age changes. Test 1 provides three identified age-sensitive parameters (1, 2, 6) related to tissue elasticity; their average values is changed by 52.8% - 67.18% (see panels A – C in Figure 3). No changes in pelvic support, which could be detected by Test 2 parameters were found. Test 3 provides three identified parameters (22, 23, 24) related to the tissue elasticity of side vaginal walls and beyond 3-7 mm (small irregularities); their average values change from 71.6% to 108%. Pelvic muscle contraction Tests 5 and 6 provide six identified parameters (35, 36, 38, 39, 41, 42) related to pelvic function; their average values are changed by 49.9% - 101.2% (see panel D in Figure 3). All these 6 parameters demonstrate a decrease in muscle contractive capabilities in Level I (levator plate muscle) and Level II support (pubovaginal and ilicoccygeal muscles). Valsalva maneuver (Test 4), involuntary muscle relaxation (Test 7), and reflex muscle contraction at cough (Test 8) demonstrated no changes in parameters with age. In total, among the 12 age parameters, 6 parameters are related to decrease in vaginal tissue elasticity and 6 parameters are related to pelvic function – the weakened muscle contractive strength with age.

Let's consider the parity changes. Test 1 provides one parity-sensitive parameter (1) related to tissue elasticity; its average value changes by 49.6% (see panel E in Figure 3). This parameter is the maximum resistance force the VTI probe insertion. This maximum resistance comes from the perineal body which deteriorates after giving birth. No changes in pelvic support (Test 2) were found except for parameter 13, which increased with the parity (see panel F in Figure 3). This, at first sight, is an unexpected result and might be easily explained by significant softening of tissues between the vaginal wall and the pubic bone. As a result, these soft tissues demonstrate an increased pressure gradient to the bone due to their low resistance to displacement *versus* that of the bone. It means that parameter 13 changes with parity and relates to the tissue elasticity change rather than to pelvic support. Test 3 provides two identified parameters (23, 24) related to tissue elasticity at the side of vaginal walls and beyond 3-7 mm (small irregularities); their average values change by 70.1% and 52.5%. With regards to pelvic muscle contraction, Tests 5 and 6 provides four identified parameters (32, 37, 40, 41) related to pelvic function; their average values are changed by 55.0% - 105.5% (see parameter 37 change in Figure 3, panel G). These changes in pelvic muscle contractive strength must be related to avulsed puborectalis [15]. Valsalva maneuver (Test 4), involuntary muscle relaxation (Test 7), and the reflex muscle contraction at cough (Test 8) demonstrated no changes with the parity. In total, among the 9 age parameters, 5 parameters are related to a decrease in vaginal tissue elasticity and 4 parameters are related to pelvic function – the weakened muscle contractive strength (avulsion) with the parity.

It is important to note that the subject sample analysed in this study with normal pelvic conditions (no POP, no SUI) was composed of the visitors of urogynecological site; these patients may have had some pelvic floor conditions that were not identified in this study. Possibly, the patients from the normal group had pre-prolapse conditions which hadn't yet transformed into anatomically visible POP. This study reasonably proposes that if more subjects with no history of consulting urogynecological clinics would be added to this sample, more significant differences in the VTI parameters with regards to age and parity may be observed.

The next step (which falls beyond the purview of this article) with these biomechanical parameters may include (a) an insight into POP *versus* normal pelvic conditions, (b) an insight into POP classes (anterior *versus* posterior *versus* uterine), (c) analysis for continence *versus* incontinence conditions, (d) analysis of urogynecological surgical outcomes as a whole as well as per specific surgical procedure, (e) a combination of VTI data with urodynamics, ultrasound, and MRI data, (f) the usage of the VTI and other clinically related data for predicative modelling of outcomes for conservative and surgical procedures (personalized predictive treatment), and (g) maintenance of the objective history of biomechanical transformation of the patient pelvic floor.

One of the strengths of this study is that the current VTI offers an opportunity to assess tissue elasticity, pelvic support structures, and pelvic function (muscle and ligaments) in high definition along the entire length of the anterior, posterior, and lateral walls at rest with applied deflection pressures and pelvic muscle contractions. All 52 parameters are calculated automatically in real-time. This allows a large body of measurements to evaluate individual variations in support defects as well as identify specific problematic structures. In addition, the technology provides the opportunity to measure pelvic floor muscle strength at specific locations along the vaginal wall and helps correlate its relative contributions to measured tissue properties. These measurements may provide insight into the functional contribution or relationship between support ligaments and the underlying muscle support. Because VTI testing is relatively easy and inexpensive to obtain, post-treatment follow-up is available to evaluate the surgical impact on functional tissue properties and pelvic floor muscles. This may provide valuable outcome measurements for evaluating current and future treatments.

One of the shortcomings of this study is its relatively small sample size. Further studies with larger patient populations, investigating varieties of other pelvic floor conditions and their use in the evaluation of interventions including physical therapy, conservative management options, and surgical correction are needed to further explore the diagnostic values of biomechanical mapping of the female pelvic floor.

CONCLUSIONS

The biomechanical mapping of the female pelvic floor with the VTI provides a unique set of parameters characterizing pelvic changes with age and parity. These objectively measurable biomechanical transformations of pelvic tissues, support structures and functions may be used in future research and practical applications.

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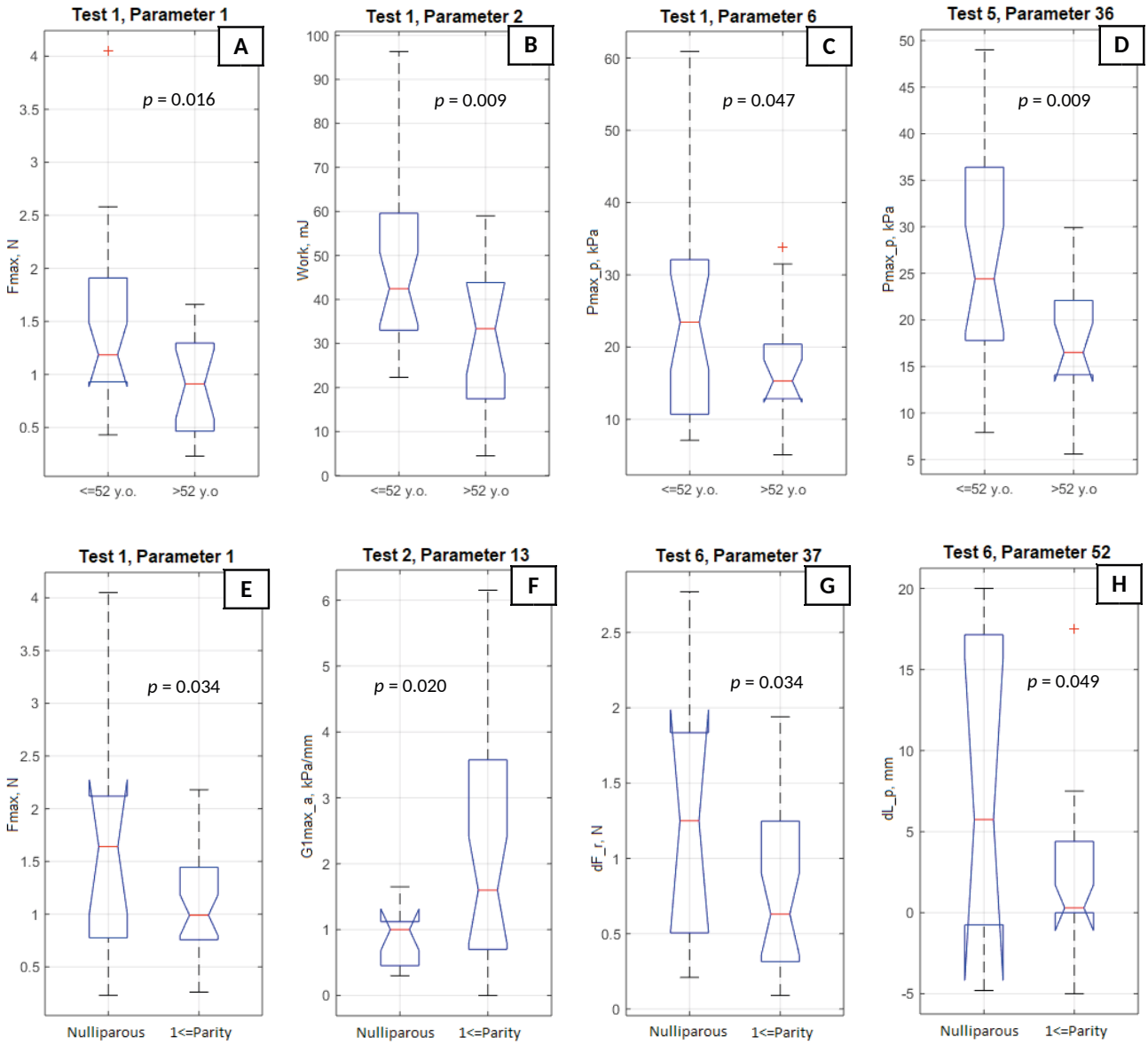

Figure 3. - Boxplots A - D for selected biomechanical parameters for the age groups and boxplots E – H for selected biomechanical parameters for the parity groups.

the authors and does not necessarily represent the official views of the National Institutes of Health.

DISCLOSURE

V. Egorov: CEO and shareholder of Advanced Tactile Imaging, Inc.
H. van Raalte: shareholder of Advanced Tactile Imaging, Inc.

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SUPPLEMENTANTARY MATERIAL (ON-LINE)

Figure 4. - A tactile image acquired during the VTI probe insertion (Test 1) with anatomical landmarks and maximum pressure graphs (green lines, kPa) along anterior and posterior compartments.

Figure 5. - A tactile image acquired during the VTI probe elevation (Test 2) with anatomical landmarks and pressure values at specified locations (see A1-A3 and P1-P3 in Figure 2) along anterior and posterior compartments. The VTI software automatically identified all these 6 locations and shows the pressure values and gradient values (nor shown) for these locations.

Figure 6. - A tactile image acquired during the VTI probe rotation (Test 3) with pressure values at specified locations (see S1 and S2 in Figure 2). The VTI software automatically identified all these 3 locations and shows the pressure values (local maximums) for these locations.

Figure 7. - A dynamic pressure patterns acquired during the Valsalva maneuver for anterior and posterior compartments (Test 4).

Figure 8. - A dynamic pressure patterns acquired during the voluntary muscle contraction for anterior and posterior compartments (Test 5).

Figure 9. - A dynamic pressure patterns acquired during the voluntary muscle contraction for left and right vaginal compartments (Test 6).

Figure 10. - A dynamic pressure patterns acquired during the involuntary muscle relaxation for interior and posterior compartments (Test 7).

Figure 11. - A dynamic pressure patterns acquired during the reflex contraction (cough) for anterior and posterior compartments (Test 8).

Systematic Review

Fowler's syndrome and posterior fornix syndrome play a similar dysfunction. A systematic review

DIANA BADIU[1], ELVIRA BRATILA[2]

[1]*Faculty of Medicine, "Ovidius" University of Constanta, Romania*
[2]*Department of Obstetrics&Gynecology, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania*

Abstract: *Introduction*. In the present review, based on the literature data search, we suggest that urinary retention or obstructed micturition in Fowler's syndrome (FS) and posterior fornix syndrome (PFS) could be functionally related disorders, consistent with both syndromes being a consequence of laxity in the uterosacral ligaments (USLs). *Methods*. This review was conducted by screening and gathering results of research papers from PubMed and Web of Science databases. External sources were not used. Relevant studies were searched by using keywords algorithm: 'posterior fornix syndrome of the bladder' [All Fields], 'urinary retention' [All Fields] AND 'Fowler's syndrome' [All Fields].
Results. After searching 'posterior fornix syndrome of the bladder', and 'urinary retention and Fowler's syndrome' keywords, in Pubmed we have found 45 articles from which 21 were abstracts and from Web of Science, we have found 48 articles from which 37 were abstracts. After including 9 articles from Pubmed and 7 from Web of Science, 4 similar articles were excluded regarding FS but with other causes than urinary retention. In the end, we gather only 7 full-articles from Pubmed and 5 from Web of Science. From the total of 12 full-text articles included from both databases, 4 were common articles in both databases and the final number of eligibility articles was 8. *Conclusions*. Finally, based on literature screening, and hand searches of results from USL repair papers, confined to the discussion section of the paper, we sustain the fact that both FS and PFS could have similar underlying abnormalities in terms of ligamentous dysfunction and, based on these similarities, both were potentially curable by repairing the USLs (i.e. posterior intravaginal sling).

Keywords: Urinary retention; Posterior fornix syndrome; Fowler's syndrome; Uterosacral ligaments; Urge incontinence; Chronic pelvic pain; Nocturia.

INTRODUCTION

Fowler's syndrome (FS) is a disorder described by Clare Fowler in 1988[1]. Its essential features were urinary retention in young women with polycystic ovaries and aberrant electrical activity in the rhabdosphincter (RS). The condition was first assumed to develop from urinary RS spasm due to aberrant electrical activity, presenting complex repetitive discharges and repetitive motor unit bursts[2]. However, it was demonstrated that electromyography (EMG) activity occurs also in healthy women[3]. This finding invalidated one fundamental aspect of traditional FS. From the anatomical point of view, RS is a small and weak muscle from the middle 1/3 of the urethra, which doesn't encircle all the urethra[4]. In this respect, we concluded that this muscle was not capable of closing alone the urethra, especially when it becomes severely atrophied with age, as demonstrated by Huisman[5]. Huisman placed electrodes in the pelvic floor and dorsal striated urethral RS muscle. In this way, he assessed EMG activity during standing, straining, using unilateral and bilateral pudental nerve blockade. After unilateral pudendal nerve blockade, he showed that EMG activity in RS still continued, maintaining the intraurethral pressure[5]. However, with bilateral blockade, there was a fall in urethral pressure and massive urine loss, despite a 500% increase in RS EMG activity[5]. This experiment demonstrated that the RS was responsible for maintaining urethral pressure, but not continence[5].
More recently Petros and contributors used previous urodynamic data in 24 patients who had symptoms of urinary retention and post-void residual of 100ml and other symptoms fitting a diagnosis of posterior fornix syndrome (PFS)[6]. PFS was first reported in 1993 and was attributed to uterosacral ligament laxity[6]. PFS consists of 4 main symptoms, urinary retention (and high post-void residuals), nocturia, urgency, chronic pelvic pain. Petros and contributors were able to check every reported characteristic of FS save RS EMG which was not performed. They demonstrated marked similarities between FS and PFS[6].

METHODS

Literature Search

This systematic review was conducted by screening and gathering results of research papers from literature search in PubMed and Web of Science databases. External sources were not used except in the discussion section at the end to further validate the conclusions reached from the results of the systematic review. Relevant studies were searched by using keywords algorithm: 'posterior fornix syndrome of the bladder' [All Fields], 'urinary retention' [All Fields] AND 'Fowler's syndrome' [All Fields].

Inclusion and Exclusion Criteria

The articles from the database with the keywords input were screened and analyzed: by using 'urinary retention and Fowler's syndrome' in PubMed were 44 articles and from Web of Sciences 48 articles. By using 'posterior fornix syndrome of the bladder' we have found only 1 full-article in PubMed. Another 2 abstracts in PubMed and 3 abstracts in Web of Science were found and were excluded.
By using PRISMA guidelines[7] we have followed the next criteria: (i) original articles; (ii) published in English language; (iii) published within year 2000-2018; (iv) obstructed micturition/urinary retention; (v) overactive bladder; and (vi) surgical revision. We excluded conference abstracts, letters, and review articles.

Data Extraction

We gathered all of the full-text articles that met the inclusion criteria. The results from 8 research articles that were relevant to this review were extracted and analyzed. The outcomes of the studies were comprehensively analyzed.

RESULTS

Study characteristics

Since by searching the words like 'posterior fornix syndrome of the bladder', we found only 1 full-text article

from PubMed database (besides 2 abstracts which were common in PubMed and Web of Science and 1 abstract found only in Web of Science), we restricted further to the words implying 'urinary retention and Fowler's syndrome'. Therefore, from PubMed we found 45 articles from which 21 were abstracts and 1 without any abstract. From 23 full-text articles, we excluded 10 articles (i.e. 3 review articles and 5 case reports, 1 which was a correspondence and 1 as reply article). After remaining 13 full-articles we excluded 4 articles (i.e. 1 article written in Portuguese, 1 in German language, 1 book of the Effective Heath Care Program and 1 article with other causes). The number of eligibility articles from PubMed was 9.
Searching from Web of Science, we found 48 articles from which 37 were abstract (i.e. 31 only abstracts, and 6 conference abstracts). From 11 free full-text from publisher, we excluded 4 articles (i.e. 1 review, 1 case report, 1 Editorial, and 1 Correspondence). The number of eligibility articles from Web of Science was 7.
After including 9 articles from PubMed and 7 from Web of Science, 4 similar articles were excluded regarding FS but with other causes than urinary retention (i.e. menstrual cycle and onabotulinumtoxinA treatment). In the end, we gather only 7 full-articles from PubMed and 5 from Web of Science. Furthermore, we did not find any conference abstracts. From the total 12 full-text articles included from both databases, 4 were common articles in both databases and the final number of included articles was 8 (Figure 1 and Table 1). Studies that were used in this review where focused on the common features of PFS, urinary retention and FS. Moreover, the studies used in this review have a low to moderate level of certainty.

Figure 1. Methodology for articles selection according to PRISMA review guidelines.

In the paper of Mehmood & Altawell, it was showed the safety and efficacy of sacral neuromodulation (SNM) in patients with idiophatic nonobstructive urinary retention on 27 patients. The results showed that 88.8% of the patients presented a 50% improvement in symptoms and underwent permanent device implanted. From the total patients, 17 patients presented adverse effect during follow-up (i.e. box site infection, box site pain, leg pain, implant migration, stroke, urinary tract infection, pelvic/urethral pain and undesirable sensation)[8].

Kavia and contributors[9] examined brain responses to bladder filling in 6 women with FS treated with SNM. The data collected from patients showed that brain responses to bladder filling are abnormal in FS. This could be due to the primary abnormality of the overactive urethra and shows strong inhibitory afferent signals, blocking bladder afferent activity. The consequences were loss of the bladder sensation and ability to void. SNM showed to act by blocking inhibition by urethral afferents[9].

References	Obstructed micturition/urinary retention	Overactive bladder	The main complication: surgical revision
(8)	27 patients with urinary retention after SNM* treatment	-	24 patients
(9)	6 patients with FS** and SNM treatment	Having overactive bladder	Without complications: Small no of patients
(10)	60 patients with urinary retention of FS and SNM treatment	-	53% of the patients
(11)	247 patients with urinary retention, which 44 had test for SNM treatment	Having overactive bladder	45% of the patients
(12)	217 patient with urinary retention and SNM treatment		41% of the patients
(13)	26 patients with urinary retention and SNM treatment		54% of the patients
(14)	8 patients with urinary retention and SNM treatment		Without any complication data
(6)	24 patients with urinary retention, FS or PFS***	Having unbalanced bladder neck	Without any complication data

**SNM= sacral neuromodulation; **FS= Fowler's syndrome; ***PFS= posterior fornix syndrome.*

Table 1. Comparison of the included full-text articles.

Another study reported a 10-year experience of sacral neurostimulation (SNS) in 60 patients with urinary retention. About 53% of the women required a surgical revision. The authors showed that SNS could sustain for long term efficacy, but the procedure has a signification complication rate[10].
Into another study, 247 women with complete or partial retention were referred to different investigations like urethral pressure profilometry, trasvaginal ultrasonographic measurement of the sphincter volume or sphincter EMG. These investigations help authors to classify the cause of retention. The common diagnosis was FS in which SNS was the only intervention that restores voiding[11].
Peeters et al. studies 217 patients who received an implantable pulse generator between 1996 till 2010. The success and cure rates were almost 70% and 20% for urgency incontinence. SNM appears effective in the long-term application, although the re-intervention rate was higher with most occurring in less than 2 years after implantation[12].
Dasgupta and contributors analysed 26 patients with urinary retention and treated by using implantation of a sacral nerve stimulator. Fourteen patients (54%) required revision surgery and the most complications included loss of efficacy, and implant-related discomfort. The authors concluded that SNM is effective for up to 5 years implantation having a significant complication rate[13].
Another study analysed 8 patients with FS and 8 healthy controls which underwent brain imaging with positron emission tomography in order to identify regions of brain activity relating to the perception of bladder fullness and their modulation by SNM. The neuroimaging data showed the existence of abnormal interaction between brainstem and cortical centers in women with urinary retention. Therefore, the authors showed evidence that the therapeutic effect of SNM is achieved through restoration of activity associated with brainstem autoregulation and attenuation of cingulated activity[14].
In the study of Petros and contributors, idiopathic urinary retention and other known FS descriptions in 24 women treated for PFS were evaluated by applying the reinforcement of the USL using tissue fixation system. The authors showed that functional disorders typical of FS, which is also presented in PFS, especially urinary retention can be cures by USLs sling repair[6].
From all this data presented, although SNM represent the single available treatment especially for urinary retention, after long-term follow-up, the recurrence rate showed to be higher, requiring another procedure to be developed.

DISCUSSION

Since obstructed micturition, pelvic pain, and overactive bladder constitute 3 of the 4 core features of the PFS and also 3 of the major features of FS, this suggests that urinary retention or obstructed micturition in FS and PFS are highly likely to be functionally related disorders, dependent on the incompetence of the USLs. Therefore it could be predicted, that treatment with reinforcement of USLs would most likely cure these FS symptoms, exactly as it cures PFS symptoms. We believe the evidence of clinical similarity[6] is such as to recommend USL repair in young women, even by native tissue ligament repair prior to using a far more expensive and invasive option, like SNM technique.
We suggest that, since the urethral RS spasm theory for FS has invalidated[8], the two syndromes may have similar underlying abnormalities in terms of USLs dysfunction or incompetence. Furthermore, by using a posterior intravaginal sling (PIVS) (infracoccygeal sacropexy) or tissue fixation system (TFS) USL repair (in older women), or even, native tissue cardinal ligament (CL)/USLs plication (in young women), it can be confidently expected, that a positive cure rate can be achieved for the symptoms.
The sympathetic fibers in the structure of the hypogastric nerves as well as the parasympathetic fibers from the S2-S3 level explain the symptoms of FS and PVS. Depending on the irritated fibers proportion, symptomatic or parasympathetic - urine retention (sympathetic nervous nerve irritation) or urinary imperiality (parasympathetic fibers) predominates.
The symptom of urinary imperiality caused by irritation of the parasympathetic threads (S2-S4) is different from the overactive bladder syndrome. In the overactive bladder we can see in urodynamic assessment uninhibited detrusor contraction, but in irritation of parasympathetic fibers we will have the same symptom but without detrusor contraction-named hypersensitivity of the bladder.
Although the symptoms of the two syndromes are common, we must bear in mind that it were described at different age groups, women with different pathologies - FS (in young women) and PVS (in women with prolapse). This was possible because these symptoms occur by irritating sympathetic and parasympathetic fibers in the hypogastric plexus. The irritation of sympathetic and parasympathetic threads can be determined by different pathologies that produced damage or laxity of the CL and USL support system. Thus, both ligaments can be affected by infiltrative processes (i.e. endometriosis) or the process of laxity of their conjunctive

tissues. Regardless of the pathogenic mechanism that affects USLs, the pelvic nerve structures found in the vicinity of these ligaments are chronically irritated, causing symptoms such as urinary imperiality, chronic pelvic pain, or difficulty in bladder evacuation.

As a result, the release of these nerves from the periligamentar adhesion syndrome in endometriosis or the reinforcement of weak ligaments that stabilize the constant irritation of the nerve threads causes the cure of the symptoms included in the 2 syndromes.

Considering these aspects, it is explicable why SNM treatment does not have very good results, modulating the activity of the S1-S3 thread, without influencing the sympathetic threads of the lower hypogastric plexus, threads originating from T12-L5.

PIVS in USL reinforcement for FS and PFS

In order to treat symptoms like chronic pelvic pain, emptying problems, or nocturia which could offen appear in PFS, it can be creating a neo-sacrouterine ligaments bridge. This technique can be achieved by using PIVS or CL/USL TFS ligament repair, creating a new collagen for long term sustaining the pelvis[15,16].

Some authors using PIVS posterior sling repair method on 40 patients (i.e. 20 younger than 60 years and 20 older than 60 years) with vaginal vault prolapse, demonstrated that significant improvements in symptoms of nocturia, urgency, fecal incontinence and chronic pelvic pain[15]. The incidence of mesh erosion varied between 0 and 13% after applying the PIVS method[17].

Balsak and contributors[18] showed a 100% surgical success rate by applying PIVS technique on 21 patients with 2, 3 or 4 pelvic organ prolapsed. It was also showed a 33.3% rate of dyspareunia and 14.2% rate of mesh erosion with an improvement in vaginal symptoms and quality of life scores in the postoperative period.

Caliskan et al.[19] used both the original and modified PIVS technique on 368 patients with symptomatic pelvic organ prolapsed in various grade. By using both methods, the authors showed a 97 and 96% apical success rate.

Another study made on fifty-two patients with vault prolapsed symptoms, using the PIVS technique, showed an incidence of 2.1% of mesh erosion in approximately 20 weeks[20].

Limitations and Future Research Suggestions

The limitation of our study consisted in a very low number of full-text articles referring to 'posterior syndrome of the bladder', and indeed FS also, from both databases. Furthermore, we did not find many articles which could include the management of FS and PFS disorders and the outcomes complications after achieving the surgical complications.

CONCLUSIONS

In the present review, we sustain the fact that similar FS and PFS symptoms like urinary retention, pelvic pain and/or bladder fullness could have similar underlying abnormalities in terms of ligamentous dysfunction or incompetence. In this respect, PIVS treatment can be achieved, being a minimally invasive procedure for repairing the USLs.

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Correspondence:

Prof. Elvira Bratila, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania,

E-mail: elvirabarbulea@gmail.com

Review

A practical update on functional and dysfunctional anatomy of the female pelvic floor - Part 1 Function

YUKI SEKIGUCHI [a], HIROMI INOUE [b], RYOKO NAKAMURA[a]

[a]*LUNA Pelvic Floor Total Support Clinic, Women's Clinic LUNA Group, Yokohama, Japan*
[b]*Urogynecology Center, Shonan Kamakura General Hospital, Kamakura, Japan*

Abstract: Background The Integral Theory System considers pelvic organ prolapse (POP), pain, bladder & bowel dysfunctions to be mainly caused by laxity in up to 5 suspensory ligaments and their vaginal attachments. *Aim* To define the role of ligaments in normal function (Part1), then dysfunction and principles of surgical cure (Part 2). *Methods* The role of pelvic ligaments and muscles in normal bladder & bowel closure, evacuation, central and peripheral neurological control is analysed. *Results* Normal function Ligaments stretch minimally, vagina stretches extensively during coughing, straining, squeezing, micturition, defecation. Competent ligaments suspend organs and act as insertion points for 3 striated muscle forces. These act in opposite directions to close and open urethra & anorectum, stretch organs to prevent inappropriate activation of micturition and defecation reflexes. Dysfunction Ligaments must be competent, otherwise the muscles which contract against them lengthen and weaken. A cascade of dysfunctions follow from elongated ligaments: prolapse; muscles cannot close urethral and anal tubes (incontinence) open them (emptying problems) or stretch organs sufficiently to support stretch receptors which may fire off prematurely to activate the micturition reflex (urge incontinence, frequency, nocturia) or defecation reflex (fecal incontinence). *Conclusions* Part 1 demonstrated that competent ligament insertion points are required for the 3 directional forces which control mechanical closure and evacuation and the neurological feedback mechanisms for defecation and micturition reflexes. Lengthening of the sarcomere due to ligament laxity was considered the ultimate link between loose ligaments and dysfunctions in these organs.

Keywords TFS; Integral Theory; Bladder function; Bowel function; Pelvic ligament ; Sarcomere; Vagina; Stress incontinence; Chronic pelvic pain; OAB; Nocturia; Fecal incontinence.

INTRODUCTION

"Precise, almost mathematical knowledge of anatomy is a highly fertile source of surgical applications, suggesting new techniques and helping to perfect and simplify existing surgical methods, making them less mutilating and more benign and, in short, raising surgery to the rank of true science."

It is a fundamental truth that an accurate knowledge of anatomy is a pre-requisite for any surgery to any part of the human body. In conceptualizing this paper, we have been guided by the writings of the great Spanish Anatomist/Urologist Salvador Gil-Vernet (1892-1987) [1] and our own practical experience of application of the Integral Theory System over a collective period of more than 60 years.

STRUCTURE

Five main ligaments suspend the organs from the pelvic girdle. They are pubourethral (PUL), arcus tendineus fascia pelvis (ATFP), cardinal (CL), uterosacral (USL) and the deep transversus perinei portion of the perineal body (PB), figs 1, 2. A 6th ligament, external urethral ligament (EUL) attaches the external urethral meatus to the anterior surface of the pubic symphysis. It mainly controls urethral mucosal sealing[2].

It is the ligaments which provide suspensory strength. The main structural component of ligaments is cross-bonded collagen 1. According to Yamada[3], the breaking strain of ligaments is approximately 300 mg/mm^2. Suspensory ligaments do not stretch significantly during the effort of closure and micturition. This is evident on simple inspection of PUL and USL in the dynamic xrays, fig. 3.

The vagina is a weak elastic organ, breaking strain 60 mg/mm^2 [3]. Its elasticity permits the independent function of the 3 opposite muscle forces (arrows), fig. 3[4]. The vagina's elastic function is self-evident on simple inspection of fig. 3: the vagina is stretched significantly during urethral closure (straining) and micturition. Though the vagina helps to support the bladder, fig. 3, its role cannot be primarily structural as its breaking strain is only 60 mg/mm^2, a consequence of the predominance of collagen 3, a more elastic, but weak collagen. Structurally, the vagina acts like an (elastic) plaster board of a domestic ceiling, with cardinal ligaments, ATFP and cervical ring acting as the joists.

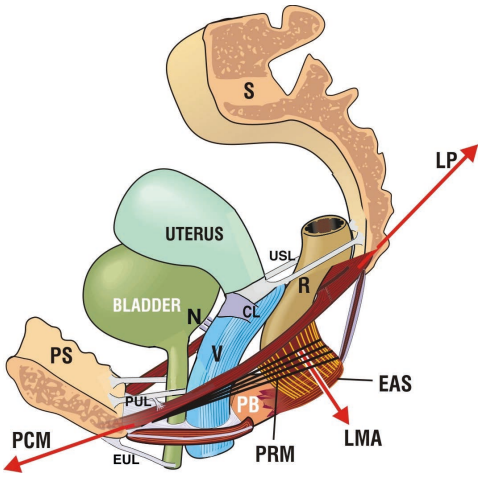

Figure 1. The relationship of ligaments, muscles and function.
The 5 ligaments which suspend the organs PUL= pubourethral ligament; CL=cardinal ligament; USL= uterosacral ligament; PB= perineal body. ATFP = arcus tendineus fascia pelvis); EUL = external urethral ligament is a 6th ligament which attaches the external meatus to the anterior surface of pubic symphysis (PS);
Forward acting muscles: m.pubococcygeus (PCM), m.puborectalis (PRM). PCM contracts against the pubourethral ligament (PUL). PRM contracts only against symphysis pubis.
Backward acting muscles: levator plate (LP) contracts backwards against PUL anteriorly; LMA contracts solely downwards against USLs . N=bladder stretch receptors; R=rectum; EAS=external anal sphincter; PS=pubic symphysis; S=sacrum.

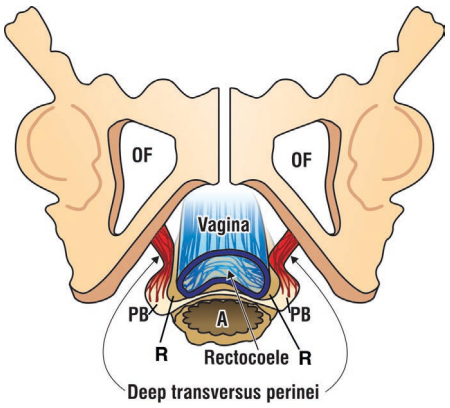

Figure 2. Deep transversus perinei ligaments[3] attach perineal body 'PB' to the descending ramus.
In this figure, the ligaments are elongated. PBs have been separated into two parts during childbirth, by stretching of their central part. This causes the rectum to protrude into the vagina as a rectocele.

The muscles The vagina and rectum rest on the pelvic floor muscles. There are four directional striated muscles vectors, fig1; two are forward acting: m.pubococcygeus (PCM), m.puborectalis (PRM). PCM is attached to distal vagina and contracts forwards against the pubourethral ligament (PUL). PRM contracts only against symphysis pubis. There are two posterior vectors (arrows, fig1): levator plate (LP) and conjoint longitudinal muscle of the pelvis (LMA). LP is attached to the posterior wall of rectum; it contracts backwards against PUL; LMA inserts into the anterior portion of LP proximally and the external anal sphincter distally; it contracts solely downwards against USLs, figs 1,3.

Muscle function
The opposite directional forces, figs 2&3, have three main functions.
1. They create shape and strength of the organs [5].
2. They close the urethral and anal tubes (continence) and help to empty them during micturition and defecation[4].
3. They stretch the vagina to support the bladder stretch receptors 'N', and tension USLs to support the nerve ganglions in the uterosacral ligaments[4].

FUNCTION

A summary of the role of the suspensory ligaments in bladder and anorectal function and dysfunction
The Integral Theory (IT) states that pelvic organ prolapse, symptoms of chronic pelvic pain, bladder and bowel dysfunction are mainly caused by laxity in 5 suspensory ligaments. The ligaments have a dual function: they suspend the organs and act as insertion points for three oppositely acting muscle forces. Lax ligaments weaken these muscle forces so they cannot adequately close the urethral or anal tubes (incontinence), evacuate them (constipation, bladder emptying), or tension the bladder and rectum sufficiently to prevent inappropriate activation of the micturition and defecation reflexes by their stretch receptors (urge incontinence of bladder & bowel). Up to 80% cure/improvement for the above conditions has been achieved following repair of one or more damaged ligaments using precisely positioned TFS tensioned tapes [6-13], Tables 1&2. The Integral Theory states "Repair the structure (ligaments) and you will restore the function". *The same operations are used for patients with major symptoms and minimal prolapse and major prolapse with no symptoms*[9].

Normal bladder function [4,5] (Fig. 3)
The bladder is a storage container for urine. Continence and evacuation are via the urethral tube. The bladder has 3 modes of function, fig. 3.
1. Resting closed mode, fig. 3, middle figure B. Urethral closure is maintained by vaginal elasticity, urethral elasticity/smooth muscle and slow twitch striated muscle contractions against PUL, 'S' acting forwards, backwards, downwards.
2. Effort closed mode fig. 3, left figure A. On effort, fast-twitch forward and backward forces act against PUL in opposite directions (arrows) to close urethra distally and proximally. A 3rd downward force (arrow) pulls down LP. This action rotates the bladder downwards to close (kink) bladder neck.

Table1: Lower und upper 95 %-confidence intervals for the observed relative frequencies of Prolapse, Urgency, Nocturia, Day time frequency, Dragging pain and Fecal incontinence.
Parallelly the results of testing the hypothesis Ho: $p \le p_0$ vs. H1: $p > p_0$ have entered.
' * ' , ' # ' and ' / ' means significant p-values when p_0 is setting equal to 0.80, 0.75 and 0.60, respectively.
With other words these symbols depict that the observed cure rates are significantly higher than 0.80, 0.75 and 0.60 respectively ($p < 0.05$; Binomial Tests)

Variable	N	No of cured	observed cure rate (%)	95 %-lower CI	96 %-upper CI	Test results Ho: $p \le p_0$ vs. H1: $p > p_0$
Prolapse	278	257	92.10	0.891	0.952	*
Urgency	133	124	93.20	0.879	0.971	*
Nocturia	86	62	72.10	0.597	0.809	/
Day time frequency	132	120	90.10	0.935	0.999	*
Dragging pain	56	52	92.90	0.862	0.998	*
Fecal incontinence	52	46	88.50	0.798	0.977	#

TABLE 1. From Inoue et al.[6]

Variable	Number	Number cured	% cure	p value
Pelvic pain	25/79	20	80%	p<0.0001
*Nocturia	37/79	30	81%	p<0.0001
*Urge incontinence	42/79	37	88%	p<0.0001
*Frequency	49/79	42	85%	p<0.0001
Apical prolapse	79	74	93%	p<0.0001

TABLE 2. From Sekiguchi et al.[8]. N=79 - Mean age 68 years - 12 months data

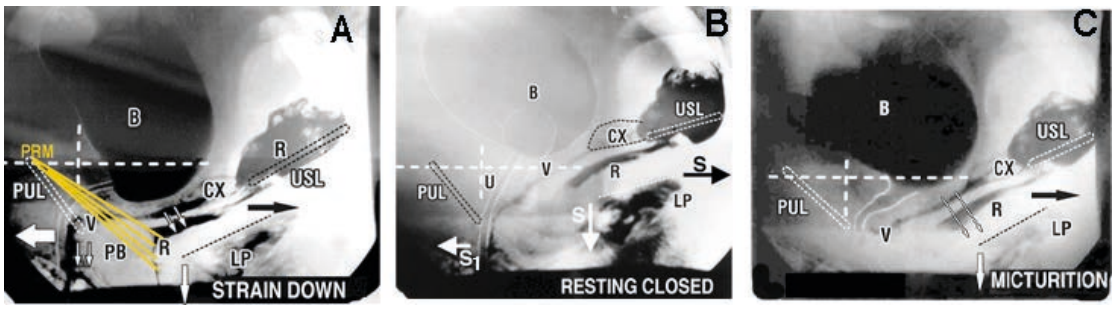

Figure 3 Normal bladder/bowel function Video xray, sitting position. Radio-opaque dye has been injected into the bladder, vagina, rectum, levator plate.
Middle figure B *At rest*, asymptomatic patient. Slow twitch diectional forces 'S' (arrows) stretch the organs bidirectionally against pubourethral ligaments 'PUL' anteriorly and uterosacral ligament 'USL' posteriorly to close urethra and anus and to support the bladder base and rectal stretch receptors, preventing activation of the micturition and defecation reflexes. LP=levator plate; U=urethra; B=bladder; U=urethra; CX=cervix.
Left figure Straining A- *urethral closure* The ligaments do not stretch. They anchor the urethra, distal vagina and rectum. USL is angulated downwards. Fast twitch muscles stretch the distal vagina forwards (arrow) to close distal urethra; backward/downward vectors (arrows) contract against PUL and USL to stretch and rotate the proximal urethra, proximal vagina and rectum, around PUL to effect bladder neck closure.
Left figure Straining A *-anorectal closure* Puborectalis muscle (PRM, yellow) contracts. The same posterior vector forces (arrows) contract against PUL and USL, to stretch rectum 'R' around a contracted PRM and perineal body (PB) to close the anorectal angle and effect anorectal closure.
Right figure C *Micturition* The ligaments do not stretch. USL is angulated downwards. There is absence of a forward vector which has been relaxed by the micturition reflex. The vagina and rectum are stretched backwards and downwards. Fast twitch backward/downward vectors (arrows) contract against USL to open out the posterior wall of urethra, thus vastly reducing the internal resistance to flow.

3. Open (micturition) mode (fig. 3), right figure C. The forward arrow, S1, fig B, relaxes. Two fast-twitch directional forces* pull the posterior wall of urethra backwards. Bladder contracts to empty.
Note relative immobility of PUL and USL in fig. 3 and very significant stretching of the vagina in both closure and micturition.

Normal anorectal function
Bladder and bowel have similar closure and opening mechanisms[14].
The rectum is a storage container for feces. Continence and evacuation are via the anal tube. The anorectum has 3 modes similar to those of the bladder in fig. 3[14].
1. Resting closed mode, is maintained by slow twitch striated muscle contraction, organ elasticity/ smooth muscle (figs. 3&4).
2. Effort closed mode is activated by 3 fast-twitch directional forces during straining or coughing (fig. 3) (left frame). The uterosacral ligaments have a key role in anorectal closure. The USLs are attached to the lateral rectal walls by thin ligamentous attachments. LP stretches the rectum backwards against PUL to tension it prior to LMA contraction, fig4. LMA contracts against USL to pull down the anterior part of LP. This downward angulation rotates the rectum around a contracted puborectalis (PRM) to 'kink' the rectum and close the anorctal angle[14]. If either PUL or USL are loose, closure may not occur (fecal incontinence 'FI'). Hocking reported cure of double incontinence USI and FI with repair only of PUL with a midurethral sling[15]. The Kamakura group reported cure of FI with a cardinal/ uterosacral sling operation Table 1
3. Open (defecation) mode is an active process[14]. Defecation is activated by 3 fast-twitch directional forces, LP/LMA posteriorly, fig4 and a forward force possibly PCM or puboanalis acting on the anterior anal wall, fig. 5, forward arrow. With reference to fig. 5, the anterior border of the levator plate has been pulled downwards apparently by a downward vector. Levator plate 'LP' is clearly shown attaching to the posterior wall of rectum. It contracts backwards towards the coccyx. The posterior wall of the rectum has been stretched downwards and backwards (red arrows) apparently by the resultant of these two vectors, The anorectal angle 'ARA' descends into the light green rectangle at 45° The opening extends all the way down the posterior wall of the anus. The anterior wall of rectum has been pulled forwards to further open out the anal canal.
The USLs have a key role in defecation. The USLs are attached to the lateral walls of rectum; the downward vector (arrow), fig. 5, contracts directly against the USLs during

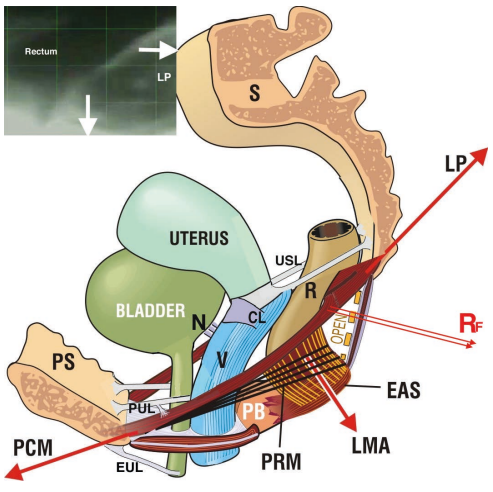

Figure 4 Anorectal closure and defecation
Closure (continence) LP/LMA stretch and rotate the rectum (R) around a contracted PRM to 'kink' the rectum closed. 'RF' is the resultant of the two forces LP/LMA and acts as the rotating vector to create the anorectal angle. See also 'straining' xray fig. 3.
Opening (defecation) PRM relaxes, LP/LMA (RF) contract to open out the anorectal angle ; rectum 'R' contracts to empty.The broken orange vertical lines behind the rectum 'R' indicate rectal position during "OPEN" mode (defecation) : Upper left corner X-ray myogram defecation mode. Anorectal angle opened out.

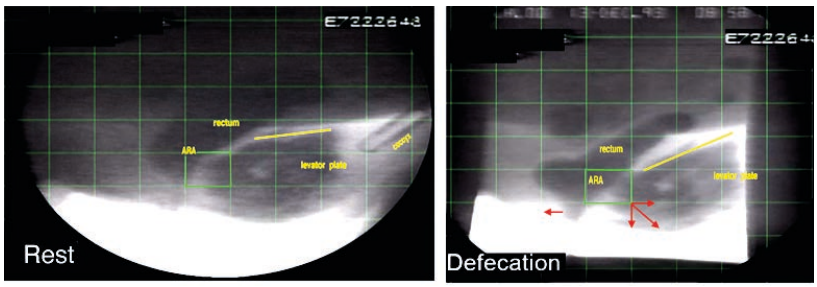

Figure 5. X-ray defecating procto-myogram
At rest The anorectal angle 'ARA' to the left of the green square is angled. The anus is closed. The superior surface of the levator plate muscle is almost horizontal.
Defecation mode ARA is opened out by backward and downward vectors LP/LMA (arrows). The anterior wall of anus is pulled forwards (arrow) further opening out the anal canal. Note the insertion of levator plate into the posterior wall of rectum. The resultant vector action (arrow) seems to be opening out ARA. The downward angulation of the anterior border of levator plate is identical with what happens during micturition[14]; conjoint longitudinal muscle of the anus (LMA) pulling down the anterior margin of levator plate (LP).

defecation. A loose USL will weaken the contractile strength of a striated muscle potentially leading to inability to properly close or evacuate. The patients senses this as "constipation: and the physician as "Obstructive Defecation syndrome' (ODS).

Control of the micturition, defecation reflexes and pain - According to the Integral Theory, urge incontinence, nocturia (OAB) even urodynamic 'detrusor overactivity' are expressions of a prematurely activated but otherwise normal micturition reflex[4]. This concept was urodynamically validated in 1993: it was demonstrated that the events which occur during micturition and the 'unstable bladder' ('OAB', 'DO') are identical[16]: 1 Sensation of urge; 2. Fall in proximal urethral pressure; 3. Rise in detrusor pressure: 4. Urine loss. The bidirectional stretching of the vagina (fig. 6), acts like a trampoline to support the stretch receptors 'N' at bladder base; as the hydrostatic pressure of urine rises, spindle cells in the oppositely acting muscles automatically cause the muscles to stretch the vagina to support 'N', thus preventing activation of the micturition reflex. A similar feedback system applies to the anorectum.
It is hypothesized that the nerve plexuses in the distal parts of the USLs, the Frankenhauser and Sacral plexuses, are similarly controlled: the opposite muscle stretching tensions the USLs to support the nerves preventing them from firing off.

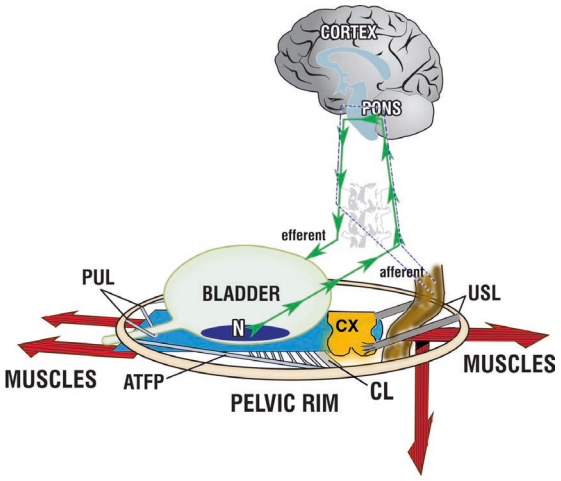

Figure 6. The trampoline analogy.
In the normal patient, the stretch receptors 'N' sense bladder fullness and send afferent signals to the cortex (green afferent arrows); afferent signals are controlled centrally and peripherally by opposite stretching of the vaginal membrane by muscle forces (arrows); the stretched vagina supports the hydrostatic pressure exerted by the urine column. At a critical point, the afferent signals (green arrows) activate the micturition reflex which causes the forward muscles to relax; the posterior muscles contract to open out the posterior urethral wall; the detrusor contracts to empty4. A similar feedback control system, opposite stretching of the posterior vectors and puborectalis supports the anorectal stretch receptors (continence). Increased afferent impulses (green arrows) activate the defecation reflex, relax puborectalis, contract the posterior vectors to open out the anorectal angle, contract the rectum to empty.

Gordon's Law- the ultimate pathway for understanding how ligament looseness may cause muscle dysfunction and symptoms.
Gordon's Law (fig. 7) is the key to understanding the causation of bladder and bowel function and dysfunction. It states *"A striated muscle contracts optimally over a short length only* ('E', fig. 7). If the ligaments against which the 3 vector muscles contract are firm, the muscles contract efficiently over a length 'E', fig. 7. However, *Lengthening the contractile length results in a rapid loss of contractile strength*."[17].
If the ligaments against which the three vector muscles contract lengthen by 'L", the muscles lengthen accordingly and their contractile strength weakens[4], from a nominal 80% to 30%, fig. 7.

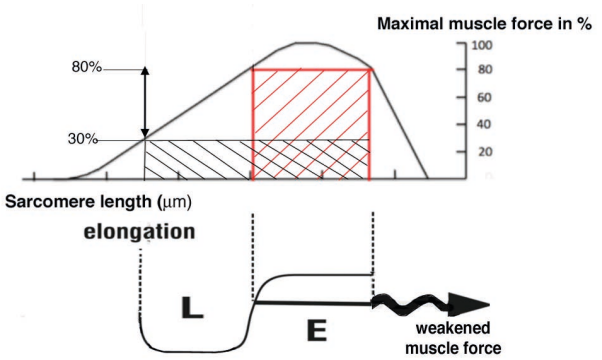

Figure 7. Gordon's Law.
A striated muscle contracts optimally over a short length only, 'E', red square. Lengthening the muscle 'L', results in a rapid loss of contractile force, black rectangle.

CONCLUSIONS

Part 1 examined the key role of competent ligament insertion points for the 3 directional forces which control normal bladder, bowel & nerve function. Part 2 builds on Part 1 to explain which ligaments may be causing the dysfunctions and how ligament shortening can reverse the cascade of events which led to the dysfunction

DISCLOSURES

Conflicts of Interest: None
IRB approval: Not required
Contributions to the article: Conceptualization writing: all authors; Figures Tables YS, HI;
Acknowledgments: Dr Liedl for permission to use fig. 7.

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Correspondence to:
Yuki Sekiguchi
3-115 Hyakudan -kan 3F Motomachi Nakaku - YOKOHAMA 232-0861 - Japan, E-mail: dumbo-ys@d9.dion.ne.jp

Review

A practical update on functional and dysfunctional anatomy of the female pelvic floor - Part 2 Dysfunction

YUKI SEKIGUCHI [a], HIROMI INOUE [b], RYOKO NAKAMURA[a]

[a]*LUNA Pelvic Floor Total Support Clinic, Women's Clinic LUNA Group, Yokohama, Japan*
[b]*Urogynecology Center, Shonan Kamakura General Hospital, Kamakura, Japan*

Abstract: Background The Integral Theory System considers pelvic organ prolapse (POP), pain, bladder & bowel dysfunctions to be mainly caused by laxity in up to 5 suspensory ligaments and their vaginal attachments. *Aim* To define the role of ligaments in normal function (Part1), then dysfunction and principles of surgical cure (Part 2). *Methods* The role of pelvic ligaments and muscles in normal bladder & bowel closure, evacuation, central and peripheral neurological control is analysed.
Results Normal function Ligaments stretch minimally, vagina stretches extensively during coughing, straining, squeezing, micturition, defecation. Competent ligaments suspend organs and act as insertion points for 3 striated muscle forces. These act in opposite directions to close and open urethra & anorectum, stretch organs to prevent inappropriate activation of micturition and defecation reflexes. Dysfunction Ligaments must be competent, otherwise the muscles which contract against them lengthen and weaken. A cascade of dysfunctions follow from elongated ligaments: prolapse; muscles cannot close urethral and anal tubes (incontinence) open them (emptying problems) or stretch organs sufficiently to support stretch receptors which may fire off prematurely to activate the micturition reflex (urge incontinence, frequency, nocturia) or defecation reflex (fecal incontinence). Conclusions Part 1 demonstrated that competent ligament insertion points are required for the 3 directional forces which control mechanical closure and evacuation and the neurological feedback mechanisms for defecation and micturition reflexes. Lengthening of the sarcomere due to ligament laxity was considered the ultimate link between loose ligaments and dysfunctions in these organs.

Keywords Loose ligaments; Integral Theory; Stress incontinence; TFS; Chronic pelvic pain; OAB; Nocturia; Fecal incontinence.

INTRODUCTION

Part 1 analysed the crucial role of pelvic suspensory ligaments in normal structure and function of bladder and bowel. Key to normal closure (continence) and opening (emptying) of the urethra and anal tubes were three oppositely acting directional muscle vectors. These contracted against firm suspensory ligaments. The same oppositely acting muscle forces controlled urge incontinence by stretching the organs to provide a firm support for peripheral stretch receptors which control the micturition and defecation reflexes.
The aim of Part 2 is to analyse the anatomical pathways from childbirth, to loose ligaments, to prolapse, to organ dysfunction, to a ligament based diagnostic system and finally, ligament based TFS surgical cure.

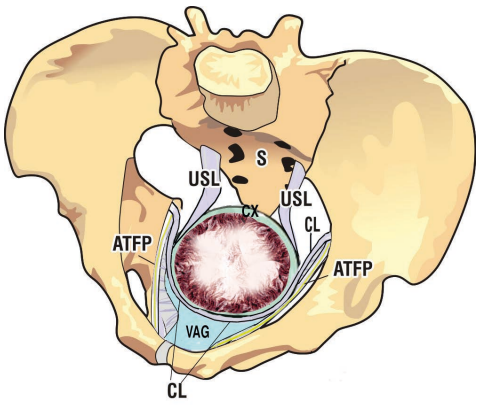

Figure 1. Ligaments are stretched by the head at full dilatation (10 cm). Birth pressure at 10cm dilatation on the cervical ring may overstretch uterosacral ligaments (USL) and cardinal (CL) ligaments. Extension or tearing of these attachments and vagina (vag) from cervix (CX) may cause uterine/apical prolapse, cystocele anteriorly, high rectocele and enterocele posteriorly. The perineal body may be damaged and separated to cause low rectocele (perineocele) and descending perineal syndrome as the head exits the birth canal.

Pathways to ligament elongation and prolapse

Fundamental to the Integral Theory's concept of dysfunction is ligament elongation and weakening mainly from childbirth, age and menopause[1].
Collagen depolymerizes to lose 95% of its strength prior to labour, allowing the cervical ring, ligaments and vagina to stretch considerably during birthing, fig1. The pelvic diameter is only 12-13 cm. Full cervical dilatation is at 10cm, where very considerable tissue stretching occurs: ligaments and vagina stretch, are pushed laterally and may remain stretched post-partum; vaginal attachments may rupture. The end result may be prolapse and dysfunction of bladder/bowel and chronic pain.

Pathway to uterine prolapse pathogenesis and surgery

Fig. 2 is self -explanatory. Uterine prolapse is clearly caused by weak and elongated cardinal (CL) and uterosacral ligaments (USL), not vagina. The only way uterine prolapse, fig. 2. can be cured is to shorten and strengthen CL and USL. 'Native tissue repair' of the vagina will not strengthen the ligaments, nor will a mesh. The ultimate proof that the vagina is not a structural organ was recently provided by the Lancet Prospect RCT[2], where native tissue repair of the vagina was compared against vaginal mesh repair. Both methods had >80% failure rate at 6 months. In contrast, The

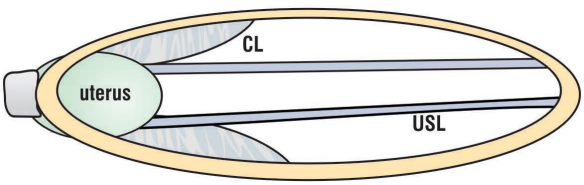

Figure 2. Both CL and USL are elongated in uterine prolapse.
It is self-evident that loose cardinal (CL) and uterosacral (USL) ligaments will elongate to cause uterine prolapse, and that only shortening and strengthening

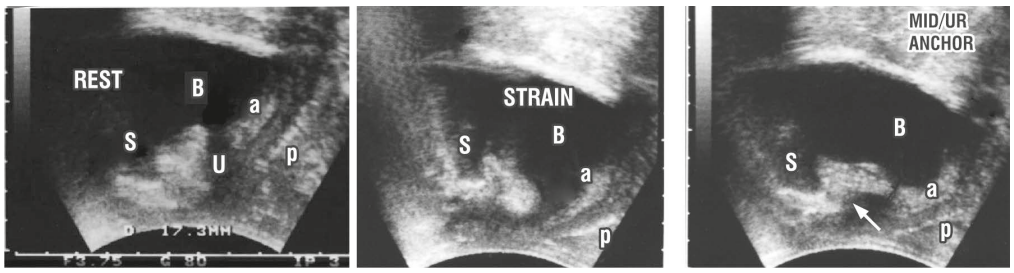

Figure 3. Direct ultrasound proof of Gordon's Law applied to lax pubourethral ligament 'PUL'[10].
Left figure, 'REST' S=symphysis; U=urethra; B=bladder; a=anterior vaginal wall; b=posterior vaginal wall.
Middle figure 'STRAIN' Inability of PUL to support posterior urethra allows the posterior pelvic muscles to stretch the vaginal walls 'a' and 'b' backwards; this action pulls open the posterior urethral wall. The urethra opens out proximally (funnelling) and distally.
Right figure 'MID/UR ANCHOR' When the PUL is shortened by pressing upwards with a hemostat (arrow), the strength of the muscle forces is restored; 'a'&'b' visibly tension; distal and bladder neck closure are restored.
Anatomical pathway to the above events In the middle figure (lax PUL), all 3 directional forces which act on PUL lengthen and lose contractile strength. Once PUL length is restored by the hemostat (white arrow), all 3 muscles can now 'grip' and close the distal urethra and bladder neck (right image). By permission of Prof P Petros.

Kamakura[3] and Yokohama[4] units reported surgical >90% cure rate for 3rd and 4th degree POP at 12 months, falling to 79.0 at 60 months[5] following TFS ligament repair.

Anatomical pathway to cystocele pathogenesis and surgery

The attachment of CL and anterior vaginal wall to the anterior cervical ring (fig1), may be dislocated or torn to cause a 'transverse defect' or 'high cystocele'. In our experience this defect is responsible for 80% of all cystoceles. A cardinal ligament TFS sling simultaneously shortens and reinforces CL and 're-glues' the anterior vaginal wall to the cervical ring[6]. If the cystocele defect is more extensive, for example a central defect due to either a dislocated pubovisceral muscle or from a lax ATFP, the TFS ATFP 'U Sling' can re-attach the muscles and tissues distally to the ATFP insertion point just behind symphysis pubis[7].

Anatomical pathway to rectocele causation and repair

The perineal body 'PB' supports the lower half of the posterior vaginal wall[8]. PB is suspended from the descending ramus by the deep transversus perinei ligaments. If these stretch or are torn, PB stretches and the rectum protrudes. The TFS PB repair shortens and tightens the deep transversus perinei ligaments; the laterally displaced parts of PB are approximated; the rectum is displaced back to its normal position.

Gordon's Law- the ultimate pathway from ligament looseness to muscle dysfunction and symptoms

The theory states that it is mainly loose ligaments which cause chronic pain, bladder & bowel dysfunction. Part 1 demonstrated how three opposite striated muscles contract against the suspensory ligaments to open or close the urethral and anal tubes. These directional muscle actions are co-ordinated by the cortex as part of the neurological reflexes which close urethra and anus (continence) and evacuate them, (micturition and defecation reflexes). A loose ligament effectively lengthens the muscles which contract against it. This weakens the muscle force according to Gordon's Law.
Gordon's Law is the key to understanding the causation of chronic pelvic pain, bladder and bowel dysfunction. It states *"A sarcomere contracts optimally over a short length only. Lengthening or shortening the contractile length results in a rapid loss of contractile strength"*[9].
Because a ligament is the effective insertion point of the muscle, if the ligament is loose, the muscle lengthens accordingly and its contractile strength weakens.

Ligament length is critical for anatomy and function- a practical objective demonstration of Gordon's Law

A hemostat (arrow, right frame, fig. 3) pressed upwards immediately behind the symphysis 'S', effectively shortens PUL, prevents the funneling seen in the middle frame, restores the contractile strength of the muscle vectors to close the urethra and bladder neck, right frame, fig. 3.
Fig. 3 objectively demonstrates the anatomical pathway to continence control by midurethral hemostat pressure demonstrated and predicts cure by the midurethral sling operation.

Muscle or ligament- which is the main cause of symptoms?

A blinded study in 47 patients[11], 46 with histologically proven pubococcygeus muscle damage had a midurethral sling procedure which involved a sling placed at midurethra to reinforce the pubourethral ligament 'PUL'; 89% became continent on the day after the procedure, suggesting that the cause of the problem was ligament rather than muscle related.

The anatomical pathway to urinary stress incontinence, low urethral pressure and fecal incontinence.

Fig. 4 demonstrates the effect of a loose pubourethral ligament 'PUL' when it lengthens from normal length 'E' by 'L'. According to Gordon's Law, if PUL is loose, the intraurethral area 'U' will expand; the force of the closure muscles m.pubococcygeus (PCM) will weaken and the urethral tube 'U' cannot be adequately closed. The rhabdosphincter (which is responsible for urethral pressure measurement) cannot contract sufficiently, so a low maximal urethral closure pressure 'MUCP' or Valslava Leak Point Pressure 'VLPP' may be recorded, as a consequence of enlarged intraurethral area (Pressure = Force/Area). The intra urethral resistance to intra abdominal pressure increase is lowered exponentially, inversely by the 4th power of the radius (Poiseuille's Law) and the patient may lose urine on effort.
We propose that a similar scenario applies for anorectal closure: if PUL or USL are loose, the muscle vectors which contract against them weaken, the anal tube cannot be closed and feces may leak (fecal incontinence).

The anatomical pathway to 'mixed incontinence'

'Mixed incontinence' is co-occurrence of USI and urge incontinence. Urinary stress incontinence (USI) and urge are two entirely different symptoms. USI is caused by PUL laxity. When USI and urge co-occur, if the cause for the urge is inability of the forward vectors to support the stretch re-

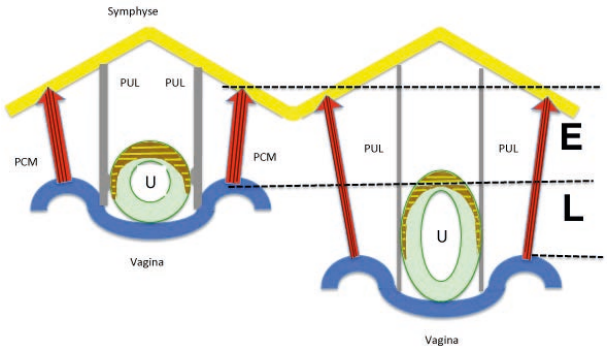

Figure 4. Stress incontinence and ISD interpreted by Gordon's Law. Schematic coronal view at midurethra.
Left figure: continent patient. The pubourethral ligament 'PUL' is of normal length 'E', as is the pubococcygeus muscle (PCM) and rhabdosphincter.
Right figure: patient with urinary stress incontinence PUL is loose, elongated by length 'L'. PCM and rhabdosphincter secondarily elongate to 'L' and lose contractile strength. U= urethral cavity.

ceptors because of PUL laxity, urge will also be cured by a midurethral sling. This explains simultaneous cure of urge in 50-60% of patients having a midurethral sling for USI. If the cause of urge is lax cardinal or uterosacral ligaments constipation (see algorithm, fig. 10), the urge won't be cured simultaneously with USI cure. In that instance, patients will likely have other posterior symptoms such as pain, abnormal emptying, nocturia.

The anatomical pathway to 'double incontinence'
'Double incontinence' is co-occurrence of USI and fecal incontinence. If PUL is loose, the directional closure forces may weaken to cause USI and sometimes fecal incontinence also, because the LP part of the LP/LMA vectors which close the anorectal angle contracts against PUL. Hocking demonstrated cure of both conditions by shortening and reinforcing PUL with a midurethral sling in 90% of cases[12].

The different pathways to retropubic and transobturator midurethral sling closure
Though many studies indicate that the transobturator approach for midurethral sling surgery gives equivalent results for cure of urinary stress incontinence as the retropubic approach, it is generally acknowledged that the retropubic is superior in patients with ISD (intrinsic sphincter defects) or those requiring repeat surgery for USI. A midurethral retropubic tape automatically grips the lateral sides of the urethra before proceeding behind the symphysis (PS), fig5; a retropubic tape will reinforce PUL and the pubovesical ligament (PVL) to restore both distal and bladder neck closure mechanisms as proven by transperineal ultrasound in fig. 3. The bladder is rotated around the precervical arc of Gilvernet by LP/LMA vector forces to effect bladder neck closure at '0-0'.
The TOT provides an anchoring point at the base of PUL for the posterior rotating closure forces LP/LMA, fig5. The TOT relies on an intact PVL and precervical arc for optimal bladder neck closure, fig5. However, a TOT cannot repair a damaged PUL or PVL, nor can it grip the sides of the urethra, potentially diminishing the traction required to fully enact the two closure mechanisms, distal and proximal. As the ultimate pathway to continence is exponentially determined by the internal resistance to flow by Poisseille's Law (inversely by the 4th power of radius narrowed), precise attention to surgical methodology as previously explained[13] may account for the high cure rates for ISD reported by Nakamura et al. Poiseuille's Law can work in reverse. A loose tape and failure to repair the distal urethral closure mechanism may explain suboptimal cure of USI and ISD.

Anatomical pathways to pain, bladder, bowel dysfunction from loose cardinal/uterosacral ligaments (CL/USL).
It is our view that the cardinal/uterosacral ligaments (CL/USL) are the most important ligaments in the pelvis. CL/USL are the main supports of the uterus and vaginal apex; they are the anchoring point for the backward/downward vectors which are critical for control of bladder & bowel function. Laxity in CL/USL will cause uterine prolapse. Because the control mechanisms for fluid flow are exponentially determined (Poiseuille's Law), even minor prolapse, fig. 6, may weaken the directional vector forces which pull against CL/USL and may affect the following functions of CL/USL
- *Control of bladder and bowel urgency and frequency.*
- *Control of nocturia.*
- *Control of bladder and bowel evacuation (obstructive defecation 'ODS').*
- *Control of anorectal closure (continence).*
- *Control of chronic of pelvic pain.*

The pathways to these dysfunctions will be examined in turn.

Anatomical pathways to 'obstructive micturition or defecation' (organ emptying problems)
X-ray video studies, Part1, demonstrated an external striated muscle opening mechanism for both bladder and anorectum. It is known that the resistance within a tube is inversely related to the radius (Poiseille's Law). A loose USL (fig. 6), may result in weakening of the urethral or anorectal LP/LMA opening forces. The bladder detrusor or rectum have to contract against an unopened tube. This is perceived by the patient as 'obstructed micturition' or 'obstructed defecation', with symptoms such as 'feeling bladder has not emptied, 'stopping and starting', multiple emptying, post-micturition dribble, raised residual urine[14] and for bowel, constipation

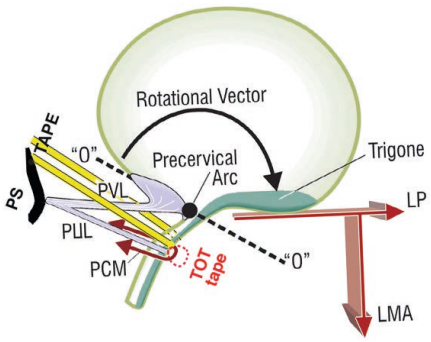

Figure 5. The anatomical difference between retropubic and TOT USI operations.
Patient in sitting position. The TOT tape rests in the horizontal position below midurethra to provide a firm rotation point for the posterior rotational vector forces which close bladder neck, m. levator plate (LP) and the conjoint longitudinal muscle of the anus (LMA); likewise with the retropubic operation; the retropubic tape (yellow) proceeds behind the symphysis (PS) to reinforce the pubourethral (PUL) and pubovesical ligaments (PVL) if they are loose. The reinforced PUL restores two urethral closure mechanisms, proximal and distal, as in the ultrasound fig. 3. PUL provides a firm anchoring point for the posterior rotating vectors which effect bladder neck closure; it anchors the anterior vector m.pubococcygeus (PCM) which stretches the suburethral vagina forwards to close the distal urethra.

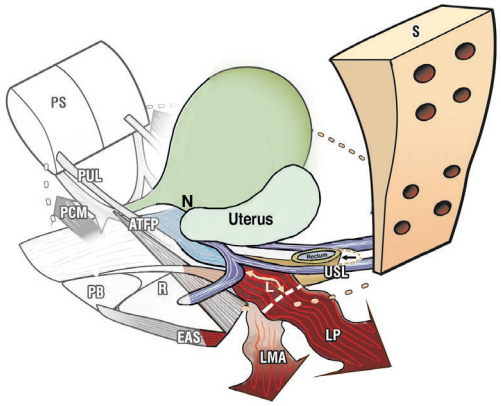

Figure 6. Potential consequences of loose uterosacral ligaments as interpreted by Gordon's Law.
View from above. The uterus has prolapsed to 1st degree. The USLs have elongated by 'L', as have LP and LMA. The rectum also has descended, by virtue of its attachments laterally to the elongated USL as indicated by the very small arrow behind rectum. The large wavy arrows signify diminished contractile strength of LP/LMA.

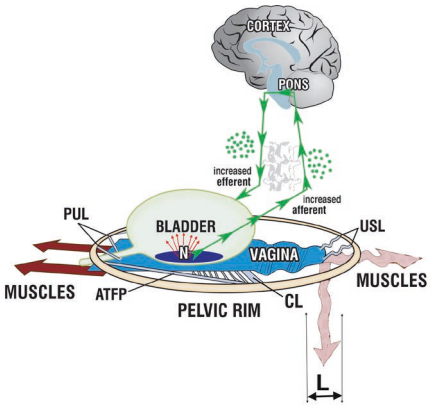

Figure 7. Urge incontinence as interpreted by Gordon's Law
The uterosacral ligaments (USL) lengthen 'L' and are unable to suspend the vagina adequately. The downward/backward muscle forces (arrows)* lengthen by 'L' and weaken. The vagina cannot be stretched sufficiently to support the stretch receptors "N". "N" fire off increased afferent impulses at a low bladder volume and this is perceived by the cortex as urgency. If the quantum of afferents is sufficient to activate the micturition reflex, the efferents are activated; the forward muscles relax; the backward muscles open out urethra; bladder contracts; the patient may uncontrollably lose urine ("urge incontinence").
*the wavy form and pink colour of the arrows denote weakened muscle contractile force.

or obstructive defecation (ODS). Shortening and reinforcing CL/USL by TFS restores prolapse and the external opening mechanism with symptom and residual urine improvement for bladder[14] and bowel[15].

Anatomical pathway to symptoms of 'overactive bladder'- urge, frequency, nocturia. Inability of the weakened LP/LMA muscles (fig. 6), to stretch the vagina or anorectum sufficiently to support the stretch receptors 'N' may cause them to fire off at a low volume to activate the micturition reflex. The cortex perceives this as urinary 'urgency', frequency (fig. 7) and at night, nocturia (fig. 8).

Anatomical pathway to chronic pelvic pain
The role of USLs in producing chronic pelvic pain was described in detail by Heinrich Martius in 1938[16] and was re-discovered independently by Petros[17].
Chronic pelvic pain is perceived in the various nerve distributions,T12-L1, S 2-4 (fig. 9): lower abdomen, groin, lower sacrum[18], introitus[19], paraurethral[20], interstitial cystitis[21], deep dyspareunia[21].
Inability of the weakened muscles to tension the uterosacral ligaments may cause unsupported nerve plexuses within the USLs to fire off (fig. 9). Objective proof of USLs as the pathway to chronic pelvic pain origin was obtained by different types of 'simulated operations**.
Wu et al. reported relief of pelvic pain and suburethral tenderness by insertion of the lower part of a bivalve speculum to support the posterior fornix[20]. Bornstein relieved vulvodynia pain by local anesthetic injection into the cervical part of the ULSs[19] as did Petros in 3 patients with Interstitial Cystitis, abdominal pain and suburethral tenderness[21]. Gunnemann reversed anterior rectal wall intussusception with a cyslindrical vaginal pessary, inserted under ultrasound control[22]. Another method, used to confirm the role of USL in nocturia causation, is to place a large tampon in the posterior vaginal fornix overnight.
** Mechanically supporting ligaments as in fig. 10 and observing change in symptoms.

Pathway from dysfunction to diagnosis- the Pictorial Algorithm.
The sum total of the pathways to prolapse and symptoms is summarized by the Pictorial Algorithm (fig. 10). The Algorithm relates the damaged ligaments to actual symptoms. Thus symptoms marked by 'X' in fig. 10 can indicate which ligaments are damaged. For example, urinary stress incontinence indicates it is PUL which is damaged. The presumptive ligament damage based on these symptoms (fig. 10), is confirmed by vaginal examination: each of the damaged ligaments has specific anatomical criteria detailed in the textbook "The Female Pelvic Floor"[24]. 'Simulated operations' apply mechanical support for specific ligaments[25] and the results are observed, either clinically by improvement in USI urge, objectively by transperineal ultrasound (fig. 3), or by 'interventional' urodynamics (raised intraurethral pressure)[24]. There is no correlation between degree of prolapse and symptom severity[26]. Fig10 is an actual case example with positive symptoms marked in.

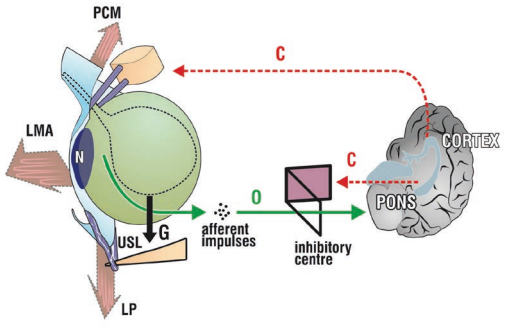

Figure 8. Mechanical origin of nocturia (patient asleep).
Pelvic muscles (large arrows) are relaxed. As the bladder (broken line outline) fills, it is distended downwards by gravity 'G'. If the uterosacral ligaments (USL) are weak, the bladder base continues to descend; at a critical point, the cortical closure reflex 'C' is overcome; the stretch receptors 'N' now activate the micturition reflex: the patient is awakened by a feeling of urgency (nocturia); PCM is actively relaxed by the cortex. If the micturition reflex is not adequately controlled, LP/LMA contract to open the urethra and the detrusor contracts; the patient may lose urine on the way to the toilet.

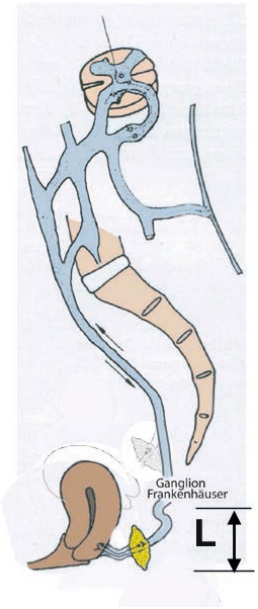

Figure 9. Pathogenesis of chronic pelvic pain.
The Ganglions of the Frankenhauser and the Sacral Plexuses are supported by uterosacral ligaments (USL) at their uterine end. 'L' indicates ligament laxity as per Gordon[9]. The posterior directional forces are weakened and cannot stretch the USLs sufficiently for them to support the nerves. The nerves may be stimulated by gravity or by the prolapse or by intercourse to fire off and be perceived as pain by the cortex.

Consequences of different strength of ligaments and vagina for pelvic surgery technique

Interpretation of the life-long experimental work of Yamada on tissues[27] is the key to understanding the ligament-based rules of surgical reconstruction according to the Integral Theory System[1]. Yamada demonstrated that the breaking strain of ligaments was approximately 300 mg/mm^2 , that of vagina, 60 mg/mm^2 [27]. This means ligaments are primarily structural, the vagina not. Xray video studies[28] confirmed that ligaments do not stretch significantly during effort or evacuation. It follows that any reconstructive surgery has to reinforce the structural part of the ligaments, collagen 1. Only an implanted tape can do this[25]. The same xray video studies indicated that the vagina has a minimal structural role. The vagina is an elastic organ which plays an important role in transmitting the vector forces to close and open the bladder[24]. As elasticity cannot be surgically reproduced, it must be conserved. Any excision of vagina reduces the quantum of elastin and collagen 3 available for function. Finally, the uterus is the direct or indirect insertion point for all the ligaments. Hysterectomy requires severing of the descending uterine artery, which is the main blood supply of the proximal ends of the cardinal and uterosacral ligaments. We have strictly followed the 3 rules of surgery which evolve from the above in our TFS surgery:

1. A loose ligament must be shortened and reinforced with thin strips of tensioned tape to create a collagenous neoligament[25]. This was the surgical principle of the original midurethral sling .
2. The vagina must be conserved, not excised.
3. The uterus must not be removed without good cause.

We have found that following these rules vastly diminishes post-operative pain and urinary retention and allows the TFS to be performed as a day or local anesthetic procedure.

The surgical pathway from ligament repair to restoration of structure (prolapse) and symptom cure by TFS

TFS Surgery precisely tensions the ligaments by using tactile feedback by the operator during tightening to sense return of muscle strength. Exactly the same surgical technique is applied to each of the 5 ligaments PUL,ATFP,CL,USL,PB: stretch the uterus downwards to locate the ligament vaginally; make a tunnel through the ligament with dissecting scissors; insert anchor and tape; repeat on the contralateral side; tension the tape. As the laxity 'L' (figs 4-6, 7-9) is removed by the tensioning, the surgeon feels a gradually increasing resistance against the tensioning. This signifies return of muscle strength as defined by Gordon's Law[9]. At this point, the surgeon stops the tensioning.
Once the ligaments have been shortened and reinforced, the directional muscle forces act immediately to restore all the functions dependent on the competent ligaments.

DISCUSSION

TFS ligament surgery shortens and reinforces the ligaments. The tape creates a linear deposition of collagen along the length of the ligament[25]. Shortening and reinforcing the ligament reverses the cascade of dysfunctions described in this work, usually by the day after surgery, with high cure rates for POP and symptoms [3-5, 13, 29-33], as noted in Tables 1&2, Part 1.

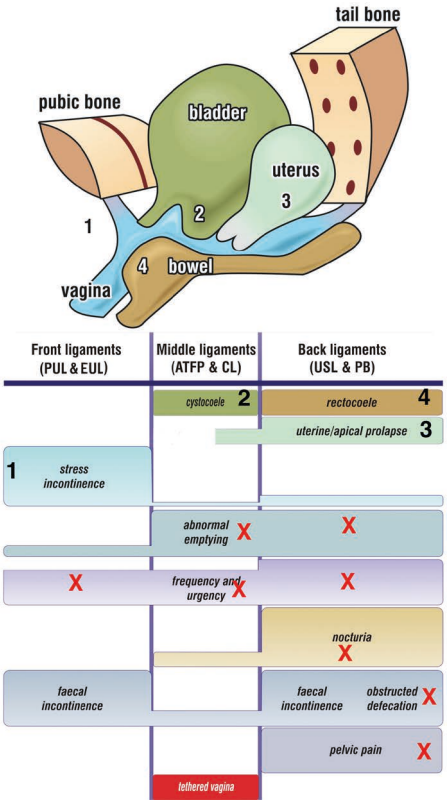

Figure 10. The Pictorial Algorithm
It is a summary guide to causation and management of pelvic floor conditions. The marks 'X' accurately indicate which ligaments are damaged. Because symptoms such as urgency and abnormal emptying may be caused by ligament damage in more than one zone, all rectangles for that symptom should be marked. The area of the symptom rectangles indicates the estimated frequency of symptom causation occurring in each zone. The main ligaments causing the symptoms and prolapse in each zone are indicated in capital letters, two in each zone: PUL 'pubourethral ligament', EUL 'external urethral ligament' (*Anterior ligaments);* ATFP 'arcus tendineus fascia pelvis', CL 'cardinal ligament complex' (*Middle ligaments)*; USL 'uterosacral ligament,' PB 'perineal body'(*Posterior ligaments)*. The numbers in the figure correlate with structural damage and with ligament damage: ; 1: USI; 2: cystocele; 3 uterine prolapse; 4:rectocele. The diagnosis for this patient is cardinal /uterosacral damage and perineal body ligament damage. Thm. NOTE Major symptoms may occur with minimal ligament damage.

CONCLUSIONS

Symptoms may occur as a consequence of minor, barely detectable, anatomical–abnormalities. Jeffcoate, in his 1962 textbook observed that some patients with gross degrees of prolapse had no symptoms at all, while others with minor degrees of prolapse "complained bitterly" of symptoms such as pelvic pain[26]. The explanation for this, as we see it, is the exponential nature of the control mechanisms and patient to patient variation in the sensitivity of the peripheral receptors

DISCLOSURES

Conflicts of Interest: None
Ethics: Not required
Contributions: Conceptualization, writing, revision: all authors

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**Correspondence to:*
Yuki Sekiguchi
3-115 Hyakudan -kan 3F Motomachi Nakaku - YOKOHAMA 232-0861 - Japan, E-mail: dumbo-ys@d9.dion.ne.jp

External urethral sphincter Original section by Salvador Gil Vernet, 1942

JOSE MARIA GIL-VERNET VILA

Emeritus Professor of Urology, University of Barcelona, Spain

Abstract: In the 1940s, Salvador Gil Vernet provided the first description of the external urethral sphincter as a vertically arranged tubular structure, refuting the existence of a plate of muscle arranged transversely between the two ischiopubic rami. An error that was established in atlases and anatomy textbooks until the end of the twentieth century.

Keywords: External urethral sphincter; Histology; Foetus

External urethral sphincter-Original section by Salvador Gil Vernet 1942.
In the drawing (Fig. 1) we can see the rectum (R), prostate (P), levator ani mucles (LA), obturator internus muscle (OI), ischiopubic rami (IR), neurovascular bundles (NVB), extensions of the pelvic plexus, the external urethral sphincter (asterisks) surrounding the prostatomembranous urethra (U), ischiopubic rami (IR), Alcock's canal with the internal pudendal nerves and vessels (AC), perineal membrane (dots), crura of the corpus cavernosum (CC), ischiocavernosus muscles (IC), penile bulb (PB) and bulbospongiosus muscle (BS).

Correspondence
Dr. JM Gil-Vernet Sedó, Centro Médico Teknon, Vilana 12 Barcelona 08022 Spain
centrogilvernet@gmail.com

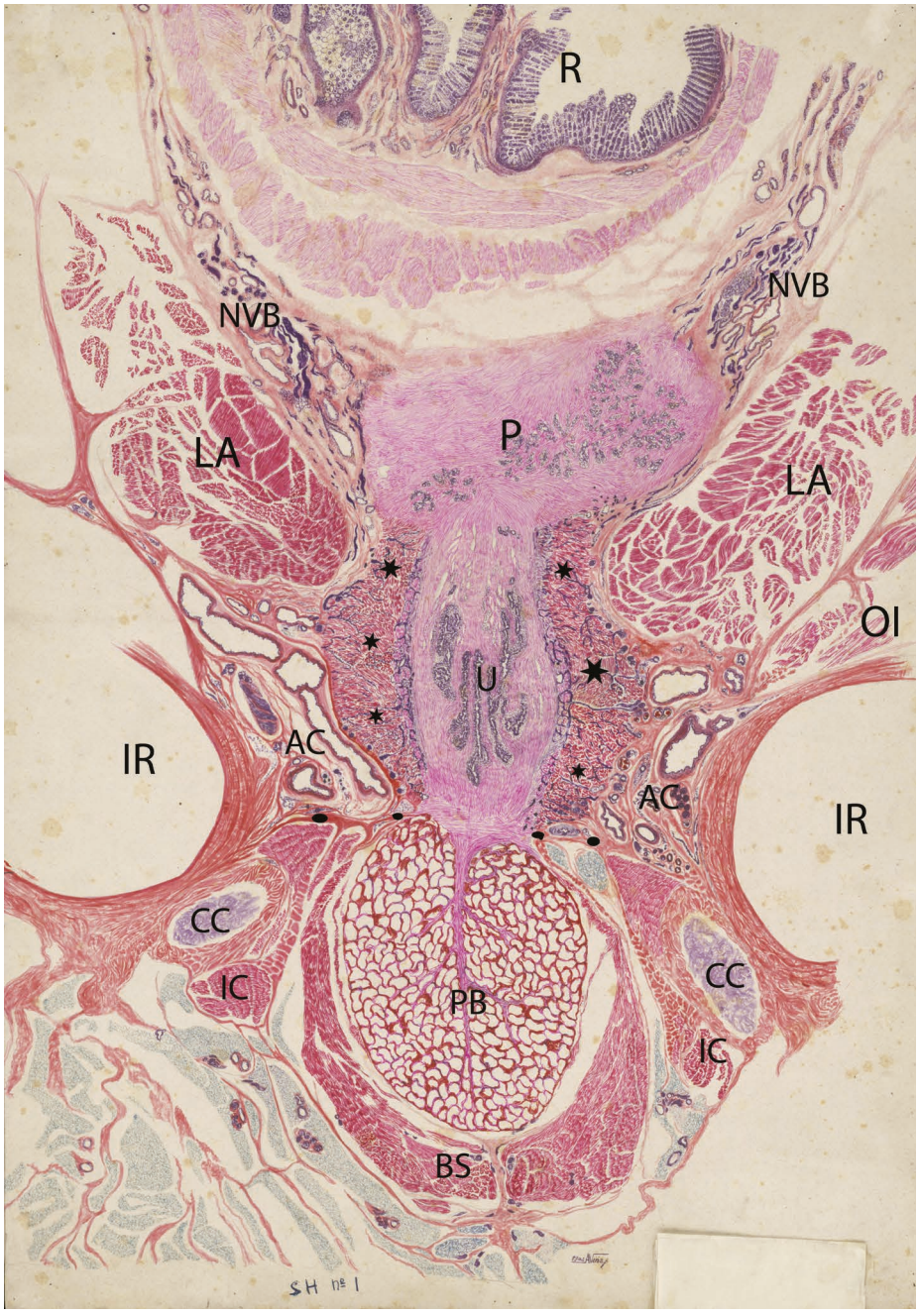

Figure 1. Oblique coronal section from a 6-month foetus. 40 *µ*m. H&E stain. Year 1942.

Case report

Repair of rectovagina fistula consequent on anal coitus

PRABHAT CHANDRA MONDAL

Bankura Sammilani Medical College Bankura, India

Keywords: Rectovaginal fistula; Coital injury

Rectovaginal injury due to coitus is a great challenge to the surgeons, as it could lead to rectovaginal fistula. Apart from child birth, surgeons and gynaecologists in India get many cases of rectal injury due to fall on sharp object, cow horn goring and coitus. Here, we got a case of complete rectovaginal tear due to coitus. A 35/-year-old woman with previous two vaginal deliveries attend at mid night due to vaginal bleeding following sexual relation. She is separated from her husband and has had sexual relations occasionally with her boy friend. It was an anal sex in supine position. Following sudden thrush, she felt pain and saw bleeding per vagina. Immediately, she was admitted in our hospital.

Vagina was packed. Intravenous fluids (IV), antibiotics and analgesics were given. One unit blood was requisitioned as she had haemoglobin of 8 gm%. Under anaesthesia, the wound was evaluated, and washed with antibiotic lotion. Rectum was found empty. Surgeon introduced left index finger perrectum into vagina easily (Figure 1 A). Colostomy was not done, as we managed similar cases previously without colostomy. Vaginal epithelium was separated from rectum (Figure 1B). Rectal mucosa was sutured with 3 O vicryl interrupted with placing the knots into the rectal lumen (Figure 1C). Denonvilliers' fascia was repaired separately. Vaginal was closed (Figure 1D). As per our hospital protocol, we kept her on IV fluid for two days, on liquid diet for three days. IV triple antibiotics were continued for 5 days. On sixth postoperative day, she was allowed normal diet with laxatives (lactulose).

On 8th day, she was discharged without any fistula with advice to take Vitamin C 500 mg once daily, lactulose for six weeks. She was examined after four months having no symptoms.

Correspondence to:
Prabhat Chandra Mondal MB BS MS (OBG), DGO Assistant Professor (91-9434438618) Jogeshpally Bankura 722101 West Bengal - India
E-mail: prabhatmndl@yahoo.co.in

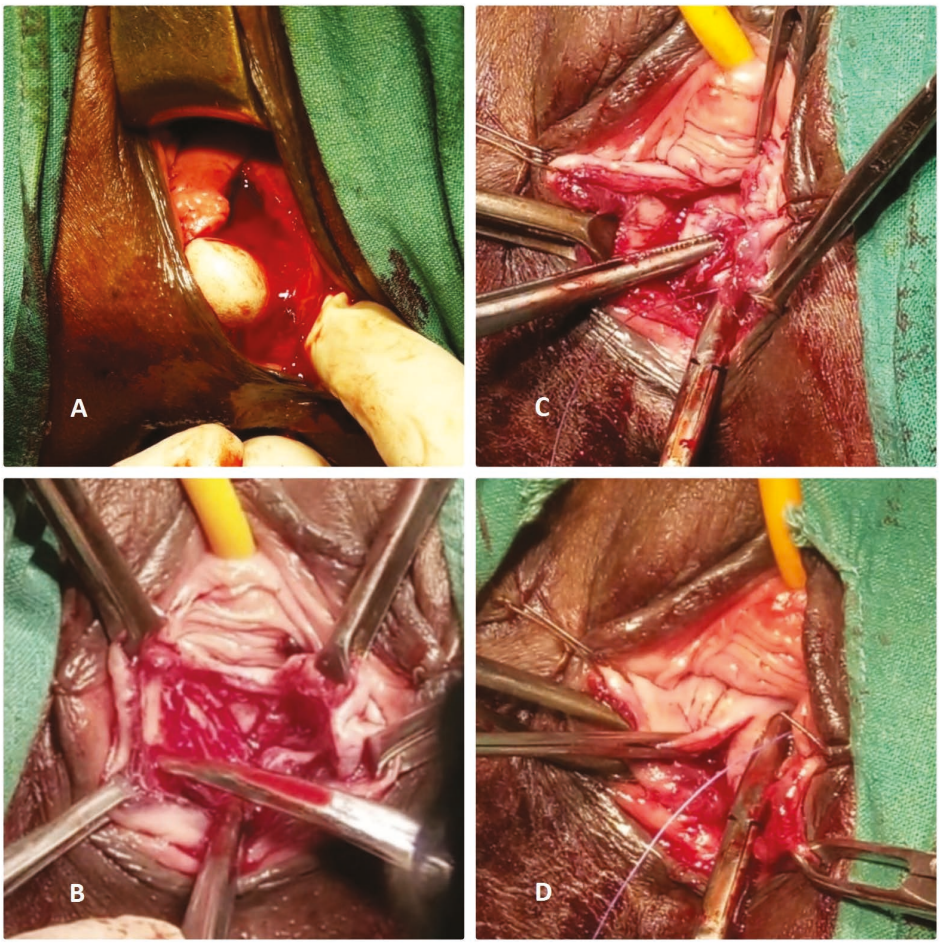

Figure 1
A. Complete perforation of recto-vaginal septum
B. Vaginal mucosa was separated from underlying fascia sharply
C. Rectal mucosa and overlying fascia were repaired separately
D. Vaginal wall was repaired

Video

Sphincteroplasty, perineoplasty, anus remodeling

ALVARO OCHOA CUBEROS

Pelvic Floor Department, Clínica Medical Duarte, Cúcuta - Colombia

Abstract: An 18 year old patient with a history of sexual abuse since she was 11 years old presented with grade IV tear after a vaginal delivery 48 hours before the surgical procedure. Deep involvement of the rectal mucosa, of the entire sphincter and absence of the perineal body was observed.

Keywords: Sphincteroplasty; Perineoplasty; Anus remodeling

https://youtu.be/UYoIuMkCg34

Correspondence to:
Alvaro Ochoa Cuberos - Vitta Centro de Especilistas Avenida 1 #7_73. Piso 10 - Cúcuta 540001 Norte de Sabtander - Colombia
E-mail: aochoa_cuberos@hotmail.com

Letter to the editor

Fine-tuning sling tension post-operatively may have longterm benefits in preventing late-onset urinary retention- the Poiseuille effect

PETER PETROS

University of NSW, Professorial Dept of Surgery, St Vincent's Hospital, Sydney

Abstract: Post-operative adjustment after midurethral sling surgery using special methodology has immediate effects in improving cure rate, reducing post-operative urinary retention and may helping reduce obstruction due to sling fibrosis many years after the surgery.

Keywords: Midurethral sling; Post-operative adjustment; Fibrosis.

Dear Editors

I write to comment on what I consider is a major development in miduretral sling methodology. Shkapura et al.[1] successfully address a major problem with midurethral slings (MUS), too tight: post-operative urinary retention, too loose: incontinence. Using cough stress test and flow measurements, they confirmed continence restoration relied on a precise, non-obstructive tape length as per the prototype operations[2], figure 1. The tape works by reinforcing the pubourethral ligament (PUL) against which opposite striated muscle forces stretch, narrow and kink urethra to close it distally and at bladder neck[2], restoring continence and geometry[3]. Narrowing exponentially increases the resistance to flow inversely by the 4th power of the radius (Poiseuille's Law). If PUL (tape) is loose, muscles lose contractile force and cannot close urethra (stress incontinence); if too tight, sling obstructs urethra (retention)[1], both modes explainable by the "Poiseuille Effect", exponentially varying resistance to flow with only minor changes in diameter. The fine-tuning of Shkapura et al's methodology is unique in being able to make such minor changes post-operatively, with excellent results as regards relief of obstruction and higher cure rates[1].

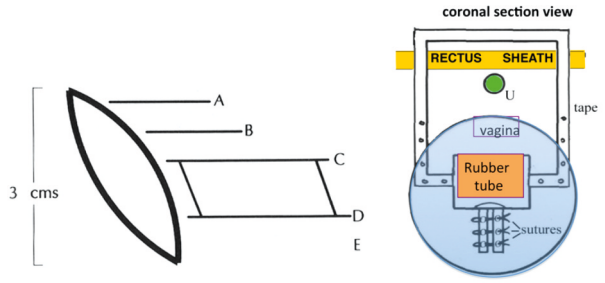

Figure 1. – Shkapura MUS. The mechanism of the midurethral sling is not obstructive. (Right) Coronal section immediately behind the pubic symphysis. The tape was configured around the rectus sheath into the vagina, through both ends of a rubber tube, exiting at its inferior end, both ends secured with interrupted sutures in holes set 0.5 cm apart, so the tape could be lowered sequentially if there was obstructed flow. (Left) symptoms experienced on lowering the tape: A, loss of sensation of bladder fullness; B loss of sensation to flow through the urethra; C-D slow stream, stopping starting, frequency nocturia, urgency (FNU); E normal flow, no FNU. Based on diagrams from the original 1990 publication of the Integral Theory (pp 53-59).

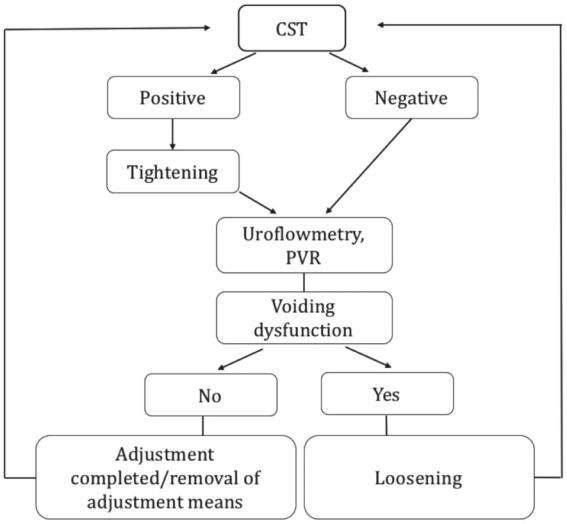

Figure 2. – Protocol for loosening or tightening sling.

The modus operandi of the MUS, musculo-elastic closure[2,3], is not just a theoretical issue. There are some who, with little scientific proof, promote physical obstruction* by the tape as the mechanism for continence restoration, which means post-MUS obstructive micturition would be considered a normal event. It is not. Tape related collagen shrinks with age. Urinary retention from MUS inserted 20 years earlier is being increasingly reported. Because the "Poiseuille Effect" exponentially magnifies such shrinkage, it would seem prudent for surgeons to perhaps be more proactive about tape loosening with persistent obstruction, as these are the very patients most likely to present years later with retention.

Though many "tips and tricks" help avoid tape obstruction, only Shkapura's method objectively addresses the Poiseuille Effect to predictably produce high cure rates. On this basis alone it is worth serious consideration.

* **A simple clinical test** can prove it is not tape obstruction which closes the urethra. Examine a USI patient with very full bladder (ready to micturate) in the semirecumbent position. Ask her to cough; press gently upwards on one side of urethra immediately behind the symphysis with curved hemostat until urine loss is controlled; then, with haemostat in place ask patient to pass urine by pushing down when she feels the urge; some succeed, proving that the mechanism for return to continence is musculoelastic as confirmed by dynamic video ultrasound[3].

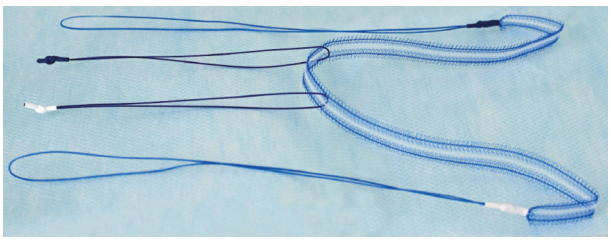

Figure 3. – How sling can be tightened or loosened.

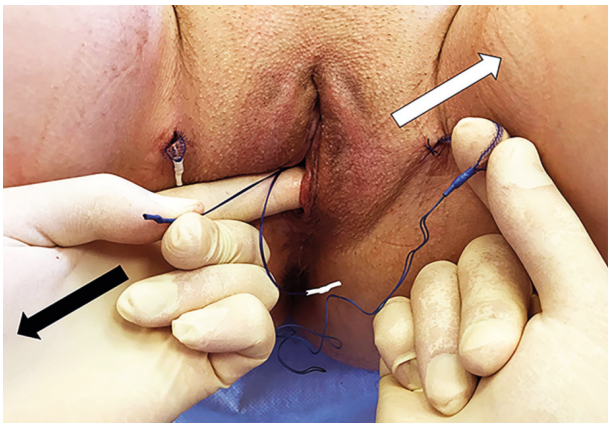

Figure 4. – Post-operative adjustment of sling.

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Correspondence to:
Peter Petros
E-mail: pp@kvinno.com

Letter to the editor

Dear Editor,
The article 'Influence of foot stool on defecation: a prospective study' by Shota Takano, Midori Nakashima, Masahiro Tsuchino, Yuya Nakao and Atsushi Watanabe (Pelviperineology 2018; 37: 101-103) reports a well designed and conducted study. The conclusion is that a footstool aids defaecation by incorporating leaning forward and raising intra-rectal pressure. However, all of the measured mechanical parameters remained unchanged and non-significant suggesting the opposite. The authors should add why they think in the absence of these changes that the abdominal pressure increases solely by leaning forward. This would promote further discussion and research into the role of intra-abdominal pressure in defaecation.

DARREN M. GOLD
Colorectal Surgeon, Senior Lecturer in Surgery,
St Vincent's Clinical School, UNSW, Sydney, Australia
dandjgold@gmail.com

The Author's reply

Dear Editor,
I would like to thank Dr. Darren Gold for his comment on our paper entitled, "Influence of foot stool on defecation: a prospective study" and I have provided an explanation related to his comment.
The pushing rectal pressure showed no significant difference between with and without a foot stool in both the upright sitting position and the upper body bent forward position. However, we found that in the upper body bent forward group, the difference of the rectal pressure between with and without a foot stool was larger than in the backward position group. Therefore, we think that the larger difference of the rectal pressure in the upper body bent forward position with a foot stool facilitates better evacuation especially among elderly patients.
We sincerely appreciate Dr. Gold's comment and we will continue to study the relationship between continence function and defecatory posture.
Best regards,

SHOTA TAKANO
Coloproctology Center Takano Hospital
3-2-55 Oe, Chuo-ku, Kumamoto 862-0971, Japan
Tel: +81-96-320-6500
micg@takano-hospital.jp

CONGRESS 2019, October 6th-8th – Treviso (Italy)

Pelvic Floor Disorders: Current Status, Technological Advances and Perspectives

CONGRESS PRESIDENTS
Giulio A. Santoro, Giuseppe Dodi

ISPP SCIENTIFIC COMMITTEE
Burghard Abendstein, Elvira Bratila, Klaus Goeschen, Darren Gold, Bruce Farnsworth, Bernhard Liedl, Naama Marcus Braun, Menahem Neuman, Peter Petros, Yuki Sekiguchi, Ding Shuqing, Akin Sivasglioglu, Peter Von Theobald, Adi Weintraub

www.pelviperineology.com

Dear Colleagues,

the 12th annual Congress of the International Society of Pelviperineology will be held in Treviso/Venice, Italy, from October 6th to the 8th. It is an honour to organize this meeting, involving ISPP members, experts in the field of the pelvic floor from other International Societies, and with an important participation of the Italian scientific societies of the various specialties having common pelvic interests. The approach to the pelvic floor belongs to urologists, gynecologists, colorectal surgeons, gastroenterologists, pyschiatrists, obstetricians, nurses, physiotherapists, psychologists, radiologists, sexologists, andrologists: a patient-centered vision is needed!

The themes chosen "Current status, technological advances and perspectives" have the ambitious aim to provide not only a consensus on the management of pelvic floor disorders, according to the evidence-based medicine and the international guidelines, but to present and discuss new and emerging technologies. We hope that the numerous topics of the Congress (pelvic anatomy, mesh in prolapse surgery, chronic pelvic pain, therapy of urinary and fecal incontinence, pelvic floor imaging, innovation in pelvic floor surgery, pelvic floor rehabilitation) will stimulate participants to submit abstracts and videos showing their personal experience and current or future research. In addition, pre and post-congress courses and workshops will give young doctors the opportunity to receive training and education on specific topics. The aims of the ISPP, which will be confirmed and developed as the congress proceeds, are the realization of formal and informal discussion groups, Masterclasses, Fellowships, in effect, a School for the formation of the Pelvic Surgeon, as well as Technological development and partnership with all interested Companies in our field.

Treviso, located in close proximity to Venice, is an amazing tourist destination and has a great deal to offer for those who are willing to explore. It has a Celtic origin, however in 89 BC the settlement was turned into a Roman commune and it developed into an important city. Throughout the middle age Treviso was part of the Lombard League and was involved in various sieges and conflicts, hence the need for stunning city walls and defensive towers. It is the site of the production of Prosecco wine and the birthplace of the dessert Tiramisù.

We wish to meet you in Treviso for a successful ISPP 2019!

Giulio A. Santoro, Giuseppe Dodi

CALL FOR ABSTRACTS
DEADLINE
July 31st

Saturday October 5th, 2019

SOCIAL EVENTS
Tour of Treviso
Tour of Venice
Tour of Prosecco production

Sunday October 6th 2019. DAY 1

08:00 - 13:00 **Workshop 1** – PF anatomy, function and the Integral Theory
08:00 - 13:00 **Workshop 2** – PF Rehabilitation
08:00 - 13:00 **Workshop 3** – Aesthetic Gynecology and Plastic Surgery in Pelvic Floor
08:00 - 13:00 **Workshop 4** – New Technologies in Pelvic Floor Surgery

14:00 - 17:00 **Round Table I** – Mesh in PF surgery: current status, pros and cons, technological advances and perspectives

Opening Cerimony

17:30 – 17:50 Welcome address: ISPP President, Congress Presidents, CEO of Treviso Hospital, Major of Treviso
17:50 – 18:20 Honorary Lecture
18:20 – 19:00 Concert
19.00 – 21:00 Welcome Buffet at the Exibition Area

Monday October 7th 2019. DAY 2

08:00 – 10:30 **Symposium I** – Posterior compartment disorders: current status, technological advances and perspectives
11:00 - 13:00 **Round Table II** – Pelvic Pain: current status, technological advances and perspectives
13:00 - 14:00 **Luncheon Symposium I** – New Horizons in the Treatment of Hemorrhoids
13:00 - 14:00 **Luncheon Symposium II** – New Horizons in the Treatment of Anal Fistulas

14:00 - 16:00 Video-Session and Podium Presentations
16:00 - 18:00 **Symposium II**: Genetics and Hormones: current status, technological advances and perspectives

ISPP Assembly

18:30 – 19:00 ISPP Assembly
19:00 – 19:15 Announcement of ISPP Congress 2020

Social Dinner

20:30 – 22:30 Social Dinner

Tuesday October 8th 2019. DAY 3

08:00 – 10:30 **Symposium III** – Anatomy, Physiology and Imaging of the PF: current status, technological advances and perspectives
11:15 - 13:15 **Round Table III** – Incontinence: current status, technological advances and perspectives

ISPP 2019 Closure

13:00 – 13:15 Final Lecture
13:15 – 13:30 Best Video and Best Podium Presentations Awards
13:30 Closure of the Congress

14:00 – 18:00 **Workshop 5** – Sacral Nerve Stimulation
14:00 – 18:00 **Workshop 6** – PF and Anorectal Ultrasound
14:00 – 18:00 **Workshop 7** – HPV related lesions in urogynecological and proctological practice
14:00 – 18:00 **Workshop 8** – Laparoscopic training in pelvic floor

ISPP

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