

# Paravaginal defects and stress urinary incontinence

PETRI ECKHARD<sup>1</sup>, ASHOK KIRAN<sup>2</sup>

<sup>1</sup>Division of Urogynecology, Department of Obstetrics and Gynecology, University of Greifswald, Germany

<sup>2</sup>Clinical Fellow in Urogynecology, University of Greifswald, Germany.

**Abstract: Objective:** To analyze the literature on the role of paravaginal defects in pathophysiology and management of stress urinary incontinence and to formulate an idea on the management according to the underlying pathophysiology in each individual patient with stress urinary incontinence. **Materials and Methods:** This is a clinical review of literature on paravaginal defects. A medline search was performed using the Medical Subject Headings (MeSH) terms “Paravaginal defect”, “stress urinary incontinence”, and “Colposuspension”. Literature from the year 1990 to 2010 is reviewed. Anatomical, clinical, imaging and surgical evidences for the existence and role of paravaginal defects is summarized and analyzed. Role of colposuspension in correcting paravaginal defects and stress urinary incontinence is discussed. **Results:** Paravaginal defects are real entities and play an important role in the patho-physiology of stress urinary incontinence. Current clinical practice ignores the anatomical causes underlying the causation of stress urinary incontinence and instead the use of a single procedure (in most cases mid-urethral slings) for all women with stress incontinence is becoming a trend. **Conclusion:** Evaluation of a woman with stress urinary incontinence should include searching for possible underlying causes (anatomical and functional) and management should be based accordingly. In the presence of paravaginal defect, a colposuspension will be a better management option. Urethral hypermobility (or a hypotonic urethra) in the absence of a paravaginal defect may be treated with mid-urethral slings.

**Key words:** Paravaginal defect; Stress urinary incontinence; Colposuspension; Mid-urethral slings.

## INTRODUCTION

The diversity of the pathologies of the pelvic floor, their origin, their degree, and their clinical relevance makes comparison of studies on diagnosis and treatment of pelvic floor disorders difficult. The focus of research in recent years was the search of a numeric classification. The description of single compartments and stages of their descent disregards quality of tissue and complex pathologies of the pelvic tissues. Even though, the entity “Paravaginal defects” is known for decades, its clinical importance is under discussion. In the last decade, a rapidly growing number of novel surgical techniques are being introduced into daily clinical practice to cure female stress urinary incontinence (SUI). Looking at abstracts of major meetings and publications, it seems that alloplastic slings are the solution for everything and these have replaced other well established procedures. The widespread acceptance of tension-free slings worldwide apparently forced colposuspension onto sidelines, even though it is accepted as an effective procedure in the long term cure of SUI by Cochrane collaboration and other groups. The aim of this paper is to analyze the literature on the role of paravaginal defects (at the level of urethra) in the pathophysiology and management of stress urinary incontinence and to formulate an idea on the management according to the underlying pathophysiology in each individual patient with stress urinary incontinence. Paravaginal defect (PVD) is defined as the medial displacement of the vaginal wall and pubocervical fascia (PCF) from its normal line of attachment to the pelvic sidewall at the Arcus tendinus fascia pelvis (ATFP).<sup>1</sup> As early as 1909, George White described attachments and supports of vagina and their role in the development of cystocele. White describes “.....the real support of the vagina comes from its attachment to the white line of the pelvic fascia, and especially a thick bundle of fibers attached to the spine of the ischium and radiating out on both the anterior and posterior surfaces of the vagina. ... a cystocele is caused by the breaking loose of the vagina from the white line, which can readily occur during labor and especially in an instrumental delivery”.<sup>2</sup> The existence of pubocervical fascia as a separate structure (or sheath) is disputed. However, most authors agree that the structural layer that supports the bladder is composed of the anterior vaginal wall and its attachment through the endopelvic fascia to the pelvic wall.<sup>3</sup> Whether it is the detachment of pub-

ocervical fascia or the detachment of anterior vaginal wall (through its attachment to the endopelvic fascia) from the ATFP, it is purely a matter of nomenclature. The process of detachment of the supporting structure from ATFP (or broadly lateral pelvic wall), called paravaginal defect, is undisputed. Paravaginal defects may occur at the level of urethra (when it can cause urethral hypermobility) (Figure 1) or bladder (causing lateral cystocele) or at both levels.

## MATERIALS AND METHODS

This is a clinical review of literature on paravaginal defects in stress urinary incontinence. A medline search was performed using the MeSH terms “Paravaginal defect”, “stress urinary incontinence”, and “Colposuspension”. Literature from the year 1990 to 2010 is reviewed. Anatomical, clinical, imaging and surgical evidences for the existence and role of paravaginal defects is summarized and analyzed. Role of colposuspension in correcting paravaginal defects and stress urinary incontinence is discussed.

## RESULTS AND DISCUSSION

### *Anatomical studies on paravaginal defects*

The arcus tendineus fasciae pelvis (ATFP), or white line, attaches to the pubic bone 1 cm lateral to midline and 1 cm superior to its inferior margin. The arcus tendinus levator ani (ATLA - tendinous origin of levator ani muscle from the lateral pelvic wall) originates from the pubic bones several centimeters above the origin of the ATFP. As the ATFP fans out from its origin, it lies on the inner surface of the levator ani fascia. According to Haderer et al. the most common ‘paravaginal’ defect occurs when the ATFP peels away from the levator ani fascia. This may also occur after the ATLA detaches from the pubic bone or, more rarely, from the ischial spine.<sup>4</sup> Because the endopelvic fascia provides support to the anterior vaginal wall and urethra, detachment of its lateral attachments can have a deleterious effect on the support of the urethrovesical junction. It is to be emphasized that the lateral portions of the pubourethral ligaments fuse strongly with the fascia of levator ani at ATLA.<sup>5</sup> Considering this fact, any detachment of ATLA also causes loss of support to the pubourethral ligaments and hence a failure of urethral suspensory mechanism. Contraction of

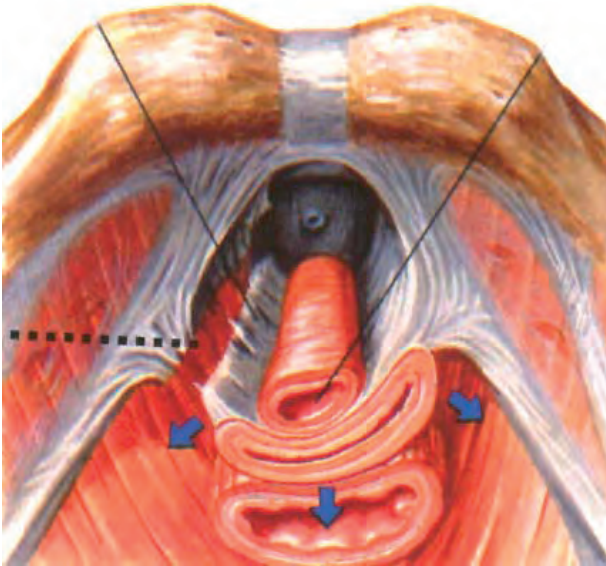


Figure 1. – Illustration of paravaginal defect from abdominal route.

the levator ani muscles is a requisite for the normal maintenance of urethral support. Levator ani muscles support the urethra through the endopelvic fascia.<sup>6</sup> If there is a break in the attachment of this endopelvic fascia to the levator ani muscle, then urethral support during increased intra-abdominal pressure is lost and may result in stress urinary incontinence.

#### Imaging studies on paravaginal defects

The first report on imaging of paravaginal defects, were described by Huddleston et al. in a magnetic resonance imaging (MRI) study of 12 women with cystourethrocele and stress urinary incontinence.<sup>7</sup> They performed MRI scans pre and post operatively on the 12 women. They describe “Mustache” sign formed by white fat in prevesical space against bilateral sag of detached vagina from arcus tendineus fascia pelvis, and “saddlebags” or “paddle” sign caused by displaced fluid-filled bladder wall into bilateral paravaginal defects. The authors were able to demonstrate a correlation between the preoperative MRI scans and the intraoperative findings. Furthermore, there was disappearance of the defects in the postoperative (all patients underwent abdominal paravaginal repair) MRI scans in all but two patients who had surgical failure. The persistence of the defects in the patients who were failures seems to be confirmation of the possible value of this technique to predict procedure failures. De Souza et al. compared paravaginal fascial volume in 11 continent women and 10 women with stress incontinence using MRI with endovaginal coil.<sup>8</sup> They documented the presence of high-signal-intensity tissue and its extent in relation to the urethra (enveloping or extending lateral to urethra). The paravaginal fascia (connective tissue containing venous plexus) was identified as a hyperintense structure surrounding the vaginal wall anteriorly, laterally, and posteriorly with variable thickness on both T2-weighted and short inversion time inversion-recovery MRI images. It was proposed that the high signal intensity of this tissue was a result of its vascularity. They reported that extension of paravaginal fascia either totally or laterally around the urethra was present in all reference subjects but in only 50% (five of 10) of SUI patients. Also the mean urethral paravaginal fascial volume was reduced in patients with SUI ( $3.5 \text{ cm}^3 \pm 2.0$ ) compared with that in reference subjects ( $5.3 \text{ cm}^3 \pm 0.6$ ,  $P = .017$ ). Although this study does not provide a direct evidence for the presence of paravaginal de-

fect, it gives an indirect clue to the role of paravaginal fascia in maintaining continence. Tunn et al. studied morphology of levator ani, endopelvic fascia and urethra in 54 women with SUI using MRI in supine position.<sup>9</sup> Lateral fascial defects were identified on both sides in 31% of the women ( $n = 17$ ) and on one side in 15% ( $n = 8$ ), as suggested by absence of a low-intensity tissue bridge indicating fusion of the lateral vaginal wall and levator ani muscle at the level of the middle urethra. They also reported central defects of the endopelvic fascia in 39% ( $n = 21$ ), and levator abnormalities such as unilateral loss of substance in 30%, a higher signal intensity in 28%, and altered origin in 19% of the patients. An interesting finding in this study was a significant association between loss of the symphyseal concavity of the anterior vaginal wall and lateral fascial defects ( $p = 0.001$ ) and levator ani changes ( $p = 0.016$ ). It is to be noted that some of the morphological changes observed in this study, can also occur because of normal variations in individual anatomy. Even in healthy, nulliparous women, variations in morphology of levator ani and attachment of endopelvic fascia to the pelvic side wall are documented. Tunn et al. remark that it is difficult to ascertain whether the abnormal morphology in a particular patient is a result of pathological injury sustained during labor, or an incomplete development of major anatomical components of continence support system.<sup>10</sup> In a prospective study, El Sayed et al. used dynamic MRI to analyze association of SUI, pelvic organ prolapse (POP) and anal incontinence with specific pelvic floor structural abnormalities.<sup>14</sup> In this well designed study of 59 women, 15 nulliparous women were taken as controls and the remaining 44 women were assigned to four groups depending on their symptoms (POP without SUI,  $n = 10$ ; SUI without POP,  $n = 10$ ; SUI with bladder and/or genital prolapse  $n = 16$ ; and anal incontinence associated with POP,  $n = 8$ ). This study reported that POP was associated with levator muscle weakness in 16 (47%) of 34 patients, with level I and II fascial defects in seven (21%) of 34 patients, and with both defects in 11 (32%) of 34 patients. SUI was associated with defects of the urethral supporting structures in 25 (86%) of 29 patients but was not associated with bladder neck descent. Of the 26 women with SUI, 12 had isolated paravaginal defects, 2 had isolated central defects and 10 had combined central and paravaginal defects. Ultrasound imaging has also been used for studying pelvic floor structures. Ostrzensky and Osborne were the first to report imaging of paravaginal defects using ultrasound.<sup>11</sup> Early studies used abdominal ultrasound to image PVD, but this method was not found to be reliable because of the confounding effect of bladder filling, uterine position and size. Imaging of paravaginal support structures requires axial or transverse view of pelvis which can be obtained by 3D ultrasound. Tenting of the vagina (presence of ventrolateral vaginal groove) is considered to represent normal paravaginal support and any loss

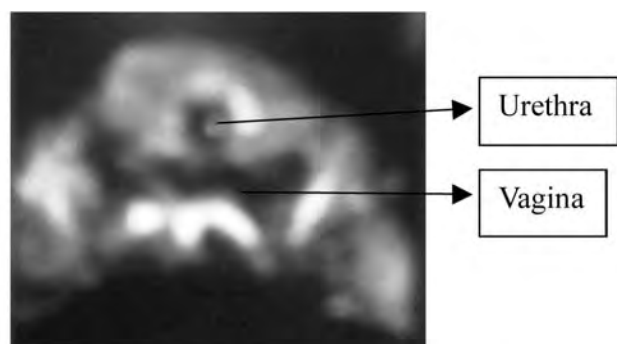


Figure 2. – 3D Perineal ultrasound showing loss of antero-lateral vaginal sulci-indicating bilateral paravaginal defect.



Figure 3. – Clinical demonstration of paravaginal defect.

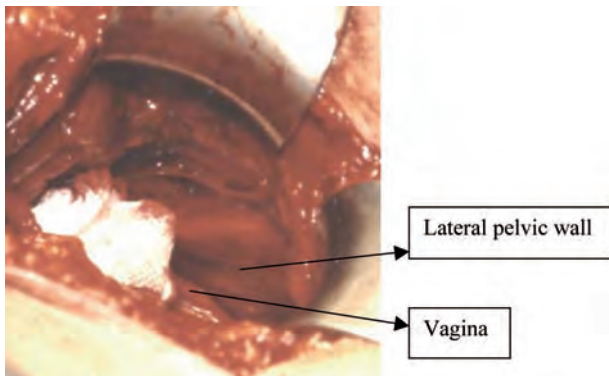


Figure 4. – Intra-operative view showing the gap between vagina and lateral pelvic wall.

of tenting is an indication of PVD (Figure 2). Dietz et al. compared paravaginal supports in 26 nulliparous women before and after 2-5 months of delivery.<sup>12</sup> Tenting was visible in all women antenatally, and in five of 21 women studied postnatally. Interestingly, there was no significant correlation between absence of tenting and bladder neck descent and the authors concluded that in some women delivery-related changes may be due to attenuation rather than disruption of structures. This study was limited by insufficient power to detect differences and lack of standardization and quantification of paravaginal support and defects. Further, the predictive value of bladder neck mobility in stress urinary incontinence is poor as demonstrated by Ultrasound and MRI studies.<sup>13,14</sup> Dietz et al. analyzed correlation between clinical detection and 3D ultrasound detection of paravaginal defects in women with anterior vaginal wall descent and reported that pelvic floor ultrasound in midsagittal, axial or coronal planes does not correlate well with clinical assessment for PVD. This result is not surprising considering the fact that clinical detection (during vaginal examination) of PVD has poor sensitivity<sup>15</sup> when compared with intra-operative detection. Further studies are required to correlate identification of PVD in imaging studies to intra-operative detection (which is the gold standard to detect



Figure 5. – Colposuspension – attachment of lateral vagina to the Cooper's ligament.

PVD) which may help in formulating guidelines for the management of SUI based on underlying morpho-pathological changes.

#### Clinical and surgical studies

A woman with paravaginal defect may present with complaints of SUI, protrusion of vaginal walls, involuntary leak of urine during intercourse, or colpophony. Colpophony was first described by Kohorn in the year 2000.<sup>16</sup> Colpophony or vaginal wind or vaginal noise is defined as a noise, typically related to the back passage, occurring during physical activity such as vaginal examination or intercourse. Clinically presence of PVD is indicated by the following signs: descent of anterior vaginal wall with intact rugae, absence of anterior vaginal groove on straining and restoration of normal position of a descended anterior vaginal wall upon supporting the anterolateral groove (Figure 3). Further confirmation of the role of a paravaginal defect in cystocele can be confirmed by supporting the lateral vaginal groove which causes restoration of normal position of the anterior vaginal wall to its normal position.

There is wide inter-observer and intra-observer variability in clinically detecting the presence of PVD.<sup>17</sup> Also clinical examination is a poor predictor for the actual presence of PVD noted intra-operatively.<sup>18,19</sup> Intra-operative identification of PVDs has been the gold standard for detecting PVDs. From the abdominal route, PVD can be seen as detachment of lateral edge of pubocervical fascia (PCF) (white structure with linear vessels running along the edge) from the ATFP, when bladder is pushed slightly away from the pelvic side wall (Figure 4). Surgical approximation of the detached edge of the PCF to ATFP is termed as Paravaginal repair (PVR). ATFP may not always be visible or accessible and in such cases, PCF can be attached to the obturator fascia. This method can be used to treat anterior vaginal wall prolapse and SUI. PVR for the treatment of SUI has lower success rates compared to Burch colposuspension, where the paraurethral fascia is attached to the iliopectineal (Cooper's) ligament on either side (Figure 5).<sup>20</sup> In a randomized controlled trial of Burch colposuspension versus PVR with follow-up of 1-3 years, Colombo et al. reported that the

subjective and objective cure rates favored the Burch colposuspension over the paravaginal repair: 100% versus 72% ( $p = 0.02$ ) and 100% versus 61% ( $p = 0.004$ ), respectively.<sup>21</sup> The higher success rate of Burch colposuspension compared to PVR in treating SUI could be because of provision of stronger point of attachment of the iliopectineal ligament as compared to ATFP.<sup>22</sup> Also, in some women ATFP may not be well formed, or ATFP itself might be detached from lateral pelvic wall.<sup>23,1</sup> It is nevertheless, a fact that Burch colposuspension corrects an existing paravaginal defect at the level of urethra. Presently there is level 1A evidence that Burch colposuspension is an effective procedure for the surgical management of SUI providing long term durability.<sup>24,25</sup> Midline fascial defects, attenuation of endopelvic fascia and intrinsic sphincter deficiency are the other proposed pathophysiologic mechanisms for the development of SUI. At present, all these defects can be visualized by MRI<sup>14</sup> and intrinsic sphincter deficiency can be documented by urodynamic studies.

We have observed an increasing incidence of obstructive voiding in patients having midurethral slings in presence of paravaginal defects (unpublished data). In our audit of 351 cases of complications of mid-urethral slings, 153 patients (44%) had underlying paravaginal defects. PCF is a broad trampoline like structure that, to some extent, supports pelvic floor against intra-abdominal pressure. It is easy to deduct that inserting a midurethral sling in presence of a PVD will lead to intra-abdominal pressure being transmitted entirely on the sling (and not uniformly across the PCF). This can lead to obstructive voiding symptoms. Given this scenario, it is our opinion that it will be illogical to place a midurethral tape in a woman with documented PVD. An important advance in the management of SUI would be to identify the underlying structural abnormality using ultrasound imaging, and functional disturbance using urodynamics, and tailor the treatment accordingly. For example, in a stress incontinent woman with midline fascial defect/attenuation of endopelvic fascia, a midurethral sling would be an ideal choice. On the other hand a stress incontinent woman with PVD would definitely benefit from retropubic colposuspension. Such an approach based on an understanding of pathophysiology would be logical and will definitely help to avoid many of the complications encountered with mid urethral slings.

**Colposuspension** – An effective way of correcting SUI due to paravaginal defect and urethral hyper mobility

The aim of colposuspension is restoration of urethral function and maintenance of continence by positioning and well supporting the proximal urethra with in the abdominal cavity, thereby raising the pressure transmission ration over 100% in the upper third of the urethra. Downward and backward movement of the bladder neck during stress (urethral hypermobility) is supposed to be an important cause of SUI. It is sufficient to elevate paravaginal tissue just to achieve some support for the bladder neck area; attempts to completely approximate vaginal wall to cooper's ligament area is likely to induce voiding difficulties, de novo urge symptoms and does not achieve long lasting results. In our practice indications for colposuspension include primary and secondary urethral incompetence in the presence of good vaginal mobility and capacity, mainly in presence of a paravaginal defect. In patients with MUCP<20 cm H<sub>2</sub>O colposuspension is more likely to fail.

Evidences for efficacy of colposuspension: Since the early description of the Classical Burch,<sup>26,27</sup> many variations in the procedural aspects of colposuspension have been described in literature.<sup>28,29,30</sup> In our experience with more than 250 re-operations after "Burch" in other institutions, more

than 90% have been anything but Burch colposuspensions. The many modifications and sub-modifications explain the wide variation in success and complication rates published, because very different procedures have been performed. A recent Cochrane evaluation of open retro-pubic colposuspension included 46 trials involving a total of 4738 women and reported an overall cure rates were 68.9% to 88.0% for open retropubic colposuspension with a few studies demonstrating lower failure rates than with conservative treatment, needle suspension, Marshall Marchetti Krantz procedure and anterior colporrhaphy. One important finding in this meta-analysis was that the benefit of colposuspension was maintained over time (RR of failure 0.51; 95% CI 0.34 to 0.76 before the first year, RR 0.43; 95% CI 0.32 to 0.57 at one to five years, RR 0.49; 95% CI 0.32 to 0.75 in periods beyond 5 years).<sup>31</sup> Even though Bombieri and Freeman stated that the position of the bladder neck and the amount of elevation do not influence the continence outcome, majority of authors using perineal or introital ultrasound found significant differences in bladder neck position, mobility at rest and during valsalva maneuver, funneling and urethral angles correlating with success or failure.<sup>32,33,34</sup> Using introital ultrasound it was demonstrated that over-elevation resulted in signs of over active bladder.<sup>34</sup>

**Personal experience of the senior author:** The senior author (EP) started with colposuspension after having seen the technique impressively demonstrated by Stuart Stanton, Sir Richard Turner-Warwick, Emil Tanagho and others in the late 70s and early 80s. He has performed more than 4600 colposuspensions in the last 30 years. Even though it was impossible to follow-up all these patients over such a long period of time, over the years, major unselected groups have been followed, looking for short term and long-term complications as well as subjective and objective success rates and satisfaction of the patients. In spite of the introduction of mid-urethral slings and injectables, the number of colposuspensions remains stable for many years in a specialized referral center in which 60% of colposuspensions are done for recurrent incontinence after mid-urethral slings. A randomized control trial of midurethral slings versus colposuspension in patients with SUI and paravaginal defects was planned. Within a short time it was observed that patients in the midurethral sling group had increasing incidence of lower urinary tract obstruction and other complications. Further continuation of this study was deemed to be unethical and the trial was terminated prematurely.

The available evidence indicates that open colposuspension is one of the most effective treatment modality for SUI especially in the long term. In competition with mid-urethral slings, colposuspension will remain the first choice for all laparotomies necessitated by other pathologies, especially in cases with paravaginal defects and in women with overactive bladders caused by anatomic defects.

## CONCLUSION

Paravaginal defects are real entities and play an important role in the pathophysiology of stress urinary incontinence. Current clinical practice ignores the anatomical causes underlying the causation of stress urinary incontinence and instead, the use of a single procedure (in most cases mid-urethral slings) for all women with stress incontinence is becoming a trend. Studies are needed to standardize the ultrasound detection, establish its efficacy and cost-effectiveness before ultrasound can be routinely used for pre-op diagnosis. Evaluation of a woman with stress urinary incontinence should include searching for possible underlying causes (anatomical and functional) and management should be

based accordingly. In the presence of paravaginal defect, a colposuspension will be a better management option. Urethral hyper mobility in the absence of a paravaginal defect may be treated with mid-urethral slings.

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Correspondence to:

ECKHARD PETRI  
Division of Urogynecology  
Department of Obstetrics and Gynecology  
University of Greifswald  
Ferdinand-Sauerbruch-Strasse  
17475 Greifswald, Germany.  
e-mail: profpetri@gmx.de