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Surgical Reconstruction of Pelvic Floor Descent: Anatomic and Functional Aspects

Key Words

Pelvic floor · Functional anatomy · Urinary incontinence · Prolapse · Surgical reconstruction

Abstract

Introduction: The human pelvic floor is a complex structure and pelvic floor dysfunction is seen frequently in females. **Materials and Methods:** This review focuses on the surgical reconstruction of the pelvic floor employing recent findings on functional anatomy. A selective literature research was performed by the authors. **Results:** Pelvic floor activity is regulated by 3 main muscular forces that are responsible for vaginal tension and suspension of the pelvic floor organs, bladder and rectum. A variety of symptoms can derive from pelvic floor dysfunctions, such as urinary urge and stress incontinence, abnormal bladder emptying, fecal incontinence, obstructive bowel disease syndrome and pelvic pain. These symptoms mainly derive, for different reasons, from laxity in the vagina or its supporting ligaments as a result of altered connective tissue. Pelvic floor reconstruction is nowadays driven by the concept that in case of pelvic floor symptoms, restoration of the anatomy will translate into restoration of the physiology and ultimately improve patients' symptoms. **Conclusion:** The surgical reconstruction of the anatomy is almost exclusively focused on the restoration of the lax pel-

vic floor ligaments. Exact preoperative identification of the anatomical lesions is necessary to allow for exact anatomical reconstruction with respect to the muscular forces of the pelvic floor.

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Introduction

Pelvic floor disorders are frequently seen in women. A variety of symptoms can be attributed to pelvic floor lesions, such as stress urinary incontinence, urge urinary incontinence, pollakisuria, nocturia, abnormal bladder emptying, fecal incontinence, obstructive bowel disease syndrome and pelvic pain. The prevalence of such pelvic floor disorders range between 10 and 25% depending on the patient population, type of study and definition used. Combinations of symptoms are frequent. In one study investigating 4,103 women, 67% exhibited more than 1 symptom. Risk factors in that study were obesity, more than 1 vaginal delivery, hysterectomy and the need for hormonal treatment [1].

One of the most important findings in the last years was that disorders of the pelvic floor muscular functions can negatively influence the physiological functions of the pelvic organs, namely the urinary bladder and the

rectum [2–5]. Thus, storage and voiding dysfunctions of the bladder and rectum can be explained by anatomical lesions within the connective tissue structures of the pelvic floor. Following these assumptions, restoration of the anatomy will therefore lead to restoration of function and physiology, as it has been elaborated in the Integral Theory [5]. Based on this, surgical reconstruction of pelvic floor defects is a rational basis for the treatment of pelvic disorders. In order to correctly apply pelvic floor surgery, the specific correlation between pelvic floor anatomy and physiology needs to be understood.

Pelvic Floor Activity

Driven into the upright position by human evolution, the human pelvic floor has experienced a significant alteration in its importance and function. The female pelvic floor enables support of the pelvic organs and provides multiple functions, such as urinary continence, bladder emptying, fecal continence, defecation, sexual intercourse and child birth. The pelvic floor itself has a 3-dimensional muscular architecture, which essentially contracts against a net of ligaments and fascias. The pelvic floor and its organs have multiple afferent and efferent neuronal systems intimately linking it to the central nervous system. Damage at any level (central nervous system, peripheral nervous system, organs and/or pelvic floor) can cause symptoms; however, the most important damage are anatomical lesions on the pelvic floor itself. Such damage can be repaired surgically. In the last years, the pelvic floor has increasingly become better understood with the result that pelvic floor surgery has been changing markedly.

The pelvic floor quickly responds to changes in intra-abdominal pressure or visceral weight. This has been elegantly investigated by Shafik et al. [6], who looked at pelvic floor activity in patients undergoing laparoscopic cholecystectomy. The pelvic floor activity was evaluated by investigating the electromyographic activity of the levator ani muscle, which is an important part of the pelvic floor musculature and is divided into the pubococcygeus muscle ventrally and the levator plate dorsally. In these patients, the intra-abdominal pressure was elevated by CO₂ insufflation in increments of 5 cm H₂O during cholecystectomy, and levator ani activity was recorded for the recumbent and vertical position during inflation and after deflation. The same procedure was also performed in patients undergoing open cholecystectomy without increasing the intra-abdominal pressure. The pelvic floor

activity increased significantly with the elevation of the intra-abdominal pressure and visceral weight, which resulted in increased muscle tone to oppose the augmented pressure or weight. The authors discuss that this effect seems to be mediated through the straining-levator reflex. A chronic increase of intra-abdominal pressure or visceral weight, e.g. in obese patients, might also affect muscle integrity and function.

Nerval Innervation

Nerval innervation of the pelvic floor musculature is not yet completely understood. The peripheral innervation is at least performed by 2 nerves: the levator ani nerve innervates the deeper part of the pelvic floor musculature and the pudendal nerve innervates the more superficial part of the musculature. In a cadaveric study on human fetuses, it was found that the levator ani nerve innervated the levator ani muscle in every pelvis, whereas a contribution of the pudendal nerve to the innervation of the levator ani muscle could be demonstrated in only 56% [7]. A communicating nerve branch between the pudendal and levator ani nerves was also observed in 56% [7].

The sensory neural innervation has been investigated even less. In a female squirrel monkey model, primary sensory neurons in the dorsal root ganglia and their central projections in the spinal cord were identified by injecting a cholera toxin B subunit unilaterally into the levator ani muscle to identify primary sensory neurons in the dorsal root ganglia and their central projections in the spinal cord [8]. It was shown that the levator ani muscle is innervated by 74% of the C-fiber neurons (putative nociceptive, mechanoreceptive, ergoreceptive and thermoreceptive neurons) and 26% of the A-fiber neurons (putative proprioceptive neurons) [8].

There are no sufficient functional data on the central nerval representation and its interaction of the pelvic floor.

Pelvic Floor Muscles and Ligaments

There are 4 main muscle groups which are of great importance for muscle action [9]. The pubococcygeus muscle inserts into the lateral part of the distal vagina and contracts forward against the pubourethral ligament and the perineal body. The levator plate complex inserts into the posterior wall of the rectum, and stretches the organs backwards, acting against the pubourethral ligament,

uterosacral ligament and the perineal body. Both muscles, the pubococcygeus muscle and the levator plate, are also described as the levator ani muscle complex.

The longitudinal muscle of the anus connects the levator plate, pubococcygeus muscle and puborectalis muscle to the external anal sphincter and creates a downward vector, which acts against the uterosacral ligaments. These muscles act during coughing and straining. The 4th muscle is the puborectalis muscle, which is part of the anorectal continence mechanism. The puborectalis muscle originates medially to the pubococcygeus muscle and is oriented vertically. It surrounds the posterior rectal wall and pulls the rectum anteriorly. It is also the muscle activated by squeezing. Squeezing elevates the whole levator plate, as well as the rectum, vagina and bladder, upwards and forwards.

These essential pelvic floor muscles result in 3 main muscle vector forces (fig. 1): (1) anterior muscle force in the direction of the symphysis, mediated by the anterior part of the pubococcygeus muscle and an additional anterior vector force mediated by the puborectalis muscle; (2) posterior muscle force in the direction of the sacrum, mediated by the levator plate complex; (3) craniocaudal muscle vector force, mediated by the longitudinal muscle of the anus.

The different actions of the pelvic floor are essentially based upon those 3-dimensional vector forces, mediated by the described muscles, their insertion ligaments and their neural peripheral and central nervous innervation. Clinically, it seems that significant parts of the problems are caused by ligamentous defects, rather than inherent muscle defects [10] or nervous lesions [11], although fecal incontinence appearing directly after childbirth, in most cases, appears to result from damage to the innervation of the pelvic floor muscles [12]. Damage to the innervation of the pelvic floor is often initiated by childbirth, but appears to progress over a period of many years, with the result that the functional disorder usually presents in midlife. Incontinence develops in some patients, but not in others [13]. The abnormalities found in electromyogram studies of the pelvic sphincter musculature and motor latency studies of its innervation do correlate with pelvic floor pathologies, but cannot solely explain the pathophysiology of the different disorders. From a therapeutic point of view, distorted pelvic floor anatomy and its potential for surgical repair is an important aspect. The pathophysiological concept of the Integral Theory introduced by Petros [14] states that distorted pelvic floor anatomy, organ prolapse and abnormal symptoms are mainly caused by laxity in the suspensory ligaments of

Fig. 1. Picture of the pelvic floor muscles. Pubococcygeus muscle (PCM), levator plate (LP), puborectalis muscle (PRM), longitudinal muscle of the anus (LMA) and the 3 main vector directions mediated by the corresponding muscles: anterior (PCM and PRM), posterior (LP), craniocaudal (LMA). EAS = External anal sphincter.

the vagina. The lax suspensory ligaments and fascias deteriorate muscle actions, thereby causing dysfunction. The pelvic organs, urethra, vagina and rectum have no inherent structure or strength which are created by the synergistic action of ligaments, fascia and muscles. With normal location of their insertion areas, the pelvic floor muscles can work normally. With dislocated insertion areas, muscle action is weakened.

Risk Factors for Ligamentous Defects

Proven risk factors for ligamentous defects are vaginal delivery, hysterectomy, chronic straining and normal ageing. These risk factors predispose some women to disruption, stretching or dysfunction of the connective tissue attachments of the vagina, resulting in ligamentous laxity, destruction and eventually prolapse of pelvic organs [15]. Abnormalities of connective tissue or connective tissue repair, however, can also significantly contribute to ligamentous defects. In this respect, the pelvic floor connective tissue structures have been subjected to struc-

Fig. 2. Mechanism of urinary continence. The pubococcygeus muscle (PCM) pulls anteriorly via the suburethral vaginal wall, the levator plate (LP) pulls posteriorly and the longitudinal muscle of the anus (LMA) pulls caudally. The bladder is rotated in a posterior-caudal vector direction and, thus, the proximal urethra is stretched and angulated in a plane around the pubourethral ligament (PUL). The proximal urethra, which is unattached, is stretched, narrowed and kinked.

tural analysis to some extent. In one study, the structural components of the uterosacral ligaments in postmenopausal women with and without pelvic organ prolapse were compared histomorphologically and immunohistochemically by quantifying their content of collagen I and III as well as smooth muscle [16]. It was found that in most of the samples (84%), the uterosacral ligaments were composed of more than 20% of smooth muscle cells and the collagen III expression was significantly ($p < 0.001$) related to the presence of pelvic organ prolapse rather than age or parity. The authors concluded that the higher collagen III expression might be a typical characteristic of pelvic organ prolapse in connective tissue, and the considerable amount of smooth muscle cells present in the uterosacral ligaments may be important for pelvic support. The same group further analyzed the expression of matrix metalloproteinases 1 and 2 in the uterosacral ligaments in women with and without pelvic organ prolapse [17]. Matrix metalloproteinases are enzymes which participate in the degradation of collagen and other extracellular matrix proteins. They found that matrix metalloproteinase-2 expression, which leads to degradation of type IV collagen, was significantly related to the presence of pelvic organ prolapse ($p = 0.004$) rather than age or parity.

Another group investigated structural and functional changes of the cardinal ligaments in patients with and without pelvic organ prolapse. They found that the cardinal ligaments of patients with pelvic organ prolapse were characterized by a higher expression of collagen III and tenascin, with lower quantities of elastin [18]. This can also be interpreted as a picture of the healing phase of traumatized tissue, which is evidenced by the raised tenascin expression. Further immunohistochemical studies on the α - and β -estrogen receptors, progesterone receptor and androgen receptor revealed that the prolapsed ligaments expressed more hormonal receptor-positive cells than the control ligaments, contributing a significant role to hormonal changes in pelvic organ prolapse [19].

A further group investigated the paraurethral connective tissue in women with stress urinary incontinence and controls [20]. They found a change in collagen metabolism resulting in an increased concentration of collagen and larger collagen fibrils in the stress urinary incontinent group. The authors concluded that these alterations might result in a more rigid form of the extracellular matrix, suggesting a connective tissue with impaired mechanical function.

Physiologic and Pathophysiologic Actions of the Pelvic Floor

A normal functioning pelvic floor musculature is critical for the physiological activities of the pelvic floor, which comprise urinary and fecal continence at rest and under stress, as well as micturition, defecation and suspension of the pelvic floor organs, bladder, uterus and rectum. In the following, only the influences on urinary continence and micturition will be described.

Urinary Continence

There are 3 distinct closure mechanisms for urinary continence: 2 involuntary and 1 voluntary. Both require a competent pubourethral ligament to function (fig. 2).

Bladder Neck Closure Mechanism. Under pressure, the bladder is pulled backwards and downwards by the levator plate and the longitudinal muscle of the anus, and the distal urethra is pulled forward by the pubococcygeus muscle. At rest, the slow twitch muscle fibers are active, under stress the fast twitch fibers [5]. Thus, the upper vagina and proximal urethra are stretched and angulated in a plane around the pubourethral ligament. The proximal urethra, which is unattached, is stretched, narrowed and

kinked. This can only be sufficiently performed if the vaginal wall below the bladder neck has sufficient elasticity. If excess scarring in this region is present after surgery, the vaginal wall is shortened and cannot be stretched sufficiently. The more powerful backward forces are tethered to the weaker forward forces. The urethra is forcibly pulled open upon pelvic floor contraction, such as getting out of bed in the morning, when the pelvic floor contracts to support all the intra-abdominal organs [9].

Urethral Closure Mechanism. If the vaginal wall beneath the rhabdosphincter at midurethra is loose, an increase of the diameter within the urethra and the rhabdosphincter is caused. According to the Hagen-Poiseuille's law, which describes flow through a stiff channel, the resistance to flow within a tube varies directly with the length of the tube and inversely with the radius (to the 4th power). Therefore, an increase of the urethra's radius also increases the flow through the urethra by the 4th power. On the other hand, according to the Laplace law, the pressure within the rhabdosphincter correlates inversely to the radius within the sphincter. If the radius of the urethra increases in the case of a loose vaginal wall, the pressure within the urethra decreases. Therefore, reduction of looseness decreases the urethral radius, which decreases the flow by the 4th power and enhances urethral pressure [5].

The Voluntary Closure Mechanism. During voluntary closure, such as squeezing, all organs and even the levator plate are actively pulled upwards and forwards [21]. Only voluntary contraction of the puborectalis muscle can explain these movements [9]. Squeezing represents mainly a voluntary control of the micturition reflex by stretching the vaginal membrane and, thus, better supporting the stretch receptors at the bladder base. This reduces the afferent impulses to the micturition center and controls detrusor instability at that stage [9].

Abnormal Bladder Emptying

If the levator plate and longitudinal muscle of the anus cannot contract sufficiently, which might be a result of a lax uterosacral ligament and perhaps a lax perineal body, the bladder must expel urine against urethral resistance, which is increased by the 4th power from the narrowing. In case of bladder decompensation, this might result in residual urine or urinary retention. The physiological funneling of the urethra at micturition can also be disturbed by the kinking of the urethra due to a ballooning cystocele, by a sling at the proximal urethra or bladder neck, or after elevation of the bladder neck by colposuspension.

Urgency and Frequency Symptoms

Urgency and frequency symptoms can also be caused by pelvic floor defects. The bladder base stretch receptors are supported by a vagina stretched tightly like a trampoline. This tightening is performed by the muscle forces mentioned above contracting against the suspensory ligaments. A lax vagina may result in the bladder stretch receptors firing off prematurely to cause urodynamic bladder instability [3]. Recently, new insights into the origin of bladder sensing have been gathered. So-called transient receptor potential channels belong to a large family of receptor structures, widely spread throughout the body, which are also found in the urinary tract [22]. These channels function as multifunctional sensors at the cellular level, activated by physical stimuli, such as mechanical stress, heat or cold, or chemical stimuli, such as pH or osmolality. Especially transient receptor potential vanilloid receptor 4, which has been shown to be present in rat bladders, appears to be shear stress sensitive and is also able to act as a flow sensor [22]. This could be of importance in the arousal of urgency in cystocele formation. Another possible role might be the detection of urine flow in the urethra, thereby activating the urethra-to-bladder reflex [22]. Thus, reconstruction of a lax vagina by specifically targeting the corresponding suspensory ligaments results in improved support of the bladder stretch receptors and might prevent activation of these multifunctional stress receptors.

Nocturia

Nocturia is a specific symptom of uterosacral ligament defects. In the recumbent position, the filling bladder is supported by the uterosacral ligaments alone, as pelvic muscles are relaxed at sleep. The intact uterosacral ligaments prevent the filling bladder from excessive posterior descent. In the case of defective ligaments, the filling bladder continues to descend until the stretch receptors are stimulated, activating the micturition reflex prematurely, which is perceived as urgency during sleep, e.g. nocturia [9].

Anatomical Representation of Defects

The pathophysiological aspects described above are able to classify the various defects into 3 anatomical zones [9]:

- 1 Anterior zone (extending from the meatus to the bladder neck)

- 2 Middle zone (extending from the bladder neck to the cervical ring or hysterectomy scar)
- 3 Posterior zone (extending from the cervix or hysterectomy scar down to the anus)

There are 3 key structures in each of the 3 zones that could require surgical repair if excess laxity is diagnosed correlating with the following symptoms [9]:

- 1 Anterior zone
 - a External urethral ligament
 - b Suburethral vagina
 - c Pubourethral ligament
- 2 Middle zone
 - a Arcus tendineus fasciae pelvis
 - b Pubocervical fascia
 - c Cervical ring attachment of pubocervical fascia
- 3 Posterior zone
 - a Cardinal ligaments
 - b Uterosacral ligaments
 - c Perineal body

Consequences for Pelvic Floor Reconstruction

These anatomical and functional aspects have to be considered in surgical pelvic floor reconstruction. In this regard, diagnosis of the anatomic localization of the ligamentous defects is extremely important. This diagnostic assessment should follow a structured path, developed by Petros [9]. This comprises history by a standardized questionnaire, a structured assessment of the pelvic floor, simulated operations to predict the efficacy of the reconstructive operations and, if necessary, additional studies such as perineal ultrasound, cystomanometry or functional MRI.

History

First, the different symptoms have to be identified by a standardized questionnaire. This should comprise questions covering the multiple symptoms that can be present in patients with pelvic floor defects. Interestingly, most of the questions represent a high likelihood of pointing to the specific anatomical defects, in particular the questions targeting the following aspects:

- 1 Symptoms of stress urinary incontinence
- 2 Symptoms of deficient bladder emptying
- 3 Urge urinary symptoms
- 4 Bowel symptoms, obstructed defecation and fecal incontinence

- 5 Symptoms of pelvic pain
- 6 Quality of life assessment
- 7 Questions about previous treatments, including operations

Structured Examination of the Pelvic Floor

After assessment of the existing pelvic floor symptoms, examining the whole pelvic floor from anterior to posterior is extremely important to be able to correlate symptoms with anatomical defects. In this regard, attention should be drawn to the following signs:

- 1 Anterior zone
 - a Laxity of the external urethral ligaments, visible through the insertion sites of the ligaments, lateral to the meatus and the shape of the meatus itself (meatal mucosal prolapse might be a sign of ligamentous laxity)
 - b Laxity of the suburethral vaginal wall (so-called hammock)
 - c Laxity of the pubourethral ligaments, which can only be assessed by a simulated operation, i.e. controlling urine loss when coughing by applying unilateral digital pressure lateral to the midurethra (e.g. insertion point of pubourethral ligaments)
- 2 Middle zone
 - a Laxity of arcus tendineus fasciae pelvis (flattened lateral vaginal sulcus) can also appear as a lateral cystocele (e.g. vaginal rugae present)
 - b Laxity of pubocervical fascia, which appears as a central cystocele (e.g. vaginal rugae have disappeared)
 - c Laxity of the pubocervical ring attachment, which appears as a high cystocele, possibly in conjunction with anterior enterocele
 - d Middle zone defects can be the cause of urgency symptoms, which can be assessed by simulated operations; forceps compress or elevate the cystocele at the exact zone of damage, and urge symptoms might disappear
 - e A cystocele can also mask stress urinary incontinence, which can be tested by a simulated operation (pushing the cystocele back into the vagina, and urine is lost upon coughing); wearing a pessary can improve emptying the bladder
- 3 Posterior zone
 - a Laxity of cardinal ligaments presents as an anterior enterocele or part of a uterine or vaginal vault prolapse. In the operation room, defects in the cardinal

Fig. 3. Defects in the cardinal ligaments can be assessed by grasping the vagina in position of the cardinal ligaments with forceps and approximating the forceps in the midline. In case of a cardinal ligament defect, the enterocele or prolapse will disappear.

Fig. 5. Assessment of loose perineal body. The index finger in the anal canal elevates the anterior rectal wall and pulls the perineal body caudally. The perineal body normally is at least 3 cm thick.

Fig. 4. Defects in the uterosacral ligaments can be assessed by grasping the vagina in position of the uterosacral ligaments with forceps and approximating the forceps in the midline. In case of a uterosacral ligament defect, the prolapse will disappear.

Fig. 6. Defects in the perineal body can be assessed by grasping the vagina laterally in position of the deep perineal muscles with forceps and approximating the forceps in the midline. In case of a perineal body defect, the rectocele will disappear.

ligaments can be assessed by grasping the vagina in position of the cardinal ligaments with forceps, which is approximately at the ventral insertion of the cervix, 1 cm above the hysterectomy scar, and approximating the forceps in the midline. In case of a cardinal ligament defect, the enterocele or prolapse will disappear (fig. 3)

- b Laxity of uterosacral ligaments presents as uterine prolapse, vaginal vault prolapse, dorsal enterocele or a rectocele. In the operation room, defects in the uterosacral ligaments, which are responsible for prolapse, can be assessed by grasping the vagina in position of the uterosacral ligaments with forceps, which is approximately at the dorsal insertion of the cervix, 1 cm below the hysterectomy scar, and approximating the forceps in the midline. In case

of a uterosacral ligament defect, the prolapse will disappear (fig. 4).

- c Laxity of the perineal body presents as a low rectocele, frequently as the cause of an obstructed defecation (fig. 5). As the longitudinal muscle of the anus inserts in the perineal body, the downward muscle vector force will disappear if the perineal body is loose, potentially resulting in abnormal bladder emptying. In the operation room, defects in the perineal body, responsible for the rectocele, can be assessed by grasping the vagina laterally in position of the deep perineal muscles with forceps and approximating the forceps in the midline. In case of a perineal body defect, the rectocele will disappear (fig. 6).

Discussion

In pelvic floor surgery, the following fundamental principles should be followed:

- 1 Restoration of form (structure) leads to restoration of function, meaning that after an anatomical exact restoration, the insertion areas of the pelvic floor muscles are reconstructed well and the muscles can act optimally; therefore, the function – even if complex – should have the optimal chance to recover.
- 2 Conventional techniques, such as suturing alone in weak tissues, have recurrence rates which are too high; therefore, the use of alloplastic materials should be an option.
- 3 The axis of the posterior vagina is nearly horizontal because the uterosacral ligaments insert dorsally between S2 and S4. If a sacrocolpopexy is performed, the elevation of the vagina to the promontorium is too high, in the case of a sacrospinal fixation on the other hand, the fixation of the vagina to the sacrospinous ligament will be too caudal. Therefore, approximation of the uterosacral ligaments in the midline is an optimal technique.
- 4 The uterus needs to be conserved whenever possible. It is the central anchoring point for the posterior ligaments (cardinal ligament, uterosacral ligament), the rectovaginal fascia and the pubocervical fascia. The descending branch of the uterine artery is a major blood supply for these structures and should be conserved where possible, even if a subtotal hysterectomy is performed.
- 5 As the tissue structures often are displaced laterally, surgical techniques should be used which bring the tissues together in the midline or bridge it with alloplastic tapes at the anatomically correct place.
- 6 It has to be considered that alloplastic meshes have the tendency to shrink and reduce the elasticity of tissues. Therefore, the use of alloplastic materials has to be reduced to the necessary amount. However, as discussed, the conventional techniques also have severe drawbacks because of the insufficient support of the defective tissues.
- 7 To minimize pain, surgery to the perineal skin and tension when suturing the vagina should be avoided. Vaginal excision should be avoided even in patients with a large bulging prolapse. After repair of the underlying ligamentous or fascial defects, the vaginal wall contracts and will be more elastic than after excision.
- 8 To avoid urinary retention, tightness or elevation of the bladder neck area of the vagina should be avoided,

as well as gross indentation of the urethra with a mid-urethral sling.

- 9 Looking at the bladder neck closure mechanism, the midurethral tape should be positioned along the pubourethral ligament, which inserts retropubically. This seems especially important in patients with severe stress urinary incontinence or recurrences. The transobturatoric approach for tape insertion may be an option for mild and moderate cases, but does not meet the exact anatomical reconstruction of the pubourethral ligaments. New techniques using mini-anchored nonstretchable tapes, such as the Tissue Fixation System [9], accurately reinforce the main suspensory ligaments (pubourethral, uterosacral, cardinal, arcus tendineus fascia pelvis and perineal body) while bringing the laterally displaced tissues towards the midline. This action more precisely restores the musculoelastic tension required to also restore function. The meshes with sling fixation transobturatorically or at the sacrospinous ligaments only produce long lasting barriers. Pelvic floor surgery is currently developing fundamentally into a minimal invasive surgery. It should be the aim to restore the defects in a way which optimizes the pelvic floor muscles and the functions.

Conclusion

The pelvic floor is a complex organ and behaves like an orchestra: the brain is the conductor, the ligaments are the strings, the tubes (urethra, vagina and anal canal) are the wind instruments, the reservoirs (bladder, uterus and rectum) are the timpani and the pelvic floor muscles are the chorus singers. The orchestra might play by itself for a while, but essentially needs the conductor for fine-tuning. If a string on the cello is broken, the orchestra is not completely distorted, but some compositions do not sound right. If later a valve of a horn breaks, further music pieces will not be able to be performed.

This allegory should explain the interactions of the different structures discussed and why some defects occasionally show different symptoms and why symptoms are sometimes due to different defects.

Recent advances in the holistic understanding of the pelvic floor have dramatically changed the options and possibilities in pelvic floor reconstruction. Currently, a great percentage of patients with pelvic floor defects experiencing symptoms can be offered treatment, leading to a favorable outcome.

References

- 1 Lawrence JM, Lukacz ES, Nager CW, Hsu JW, Luber KM: Prevalence and co-occurrence of pelvic floor disorders in community-dwelling women. *Obstet Gynecol* 2008; 111:678–685.
- 2 Petros PE, Ulmsten U: Tests for 'detrusor instability' in women: these mainly measure the urethral resistance created by pelvic floor contraction acting against a premature activation of the micturition reflex. *Acta Obstet Gynecol Scand* 1993;72:661–667.
- 3 Petros PE, Ulmsten U: Bladder instability in women: a premature activation of the micturition reflex. *Neurourol Urodyn* 1993;12: 235–239.
- 4 Petros PE, Ulmsten U: Urethral pressure increase on effort originates from within the urethra, and continence from musclovaginal closure. *Neurourol Urodyn* 1995;14:337–346; discussion 346–350.
- 5 Petros PE, Ulmsten UI: An integral theory and its method for the diagnosis and management of female urinary incontinence. *Scand J Urol Nephrol Suppl* 1993;153:1–93.
- 6 Shafik A, Doss S, Asaad S: Etiology of the resting myoelectric activity of the levator ani muscle: physioanatomic study with a new theory. *World J Surg* 2003;27:309–314.
- 7 Wallner C, van Wissen J, Maas CP, Dabhoiwala NF, DeRuiter MC, Lamers WH: The contribution of the levator ani nerve and the pudendal nerve to the innervation of the levator ani muscles: a study in human fetuses. *Eur Urol* 2008;54:1136–1142.
- 8 Pierce LM, Rankin MR, Foster RT, Dolber PC, Coates KW, Kuehl TJ, Thor KB: Distribution and immunohistochemical characterization of primary afferent neurons innervating the levator ani muscle of the female squirrel monkey. *Am J Obstet Gynecol* 2006;195:987–996.
- 9 Petros PE: The Female Pelvic Floor. Function, Dysfunction and Management according to the Integral Theory, ed 2. Heidelberg, Springer, 2007.
- 10 Kramer LA, Gendron JM, Pierce LM, Runge VM, Shull BL, Kuehl TJ: Magnetic resonance imaging of the levator ani in the squirrel monkey: a comparison of muscle volume between a cohort with pelvic organ prolapse and matched normals. *Am J Obstet Gynecol* 2006;194:1467–1471.
- 11 Neill ME, Parks AG, Swash M: Physiological studies of the anal sphincter musculature in fecal incontinence and rectal prolapse. *Br J Surg* 1981;68:531–536.
- 12 Snooks SJ, Setchell M, Swash M, Henry MM: Injury to innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 1984;2: 546–550.
- 13 Swash M, Snooks SJ, Henry MM: Unifying concept of pelvic floor disorders and incontinence. *J R Soc Med* 1985;78:906–911.
- 14 Petros PE, Ulmsten UI: An integral theory of female urinary incontinence: experimental and clinical considerations. *Acta Obstet Gynecol Scand Suppl* 1990;153:7–31.
- 15 Jelovsek JE, Maher C, Barber MD: Pelvic organ prolapse. *Lancet* 2007;369:1027–1038.
- 16 Gabriel B, Denschlag D, Gobel H, Fittkow C, Werner M, Gitsch G, Watermann D: Uterosacral ligament in postmenopausal women with or without pelvic organ prolapse. *Int Urogynecol J Pelvic Floor Dysfunct* 2005;16: 475–479.
- 17 Gabriel B, Watermann D, Hancke K, Gitsch G, Werner M, Tempfer C, zur Hausen A: Increased expression of matrix metalloproteinase 2 in uterosacral ligaments is associated with pelvic organ prolapse. *Int Urogynecol J Pelvic Floor Dysfunct* 2006;17: 478–482.
- 18 Ewies AA, Al-Azzawi F, Thompson J: Changes in extracellular matrix proteins in the cardinal ligaments of post-menopausal women with or without prolapse: a computerized immunohistomorphometric analysis. *Hum Reprod* 2003;18:2189–2195.
- 19 Ewies AA, Thompson J, Al-Azzawi F: Changes in gonadal steroid receptors in the cardinal ligaments of prolapsed uteri: immunohistomorphometric data. *Hum Reprod* 2004; 19:1622–1628.
- 20 Falconer C, Blomgren B, Johansson O, Ulmsten U, Malmstrom A, Westergren-Thorsson G, Ekman-Ordeberg G: Different organization of collagen fibrils in stress-incontinent women of fertile age. *Acta Obstet Gynecol Scand* 1998;77:87–94.
- 21 Kegel AH: Progressive resistance exercise in the functional restoration of the perineal muscles. *Am J Obstet Gynecol* 1948;56:238–248.
- 22 Everaerts W, Gevaert T, Nilius B, De Ridder D: On the origin of bladder sensing: Tr(i)ps in urology. *Neurourol Urodyn* 2008;27:264–273.