REVIEW

Current treatment of pelvic organ prolapse correlated with chronic pelvic pain, bladder and bowel dysfunction

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Purpose of review
The purpose of this review is to critically analyze the relationship between symptoms of abnormal emptying of the bladder, urgency, pelvic pain, anorectal dysfunction and pelvic organ prolapse (POP) and to present evidence in order to show how many of the above mentioned symptoms can be cured or substantially improved by repair of specific pelvic ligaments.

Recent findings
In this review, we provide evidence to show how often these dysfunctions occur and how they can be cured in 42–94% by appropriate pelvic floor surgery in the longer term, up to 2 years. Laxity in ligaments and/or vaginal membrane due to damaged connective tissue may prevent the normal opening and closure mechanism of urethra and anus, because muscles need finite lengths to contract properly. Hypermobility of the apex can irritate the pelvic plexus causing chronic pelvic pain. In consequence, dysfunctions as abnormal emptying of the bladder, urgency, pelvic pain, fecal incontinence and obstructed defecation can occur in women with different degrees of POP.

Summary
In conclusion, it has to be recognized that women bothered by these symptoms should be examined for POP and appropriately advised for possibility of cure by pelvic floor surgery after careful selection.

Video abstract
http://links.lww.com/COU/A9

Keywords
anorectal dysfunction, over active bladder, pelvic floor surgery, pelvic organ prolapse, pelvic pain

INTRODUCTION

In the female organism, the pelvis is an especially vulnerable site for major, often disabling disease, in particular, pelvic organ prolapse (POP), pain, bladder and bowel disorders. The anatomical and symptomatical problems are caused by damaged or altered connective tissue in the pelvic organs and their suspending and supporting structures [1].

Our current population of the developed world is aging and life expectancy is growing. The incidence of POP and troubling symptoms such as urgency, nocturia, chronic pelvic pain (CPP), bladder emptying problems increases with age, especially after the menopause. These conditions constitute major problems for patients, quality of life (QOL), the health system, the community and government expense.

In their lifetime, almost 11% of women will undergo pelvic POP surgery, with a reoperation rate of 12–30% [2,3]. Approximately 300,000 surgeries for prolapse are performed annually in the United States [4].

Up to now, the discussion about the significance of the supporting and suspending system of the pelvic organs is still largely unknown in the English literature. International learned committees do not refer to connective tissue laxity as a major cause of

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KEY POINTS

- Symptoms of abnormal emptying of the bladder, over active bladder, nocturia, pelvic pain, obstructive defecation and fecal incontinence can be caused by pelvic organ prolapse.
- Laxities of connective tissue (ligaments and fascias) in the pelvic floor area are mainly responsible for pelvic organ prolapse and can be repaired by appropriate pelvic floor surgery.
- Symptoms of abnormal bladder emptying, over active bladder, nocturia, pelvic pain, obstructive defecation and fecal incontinence can be cured in 42–94% by adequate pelvic floor surgery.
- Women with these symptoms of pelvic floor dysfunctions should be examined for POP and advised for the possibility of cure by pelvic floor surgery after careful selection.

POP symptoms [5,6], though several publications concentrate intensively on this focus [7–14,15▪,16].

The aim of this review is to critically analyze the relationship between symptoms and POP and to present evidence in order to show how many of the above mentioned symptoms can be cured or substantially improved by repair of specific pelvic ligaments.

STATE OF THE ART TODAY

According to the ICS [5], bowel, bladder and pain symptoms are major problems in up to 30% of women. The cause is said to be unknown. Other than urinary stress incontinence, none of these conditions are considered to be surgically curable by leading societies [5,6]. Since decades this resulted in the fact that 3 major specialties, Urology, Gynecology and Coloproctology offer a wide variety of treatments within each specialty. At first glance, this three-fold division seems to be logical, because patients suffer from bladder, prolapse and ano/rectum symptoms:

The urologist is mainly confronted with symptoms such as urge incontinence, nocturia, frequency, obstructed micturition, painful bladder, interstitial cystitis and urinary stress incontinence.

The coloproctologist is visited by patients with hemorrhoids, low sacral backache, perineal pain, anal mucosal prolapse, fecal incontinence, obstructed defecation, descending perineal syndrome and rectocele.

Gynecologists are contacted from patients with cystocele, rectocele, enterocele, uterine prolapse, abdominal pain, vulvodynia, dyspareunia and urine loss during intercourse.

However, this review will show that all these problems are interrelated and in varying degrees frequently appear parallel.

According to the 2013 [17] 5th Consultation on Incontinence, POP 'refers to loss of support for the uterus, bladder, colon or rectum leading to prolapse of one or more of these organs into the vagina'. None of the above mentioned symptoms are attributed directly to POP.

On the contrary, the integral theory [1] views the pelvic floor holistically, as an interrelated system of organs, muscles, nerves and connective tissue structures, where the whole is greater than the sum of its parts, with connective tissue being the most vulnerable to damage.

SUPPORT OF PELVIC ORGANS

Just five pelvic ligaments and the vaginal membrane provide the structural suspension and support for the three organs, uterus, rectum, bladder and their outflow tracts vagina, anus and urethra. The ligaments attach the organs to the skeleton sidewall from above (Fig. 1) [18]. The pelvic muscles support the organs and tension them from below [18,19].

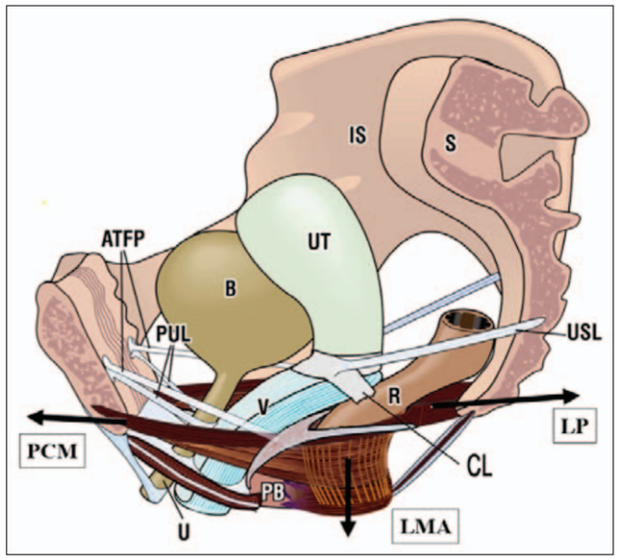

FIGURE 1. Important pelvic ligaments and muscles. Attachment of the organs. ATFP, arcus tendineus fasciae pelvis; B, bladder; CL, cardinal ligament; PB, perineal body; PUL, pubourethral ligament; R, rectum to the skeleton by five ligaments; U, urethra; USL, uterosacral ligament; UT, uterus; V, vagina. Three important muscle groups with foreward, backward and downward force (black arrows): PCM pubococcygeus muscle, LMA conjoint longitudinal muscle of the anus and LP levator plate. From Petros [18] with permission.

Ligaments are strong, relatively inelastic [20] and contain collagen 1, smooth muscle, elastin, nerves and blood vessels. Organs and the vagina are highly elastic, as they must expand and contract according to the particular function. Organs and vagina contain significant quantities of elastin, but have far less structural strength than ligaments.

The vaginal membrane is supported by ligaments. Elasticity of the vagina is fundamental to its function during organ closure, evacuation and intercourse. Elasticity provides a low-energy mechanism for closure of the urethra and anus. Therefore, connective tissue laxity can cause prolapse and dysfunction.

To find an answer to this important interrelationship, Petros [1] created a new strategy for pelvic floor understanding in 1993. The so-called 'Integral Theory' states that prolapse, symptoms of CPP, bladder and bowel dysfunction are mainly caused for different reasons by laxity in the vagina or its suspensory ligaments as a result of altered collagen/elastin.

Because the pelvic muscles contract against the pelvic ligaments, their forces can only be transmitted to act properly on the organs if the ligaments are firm. If the suspensory ligaments are loose, the striated muscles effectively lengthen. Because muscles need finite lengths to contract properly [21], an elongated muscle may prevent the normal opening and closure mechanism of urethra and anus [22,23▪,24,25▪▪].

Furthermore, a lax vaginal membrane cannot support the stretch receptors in the overlying bladder. This may cause a premature activation of the micturition reflex with the consequence of urge, detrusor instability and urine loss, 'OAB' [1].

CPP often is also the result of ligament deficiency or damaged support system. If the uterosacral ligaments fail to suspend/support the Plexus of Frankenhäuser [26▪▪], and the sacral plexus in standing position or in motion, Plexus of Frankenhäuser fires off to cause chronic pain in the sites of anatomic distribution, abdomen, vagina, vulva, anus and even muscles. Moreover, descending intestine creates a serious tension against the plexus sacralis with the result of severe back pain in this area [25▪▪].

To summarize the relationship between POP, symptoms and ligament damage in the front, middle and back parts of the vagina, Petros [18] developed a pictorial diagnostic algorithm (Fig. 2) [18]. A fundamental part of the integral theory is that major symptoms may occur with minimal prolapse and that the cascade of symptoms and conditions detailed in the algorithm can be largely reversed by shortening and reinforcing these five major ligaments and the vaginal membrane.

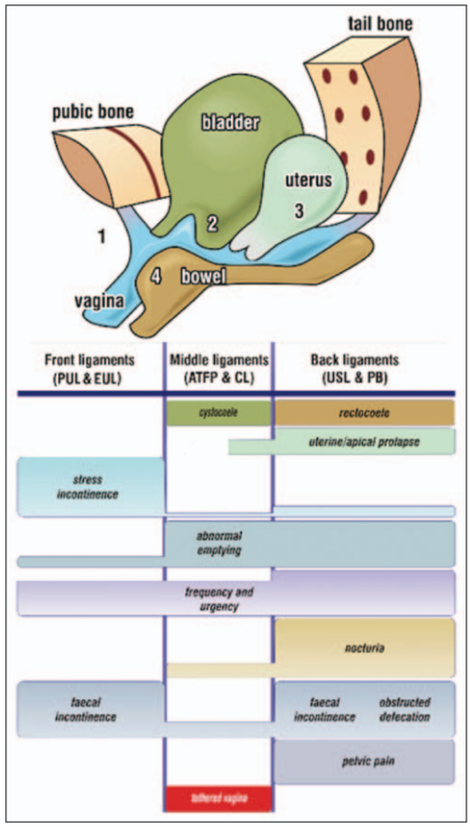

FIGURE 2. Simplified pictorial diagnostic algorithm. Relation of structural damage (prolapse) and symptoms: 1: stress incontinence; 2: cystocele; 3: uterine prolapse; and 4: rectocele. The size of the bar gives an approximate indication of the prevalence (probability) of the symptom. Ligaments which can be repaired are: external urethral ligament (EUL), pubourethral ligament (PUL); CX ring/ cardinal ligament (CL); arcus tendineus fascia pelvis (ATFP); uterosacral ligament (USL); and perineal body (PB). From Petros [18] with permission.

THERAPY OPTIONS

The relationship between POP, urogenital or ano/ rectal disorders and CPP is well known since decades [27] and has been especially noted by leading Expert committees such as the ICS [5] and the EUA [6] However, the relationship between specific symptoms and POP is in discussion. Recommendations for treatment of organ prolapse, CPP, bladder and bowel dysfunction are still largely single-symptom specific. The result is, that treatment is carried out by three different disciplines: urology, gynecology or coloproctology.

The problem of the fractured unidisciplinary approach was clearly addressed by Pescatori *et al.* [28]. They investigated patients with obstructive defecation syndrome thoroughly for other conditions and these were noted in a novel 'iceberg'

diagram. The result was that numerous additional symptoms, though present, were not uppermost in patients' consciousness and had to be searched for.

As traditional understanding and surgery only focus on the top of the iceberg, all other symptoms remain unchanged.

Three examples from urology, coloproctology and gynecology will illustrate the still unsatisfactory situation today:

1. Urology: To treat patients with overactive bladder (OAB) is disappointing. OAB has greater impact on QOL than stress urinary incontinence [29,30] and is responsible for several medical comorbidities such as depression [31], falls and fractures and increased admissions to hospitals and nursing homes [32] The prevalence of OAB is reported at 11.8%–17% in women increasing with age [33,34].

The ICS defines OAB as the presence of 'urinary urgency, usually accompanied by frequency and nocturia, with or without urgency urinary incontinence, in the absence of urinary tract infection or other obvious pathology'[35].

According to the American Urological Association guideline 2014 [36], there are four treatment options:

(1) behavioral therapies such as bladder training, bladder control strategies, pelvic floor muscle training and fluid management
(2) pharmacotherapy with antimuscarinics or β3-adrenoceptor agonists
(3) Botulinum toxin A injections, sacral nerve stimulation – also known as sacral neuromodulation and posterior tibial nerve stimulation
(4) augmentation cystoplasty

Unfortunately, all these strategies are not able to normalize the physiological bladder function. This means that these recommendations can only alleviate complaints, but cannot cure them.

2. Coloproctology: A 2015 published review on descending perineal syndrome [37] does not address coexisting pain and bladder symptoms nor successful simple recent vaginal methods for repair. A laparoscopic ventral rectopexy makes no mention of the far easier and safer vaginal route, which can reinforce the uterosacral ligaments directly [38].

3. Gynecology: CPP syndrome is a major health problem not only for the individual, but also for society [5,6]. Chronic pain of moderate-to-severe intensity occurs in 19% of adult Europeans, seriously affecting the quality of their social and working lives [39]. Investigation by laparoscopy frequently reveals no obvious cause for the pain [40] leading to ascribe causation to psychological reasons.

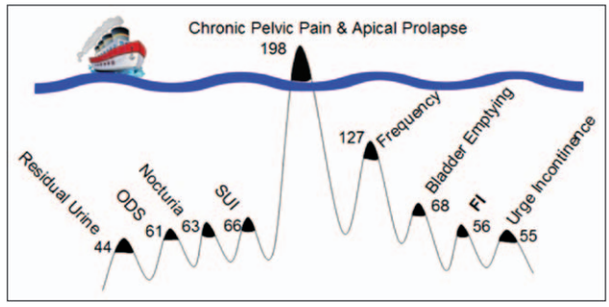

FIGURE 3. Iceberg concept: chronic pelvic pain, peak of the iceberg, was the patient's presenting symptom and reason for surgery. Below the water surface, coexisting bladder and bowel symptoms are located. From Gold and Goeschen [45] with permission.

Otherwise, the association of CPP with other urogenital disorders is well known and has been especially noted by expert committees from the ICS [5] and the EUA [6]. However, no specific relationship between specific symptoms was described by these committees in their deliberations.

On the contrary, since 1993, Petros [1] offered a solution for cure of interrelated symptoms. Numerous publications in the meantime [7–16,40–44] show the reliability of Petros work.

Inspired by Petros and the iceberg concept of Pescatori [28] in 2016, Gold and Goeschen [45▪] rechecked their data from patients primary operated because of unbearable pelvic pain due to posterior fornix syndrome [8,15▪]. Their preoperative data support the iceberg concept, that patients usually present with one main symptom, whereas other symptoms, though present, may be latent. They found a 30–40% coexistence of bladder and bowel dysfunctions in patients presenting with CPP (Fig. 3) [45].

In order to cure all symptoms, if possible, it is necessary to know the interrelationships of the suspension and support system summarized in the integral theory and the pictorial diagnostic algorithm [18,19].

From the therapeutic point of view, POP patients with pain, bladder and/or bowel symptoms can be pooled in three groups [26▪▪]:

Group 1: Patients with intact pelvic floor, but damaged ligamental suspension. In these cases, uterus, vagina, rectum and bladder can leave their normal position causing prolapse of varying degrees, pain, bladder and bowel dysfunction, Fig. 2.

Group 2: Patients with damaged pelvic floor but sufficient ligaments. These patients have major symptoms with minor prolapse.

Group 3: Patients often have a combination of both, for example, third-degree cystocele with

first-degree uterine prolapse with all the symptoms in the posterior column, Fig. 2.

This differentiation has important therapeutic consequences and allows explanations for the different cure rates after vaginal or abdominal surgery in the literature:

(1) If the entire suspension system is deficient, it might be enough to repair only the loose ligaments.
(2) In case of pelvic floor damage, this problem must be solved by restoration of the base in order to support the pelvic organs and the intestine.
(3) If ligaments and pelvic floor are lax, both structures have to be renewed.

Numerous surgeons still favor the abdominal way to restore the anatomy either by laparoscopy [46–51] or by laparotomy [52–55].

If exclusively uterosacral and/or cardinal ligaments are damaged, this problem can be solved abdominally as well as vaginally with artificial neoligaments. However, abdominal surgery does not recreate the natural axis of the vagina if the attachment area of mesh is the promontorium. Therefore, this procedure creates an abnormal vertical inclined vaginal axis causing pain, entero/rectocele with bowel symptoms [56].

In case of stress incontinence due to Pubourethral ligament deficiency, the gold standard is the vaginal renewal of the ligament.

Patients with damaged pelvic floor but sufficient ligament suspension primarily need a reconstruction of the base. This can be done only vaginally. Via laparotomy, it is not possible to repair a dilated or prolapsed levator ani muscle or damaged perineal body.

Keeping this in mind, Petros [1] created a new vaginal strategy of pelvic floor surgery based on the integral theory, which regards symptoms and organ prolapse as being both caused by lax suspensory ligaments. Application of the neoligament principle was used in the Tenseion-free vaginal tape [41], and cure rates have been reported that have not been achieved before [7–14,15▪,16,40–44,57]. The three levels described by De Lancey [58] have to be reconstructed precisely by:

Table 1. Current data from 2015 to 2016 about symptomatic cure rates of coexisting pain, bladder and bowel symptoms in POP patients

Cure rates of various symptoms	Goeschen 2015 [15▪] Pat. with POP *N* = 198	Caliskan 2015 [16] Pat. with POP *N* = 268	Müller-Funogea 2015 [43] Pat. with POP *N* = 453	Inoue 2015 [44] Pat. with POP *N* = 278	Propel-study 2017 Pat. with POP *N* = 277
Frequency (>8 per day)	102/127 (80%)		332/452 (73%)	120/132 (91%)	107/133 (80%)
[95% Confidence interval]	[73%; 87%]		[69%; 78%]	[86%; 96%]	[73%; 87%]
Urge	102/127 (80%)	70/95 (74%)	332/452 (74%)	NA	92/131 (70%)
[95% Confidence interval]	[73%; 87%]	[65%; 83%]	[69%; 78%]		[62%; 78%]
Urge wet	44/55 (80%)	49/70 (70%)	332/452 (74%)	124/133 (93%)	72/106 (68%)
[95% Confidence interval]	[69%; 91%]	[59%; 81%]	[69%; 78%]	[89%; 98%]	[59%; 77%]
Nocturia	50/88 (57%)	27/65 (42%)	398/452 (88%)	62/86 (72%)	81/135 (60%)
[95% Confidence interval]	[46%; 67%]	[30%; 54%]	[85%; 91%]	[63%; 82%]	[52%; 68%]
Bladder emptying problems	54/68 (79%)		426/452 (94%)	35/38 (92%)	73/87 (84%)
[95% Confidence interval]	[70%; 89%]		[92%; 96%]	[84%; 100%]	[76%; 92%]
Residual urine (>50 ml)	28/44 (64%)		NA	NA	NA
[95% Confidence interval]	[49%; 78%]				
Stress incontinence	62/66 (94%)	113/121 (93%)	NA	NA	33/55 (59%)
[95% Confidence interval]	[88%; 100%]	[89%; 98%]			[46%; 72%]
Stool outlet obstruction	47/61 (77%)	58/70 (83%)	442/452 (98%)	NA	11/17 (66%)
[95% Confidence interval]	[66%; 88%]	[74%; 92%]	[96%; 99%]		[43%; 89%]
Fecal incontinence	45/56 (80%)	3/3 (100%)	296/452 (65%)	46/52 (88%)	35/49 (72%)
[95% Confidence interval]	[70%; 91%]	[100%; 100%]	[61%; 70%]	[80%; 97%]	[59%; 85%]
Pain	159/198 (80%)	58/70 (83%)	283/452 (63%)	52/56 (93%)	103/126 (82%)
[95% Confidence interval]	[75%; 86%]	[74%; 92%]	[58%; 67%]	[86%; 100%]	[75%; 89%]

The Propel-study (CllinicalTrials.gov Identifier: NCT00638235) was analyzed accordingly for this table by Dr Alexander Yassouridis.
Follow up at least 1 year after surgery according to the integral theory. For each of the considered cure rates, the 95% confidence intervals (CIs) were calculated. From the 95% CIs, it is to conclude that with a little exception, all cure rates are with a reliability of more than 95% greater than 42%.

Table 2. Prevalence rates (rel. frequencies) of 'moderate or quite a bit' symptom bother before (baseline) and 6, 12 and 24 months after surgery for various PFDI-questions concerning different symptoms

Symptoms and corresponding PFDI questions	Prevalence rates of 'moderate and/or quite a bit' bother before and after surgery								Cochran's Q-tests for testing significance of global effects (*P*-values)	McNemar's tests for identifying (localizing) simple effects (phase pairs)
	Baseline (:=0) *N* = 277		6 months (:=1) *N* = 257		12 months (:=2) *N* = 248		24 months (:=3) *N* = 185			
	abs. frq.	rel. frq.	abs. frq.	rel. frq.	abs. frq.	rel. frq.	abs. frq.	rel. frq.		
Abnormal emptying of the bladder										
11 difficulty emptying the bladder	94	33.90%	18	7.00%	14	5.60%	10	5.40%	*P* < 0,00001	0/1, 0/2, 0/3
12 feeling of not completely emptying the bladder	95	34.30%	23	8.90%	19	7.70%	13	7.00%	*P* < 0,00001	0/1, 0/2, 0/3
13 slow stream, prolonged micturition	93	33.60%	25	9.70%	24	9.70%	18	9.70%	*P* < 0,00001	0/1, 0/2, 0/3
OAB-symptoms										
17 urinary frequency	133	48.00%	31	12.10%	26	10.50%	18	9.70%	*P* < 0,00001	0/1, 0/2, 0/3
18 urgency	131	47.30%	33	12.80%	29	11.70%	26	14.10%	*P* < 0,00001	0/1, 0/2, 0/3
19 urge incontinence	106	38.30%	33	12.80%	29	11.70%	23	12.40%	*P* < 0,00001	0/1, 0/2, 0/3
Nocturia										
27 nocturia	135	48.70%	36	14.00%	48	19.40%	36	19.50%	*P* < 0,00001	0/1, 0/2, 0/3
Obstructive defecation										
8 have to push on the vagina or around the rectum to complete a bowel movement	78	28.20%	24	9.40%	17	6.90%	11	5.90%	*P* < 0,00001	0/1, 0/2, 0/3
9 feeling of need to strain too hard to have a bowel movement	91	32.90%	38	14.80%	29	11.70%	10	5.40%	*P* < 0,00001	0/1, 0/2, 0/3
10 feeling of not completely emptied your bowels?	82	29.60%	28	10.90%	21	8.50%	16	8.60%	*P* < 0,00001	0/1, 0/2, 0/3
fecal incontinence										
38 lose stool well formed (3. degree)	17	6.10%	7	2.80%	10	4.00%	4	2.10%	*P* = 0,03750	0/1, 0/3
39 lose stool loose (2. degree)	49	17.70%	20	7.80%	12	4.80%	9	4.90%	*P* < 0,00001	0/1, 0/2, 0/3
40 lose gas from the rectum (1. degree)	92	33.20%	41	16.00%	32	13.30%	26	14.10%	*P* < 0,00001	0/1, 0/2, 0/3
Pain										
1 pressure in the lower abdomen	91	32.90%	21	8.20%	17	6.90%	10	5.40%	*P* < 0,00001	0/1, 0/2, 0/3
2 pain or discomfort in the lower abdominal or genital area	63	22.70%	22	8.60%	14	5.60%	5	2.70%	*P* < 0,00001	0/1, 0/2, 0/3
3 heaviness or dullness in the pelvic area	75	27.10%	9	3.50%	12	4.80%	5	2.70%	*P* < 0,00001	0/1, 0/2, 0/3
6 pelvic discomfort when standing or physically exerting	113	40.80%	20	7.80%	15	6.00%	9	4.90%	*P* < 0,00001	0/1, 0/2, 0/3
7 pain in lower back most days	105	37.90%	50	19.50%	48	19.40%	33	17.80%	*P* < 0,00001	0/1, 0/2, 0/3

The significance of the treatment effects over time was evaluated statistically with the nonparametric Cochran's Q-tests and McNemar tests. Cochran's Q-tests were first applied to test about significance of the global time effects of surgery on the rel. frequencies of the considered combined symptom outcome (moderate or quite a bit), whereas the McNemar tests were performed only by significant global time effects to localize phase pairs with significant differences in the investigated frequencies.
Black colored *P*-values and/or phase numbers indicate statistical significance at a Bonferroni corrected level of significance $\alpha*$ with $\alpha* < \alpha$ (=0.05), whereas gray colored *P*-values and/or phase numbers denote statistical significance at the nominal level of significance α (=0.05).
Note: Data were gained from the prospective multicenter (10 US and six EU) Propel-study (IRB/EC approved protocol & ICF, ClinicalTrials.gov Identifier: NCT00638235). Women with anterior and/or posterior prolapse stage 2–4 (POP-Q), mostly with apical prolapse, underwent pelvic floor surgery with elevate anterior/apical and/or elevate posterior/apical and were followed over a period of 24 months Symptom assessment with Pelvic Floor disorder inventory questionnaire. The PFDI scale provides for any of its items five possible answers: no symptom, yes-not at all, somewhat, moderate and quite a bit. Data from this study, parts of it published [57,60,61], were statistically analyzed by Dr Alexander Yassouridis.
PFDI, pelvic floor disorder inventory.

(1) insertion of a tension-free tape to create an artificial pubourethral, uterosacral and cardinal neoligament (level 1 repair)
(2) reinforcement of rectovaginal fascia and narrowing the genital hiatus (level 2 repair) and
(3) repair of perineal body and membrane (level 3 repair)

Because of the fact that deficient connective tissue is mainly responsible for prolapse and pelvic floor dysfunction [22,23▪,59], an isolated damage of ligaments represents an exception. In the majority of cases, a descent of pelvic organs is the consequence of both, insufficient support and suspension. In these cases, it makes sense to renew all damaged compartments simultaneously using artificial mesh for damaged ligaments or fascia.

Recent results of this strategy are summarized in Table 1. The diagnostic and surgical procedures described above lead to excellent anatomical results and a cure rate of coexisting symptoms from 42 to 94% [15▪,16,43,44]. Apart from nocturia in the protocol of Caliscan [16] for all other studies and considered symptoms, the 95% confidence intervals (CIs) indicate cure rates being in any case significant greater than 42%. Only nocturia in study [16] revealed for the cure rate a low CI limit of 30%, but this is also highly significant compared with zero (no symptom improvement).

Table 2 summarizes the symptom outcome of POP patients with second-degree to fourth-degree anterior/apical and/or posterior/apical prolapse after mesh-supported pelvic floor surgery using mini-anchors for additional fixation at the sacrospinous ligament [57,60,61]. The data show that surgery leads to a significant reduction of all POP-related symptoms.

CONCLUSION

It is time to accept that numerous symptoms such as abnormal emptying of the bladder, urgency, nocturia, fecal incontinence, obstructed defecation and pelvic pain can be caused by POP due to laxity of the supporting and/or suspending connective tissue structures. This is of upmost importance as laxity can be surgically repaired and symptoms thereby cured or improved in a high percentage. The pathophysiology of pelvic floor dysfunction has thoroughly been described. The main goal and indication for pelvic floor surgery – derived from this knowledge – must be the cure or improvement of symptoms generated by vaginal prolapse. In many cases, so-called simulated operations, as described by Petros [18], can be helpful. Further studies on pelvic floor surgery have to find out, which kind of surgery is best in enabling cure of these bothersome symptoms.

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B.L. has received honorary for data collection during the Propel study of American Medical Systems, which no more exists.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:
- ▪ of special interest
- ▪▪ of outstanding interest

1. Petros PE, Ulmsten U. An integral theory and its method for the diagnosis and management of female urinary incontinence. Scand J Urol Neph 1993; 27 (Suppl. No 153):1–93.
2. Olsen AL, Smith VJ, Bergstrom JO, *et al.* Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol 1997; 89:501–506.
3. Clark A, Gregory T, Smith VJ, *et al.* Epidemiologic evaluation of reoperation for surgically treated pelvic organ prolapse and urinary incontinence. Am J Obstet Gynecol 2003; 189:1261–1267.
4. U.S. Food and Drug Administration. FDA executive summary: urogynecologic surgical mesh- update on the safety and effectiveness of transvaginal placement for pelvic organ prolapse. http://www.fda.gov/downloads/MedicalDevices/Safety/AlertsandNotices/UCM262760.pdf; 2011. (Accessed 21 December 2014).
5. Abrams P, Cardozo L, Fall M, *et al.* The standardisation of terminology of lower urinary tract function: report from the standardisation sub-committee of the ICS. Neurourol Urodyn 2002; 21:167–178.
6. Fall M, Baranowski AP, Elneil S, *et al.* Guidelines on chronic pelvic pain. Eur Urol 2010; 57:35–48.
7. Farnsworth BN. Posterior intravaginal slingplasty (infracoccygeal sacropexy) for severe posthysterectomy vaginal vault prolapse: a preliminary report on efficacy and safety. Int Urogynecol J Pelvic Floor Dysfunct 2002; 13:4–8.
8. Goeschen K, Gent HJ. Das posteriore fornix syndrom. Frauenarzt 2004; 45:104–112.
9. Bjelic-Radisic V, Hartmann G, Abendstein B, *et al.* The posterior intravaginal slingplasty operation: results of the Austrian registry. Eur J Obstet Gynecol Reprod Biol 2009; 144:88–91.
10. Capobianco G, Donolo E, Wenger JM, *et al.* Efficacy and 9 years' follow-up of posterior intravaginal slingplasty for genital prolapse. J Obstet Gynaecol Res 2014; 40:219–223.
11. Cosma S, Preti M, Mitidieri M, *et al.* Posterior intravaginal slingplasty: efficacy and complications in a continuous series of 118 cases. Int Urogynecol J 2011; 22:611–619.
12. Kolusari A, Yildizhan R, Adali E, *et al.* Short-term results of posterior intravaginal slingplasty in grade 4 uterine prolapse. Arch Gynecol Obstet 2010; 281:55–58.
13. Lee YS, Han DH, Lee JY, *et al.* Anatomical and functional outcomes of posterior intravaginal slingplasty for the treatment of vaginal vault or uterine prolapse: a prospective, multicenter study. Korean J Urol 2010; 51:187–192.
14. Neuman M, Lavy Y. Posterior intra-vaginal slingplasty for the treatment of vaginal apex prolapse: medium-term results of 140 operations with a novel procedure. Eur J Obstet Gynecol Reprod Biol 2008; 140:230–233.
15. ▪ Goeschen K. Posterior fornix syndrome: comparison of original (2004) and modified (2015) post-PIVS anatomic and symptomatic results – a personal journey. Pelviperineology 2015; 34:85–91; http://www.pelviperineology.org/march-2015/pdf/pelviperineology-march-2015-hd.pdf.
An interesting overview about the posterior fornix syndrome with own experiences over a long period of peronal surgery.
16. Caliskan A, Goeschen K, Zumrutbas AE. Long term results of modified posterior intravaginal slingplasty (P-IVS) in patients with pelvic organ prolapse. Pelviperineology 2015; 34:94–100.

17. Milsom D, Altman R, Cartwright MC, *et al.* Epidemiology of Urinary Incontinence (UI) and other Lower Urinary Tract Symptoms (LUTS), Pelvic Organ Prolapse (POP) and Anal Incontinence (AI) Chapter in Incontinence, 5th International Consultation on Incontinence. Abrams P, Cardozo L, Khoury S, Wein A, editors. , 5th Ed. ICUD-EAU; 2013. p. 57.
18. Petros P. The anatomy and dynamics of pelvic floor function and dysfunction. In: Petros P, editor. The Female Pelvic Floor, 3rd Ed. Heidelberg: Springer; 2010. pp. 17–77.
19. Goeschen K, Petros P. Der weibliche Beckenboden. Heidelberg-New York: Springer; 2008.
20. Yamada H. Aging rate for the strength of human organs and tissues. In: Evans FG, editor. Strength of Biological Materials. Baltimore: Williams & Wilkins Co; 1970. pp. 272–280.
21. Gordon AM, Huxley AF, Julian FJ. The variation in isometric tension with sarcomere length in vertebrate fibres. J Physiol 1966; 184:170–192.
22. Liedl B, Markovsky O, Wagenlehner F, *et al.* The role of altered connective tissue in the causation of pelvic floor symptoms. In: Ammar Alhasso, Ashani Fernando, editors. Urinary Incontinence. Rijeka: Intech; 2012. pp. 1–20.
23. ■ Liedl B, Riegel M, Markovsky O. Pathophysiology of urinary incontinence and pelvic floor dysfunctions in females. In: Chung E, editor. Urinary Incontinence. Nova Science Publishers; 2013. pp. 1–18.

An interesting insight into the pathophysiology of prolapse-induced anorectal dysfunction.

24. Petros P, Ulmsten U. Role of pelvic floor in bladder neck opening and closure: I. Muscle forces. Int Urogynecol J 1997; 8:74–80.
25. ■■ Bush MB, Liedl B, Wagenlehner F, *et al.* A finite element model validates an external mechanism for opening of the urethral tube prior to micturition in the female. World J Urol 2015; 33:1151–1157.

An outstanding analysis that shows how voiding without contraction of the posterior pelvic floor muscles would need 100-fold bladder pressure.

26. ■■ Goeschen K. Role of uterosacral ligaments in the causation and cure of chronic pelvic pain syndrome. Pelviperineology 2015; 34:2–20; http://www.pelviperineology.org/march-2015/pdf/pelviperineology-march-2015-hd.pdf.

An outstanding review article about the causation of prolapse-induced pelvic pain.

27. Martius H. Lehrbuch der Gynäkologie. Stuttgart: Thieme; 1946.
28. Pescatori M, Spyrou M, Pulvirenti d'Urso A. A prospective evaluation of occult disorders in obstructed defecation using the 'iceberg diagram'. Colorectal Dis 2007; 9:452–456.
29. Bartoli S, Aguzzi G, Tarricone R. Impact on quality of life of urinary incontinence and overactive bladder: a systemic literature review. Urology 2010; 75:491–500.
30. Kelleher CJ, Cardozo LD, Khullar V, Salvatore S. A new questionnaire to assess the quality of life of urinary incontinent women. Br J Obst Gynaecaol 1997; 104:1374–1379.
31. Zorn BH, Montgomery H, Pieper K, *et al.* Urinary incontinence and depression. J Urol 1999; 162:82–84.
32. Brown JS, Vittinghoff E, Wyman JF, *et al.* Urinary incontinence: does it increase risk for falls and fractures? Study of Osteoporotic Fractures Research Group Collaboration. J Am Ger Soc 2000; 48:721–725.
33. Milsom I, Abrams P, Cardozo L, *et al.* How widespread are the symptoms of overactive bladder and how are they managed? A population-based prevalence study. Br J Urol Int 2001; 87:760–766.
34. Stewart WF, Van Rooyen JB, Cundiff GW, *et al.* Prevalence and burden of overactive bladder in the United States. World J Urol 2003; 20:327–336.
35. Haylen BT, de Ridder D, Freeman RM, *et al.* An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn 2010; 29:4.
36. Gormley EA, Lightner DJ, Faraday M, *et al.* American Urological Association; Society of Urodynamics, Female Pelvic Medicine. Diagnosis and treatment of overactive bladder (nonneurogenic) in adults: AUA/SUFU guideline amendment. J Urol 2015; 193:1572–1580.
37. Pucciani F. Descending perineum syndrome: new perspectives. Techn Coloproctol 2015; 19:443–448.
38. Francheschelli L, Varvaras D, Capuano I, *et al.* Laparoscopic ventral rectopexy using biologic mesh for the treatment of obstructed defaecation syndrome and/or faecal incontinence in patients with internal rectal prolapse: a critical appraisal of the first 100 cases. Techn Coloproctol 2015; 19:209–219.
39. Breivik H, Collett B, Ventafridda V, *et al.* Survey of chronic pain in Europe: prevalence, impact on daily life, and treatment. Eur J Pain 2006; 10:287–333.
40. Petros P. Severe chronic pelvic pain in women may be caused by ligamentous laxity in the posterior fornix of the vagina. Aust NZ J Obstet Gynaecol 1996; 36:351–354.
41. Petros P. New ambulatory surgical methods using an anatomical classification of urinary dysfunction improve stress, urge, and abnormal emptying. Int J Urogynecol 1997; 5:270–278.
42. Petros P, Richardson PA. TFS posterior sling improves overactive bladder, pelvic pain and abnormal emptying, even with minor prolapse: a prospective urodynamic study. Pelviperineology 2010; 29:52–55; http://www.pelviperineology.org/june_2010/index_vol29_2.html.
43. Müller-Funogea A. Posterior fornix syndrome: a new urogynecologic entity. Ethiopathogenesis and proposal for surgical therapy. Thesis. Romania: Medical University of Bucharest; 2014.
44. Inoue H, Kohata Y, Sekiguchi Y, *et al.* The TFS minisling restores major pelvic organ prolapse and symptoms in aged Japanese women by repairing damaged suspensory ligaments 12-48 month data. Pelviperineology 2015; 34:79–83; http://www.pelviperineology.org/september-2015/pdf/Pelviperineology-september-2015-hd.pdf.
45. ■ Gold DM, Goeschen K. Application of the Pescatori iceberg to 198 patients presenting with chronic pelvic pain before and after posterior sling surgery. Pelviperineology 2017; in press.

An interesting article upon the variability of pelvic floor dysfunctions.

46. Baujahr B, Tchartchian G, Waldschmidt M, *et al.* Laparoscopic sacropexy: a retrospective analysis of the subjective outcome in 310 cases. Obstet Gynecol Int 2012; 2012:538426. doi:10.1155/2012/538426.
47. Ganatra AM, Rozet F, Sanchez-Salas R, *et al.* The current status of laparoscopic sacrocolpopexy: a review. Eur Urol 2009; 55:1089–1103.
48. Agarwala N, Hasiak N, Shade M. Laparoscopic sacral colpopexy with Gynemesh as graft material-experience and results. J Minim Invasive Gynecol 2007; 5:577–583.
49. Sundaram CP, Venkatesh R, Landman J, *et al.* Laparoscopic sacrocolpopexy for the correction of vaginal vault prolapse. J Endourol 2004; 18:620–623.
50. Ross JW, Preston M. Laparoscopic sacrocolpopexy for severe vaginal vault prolapse: five-year outcome. J Minim Invasive Gynecol 2005; 12:221–226.
51. Higgs PJ, Chua HL, Smith ARB. Long term review of laparoscopic sacrocolpopexy. BJOG 2005; 112:1134–1138.
52. Nygaard LE, McCreery R, Brubaker L, *et al.* Abdominal sacropexy: a comprehensive review. Obstet Gynecol 2004; 104:805–823.
53. Baessler K, Schuessler B. Abdominal sacropexy and anatomy and function of the posterior compartment. Obstet and Gynecol 2001; 97:678–684.
54. Maher CF, Qatawneh AM, Dwyer PL, *et al.* Abdominal sacral colpopexy or vaginal sacrospinous colpopexy for vaginal vault prolapse: a prospective randomized study. Amer J Obstet Gynecol 2004; 190:20–26.
55. Maher C, Feiner B, Baessler K, *et al.* Surgical management of pelvic organ prolapse in women. Cochrane Database Syst Rev 2010; 4:CD004014.
56. Petros P, Swash M. Sacrocolpopexy may cause difficult defecation by inhibiting the external opening mechanism. Int Urogynecol J 2011; 4:CD004014.
57. Liedl B, Lukban J, Stanford E, *et al.* Besserung von Symptomen der Stuhlinkontinenz durch Beckenbodenrekonstruktion mit elevate posterior/apical und elevate anterior/apical. Coloproct 2016; 38:75–76.
58. DeLancey JO. Anatomic aspects of vaginal eversion after hysterectomy. Am J Obstet Gynecol 1992; 166:1717–1724.
59. Petros P, Swash M. The musculo-elastic theory of anorectal function and dysfunction. Pelviperineology 2008; 27:89–93.
60. Lukban JB, Roovers JP, VanDrie DM, *et al.* Single-incision apical and posterior mesh repair: 1 year prospective outcomes. Int Urogynecol J 2012; 23:1413–1419.
61. Stanford EJ, Moore RD, Roovers JP, *et al.* Elevate anterior/apical: 12 month data showing safety and efficacy in surgical treatment of pelvic organ prolapse. Female Pelvic Med Reconstr Surg 2013; 19:79–83.