**Book review**

*As announced in the Editorial by Bruce Farnsworth (Pelviperineology 2011; 30:5) this is the first of a series of articles highlighting the different sections of the book "Pelvic Floor Disorders, Imaging and a Multidisciplinary Approach to Management" edited by G.A. Santoro, P. Wieczorek, C. Bartram, Springer Ed, 2010.*

# Pelvic floor anatomy

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The *first section* of the book "Pelvic floor disorders - Imaging and Multidisciplinary Approach to Management" is entitled "Pelvic Floor Anatomy" and consists of five chapters describing the detailed anatomy of female pelvic floor, its physiology and patophysiology as well as neural control.

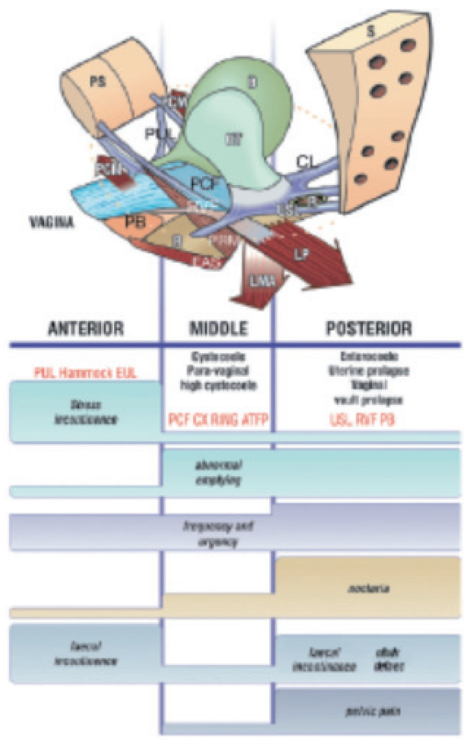
In the first chapter "*State of the Art: Pelvic Floor Anatomy*" J. O. L DeLancey and S. A. Shobeiri describe the functional anatomy of the pelvic floor in women especially highlighting supportive function of muscles and fasciae for pelvic organs and changes in their structures with the presence of prolapse. The female pelvis is naturally divided into anterior, posterior and lateral compartments, and its bottom is formed by levator ani muscles. Pelvic organs support system is multifaceted including the perineal membrane, the endopelvic fascia, and the levator ani muscles. The support of uterus and vagina differs in various regions: the upper-third of the vagina and the cervix (1st level) has relatively long suspensory fibers, attached vertically; mid-portion of the vagina (2nd level) has more direct attachment laterally to the pelvic wall and the most caudal portion of the vagina (3rd level) is attached directly to the surrounding structures and the elevator ani muscle and the perineal membrane are important supportive structures in this level. Damage in level 1 results in uterine or vaginal prolapse of the apical segment, while damage in levels 2 and 3 cause anterior and posterior vaginal wall prolapse. Various combinations of these defects are responsible for diversity of clinical problems.

The authors describe very precisely the anatomy and morphology of each of these supportive structures correlating their function with histological and patophysiological studies. Separate subdivision of this chapter provides information about levator ani muscles anatomy. There are three major components: iliococcygeal muscle, pubovisceral muscle and puborectalis muscle. The anatomical characteristics, origins and insertions of these muscles are also precisely described. The opening between levator ani muscles through which pelvic organs pass, forms the levator hiatus and its part ventral to the perineal body is called urogenital hiatus. The levator ani tonic activity keeps the hiatus closed by compressing the urethra, vagina and rectum to the pubic bone and pulling the pelvic floor upwards. This constant action eliminates any opening within the pelvic floor preventing prolapse to occur. Damage to the levator ani muscles caused by nerves or connective tissue damage will result in prolapse. Authors highlight the role of endopelvic fascia, which in the case of the levator ani damage, is the last supporting structure of pelvic organs preventing the prolapse to develop for short periods of time. The interaction between pelvic floor muscles and endopelvic fascia maintains the "flap valve" configuration in the pelvic floor lessening ligaments tension.

The second chapter "*The Integral Theory: A Musculo-elastic Theory of Pelvic Floor Function and Dysfunction*" by Peter Papa Petros and Michael Swash, provides information about accurate diagnosis and surgical treatment for pelvic floor symptoms, such as: urinary and fecal incontinence, pelvic pain and defecation difficulties, basing on the Integral Theory. The authors explain essential concepts of this theory. The essential statement is that pelvic organs are suspended by four suspensory ligaments and are controlled by neurologically coordinated muscle forces contracting against these ligaments. Any laxity of these suspensory ligaments might hamper muscle forces resulting in organ prolapse or dysfunction in organ closure or opening causing incontinence or evacuation symptoms, respectively. Thus, to perform a successful reconstructive surgery, knowledge about which ligament is affected is required (Figure 1).

According to integral theory, an accurate diagnosis of ligaments laxity could be made with the use of symptom-based algorithm and three zones (anterior, middle, posterior compartments) examination system. Some symptoms are zone specific, however some of them may occur due to the damage in at least two compartments (e.g. fecal incontinence in the anterior and posterior zones, urge incontinence in all three zones). In such cases, coexisting symptoms and vaginal digital examination should be involved to diagnose and confirm the site of damage. Once it is done, proper surgery, which aim is to reconstruct affected ligaments with the use of propylene tapes, could be performed.

The third chapter entitled "*Pathophysiology of the Pelvic Floor: Basic Physiology, Effects of aging, and Menopausal Changes*" written by Dee E. Fenner and Yvonne Hsu describes etiologic risk factors and associations contributing to pelvic floor disorders such as vaginal parity, ageing, hormonal status, pelvic surgery, collagen diseases and depression. Many of these relationships are poorly understood, moreover, various pelvic floor disorders may be explained by different etiological risk factors or pathophysiological mechanisms. For example, vaginal parity predisposes to developing pelvic organ prolapse, while urinary incontinence has got many other risk factors with equal or greater influence including obesity and ageing. Differences found in women with prolapse suggest that biochemical changes in the connective tissue may play and important role in prolapse, however these studies are unable to explain the sequence of prolapse progression. Thus, it is not defined whether alterations in connective tissue lead to prolapse or are a response to the mechanical effects of prolapse. Electromyographic (EMG) studies of women with pelvic floor disfuctions, including prolapse and incontinence, found changes consistent with motor unit loss or failure of central activation. It has been confirmed that vaginal delivery con-




**Pelvic Floor Laxities which can be repaired**

Pubourethral ligament (*PUL*); hammock; external urethral ligament (*EUL*)

pubocervical fascia (*PCF*); arcus tendineus fascia pelvis (*ATFP*); cardinal ligament/cervical ring (*CL*)

uterosacral ligament (*USL*); rectovaginal fascia (*RVF*); perineal body (*PB*)

**Fig. 2.8** The pictorial diagnostic algorithm schematically indicates the sites of connective tissue defects (laxities of ligaments or fascias), which can be repaired by surgery, and the association of each zone with symptoms of pelvic floor dysfunction. The area of the symptom rectangle indicates the estimated frequency of symptom causation in each zone. Anterior zone: external meatus to bladder neck; middle zone: bladder neck to anterior cervical ring; posterior zone: posterior vaginal wall from the cervix to the perineal body. *B*, bladder; *EAS*, external anal sphincter; *LMA*, longitudinal muscle of the anus; *LP*, levator plate; *PCM*, pubococcygeus muscle; *PS*, pubic symphysis; *R*, rectum; S, sacrum; *UT*, uterus

Figure 1. – The Integral Theory: A Musculo-elastic Theory of Pelvic Floor function and dysfunction.

fers a four-to eleven-fold higher risk of prolapse that increases with parity. The structural components that include the pelvic floor musculature, connective tissue condensations and fibromuscular walls of the pelvic viscous work together to provide pelvic organ support. When components of pelvic support and their relations to each other have been identified, we will be able to understand how disruptions result in failure and the resulting sypmtoms. Investigations into the biochemical processess are lacking. It has been proven however, that hormonal replacement therapy (HRT) clinically does not improve urinary incontinence and may in fact promote symptoms and increase severity of incontinence. In contrast, HRT may protect from developing POP. Much work is needed at the molecular, cellular, and clinical levels to understand the mechanisms and associations between HRT, ageing, and the impact of vaginal birth on pelvic floor disfunctions.

The fourth chapter "*The Pelvic Floor: Functional Concepts and Neurocontrol*" by Michael Swash includes the rewiev of the development of current ideas of pelvic floor function. The importance of understanding motor control mechanisms of pelvic floor responsiveness in health and disease is an underlying theme of the response to all forms of treatment in pelvic floor disorders. This is an adaptive response based on the capacity of the system to respond when there is significant functional damage. The author underlines that the pelvic floor should be concidered as a holistic functional entity, rather than three separate anterior, middle, and posterior perineal systems or structures. Continence and evacuation of feces and urine are under the control of the will, and therefore of the brain. The necessity to initiate evacuation, however, is almost always triggered by sensory input from the bladder or lower colon and rectum, indicating a need to switch from storage to evacuation. When the pelvic floor is dysfunctional, whether beacuse of stretch injury to ligaments, or beacuse of denervation-induced weakness to the pelvic floor musculature, or as a result of direct injury during childbirth, the central nervous system (CNS) is capable to adapt the action of the weakened muscular system to maintain function, even when the damage to the effector is quite severe.

Biofeedback has been propounded as a treatment for fecal incontinence. The technique utilizes several methods involving feedback from EMG signals of pelvic floor muscle activity in order to "teach" the subject how better activate and control these muscles. In mild dysfunctional states, it can be quite effective.

Much has been learned in recent years, however a full understatnding of the effects of weakness in specific muscular systems, and of the effects of loss of elastic resistance in pelvic floor ligaments, is not yet accomplished. The compensatory capacity of the CNS to modulate pelvic floor function is also not yet well understood. Inly a full knwoledge of this capacity will enable appropriate and testable procedures to be develloped. Much progress has been made, but there is much more to be learned.

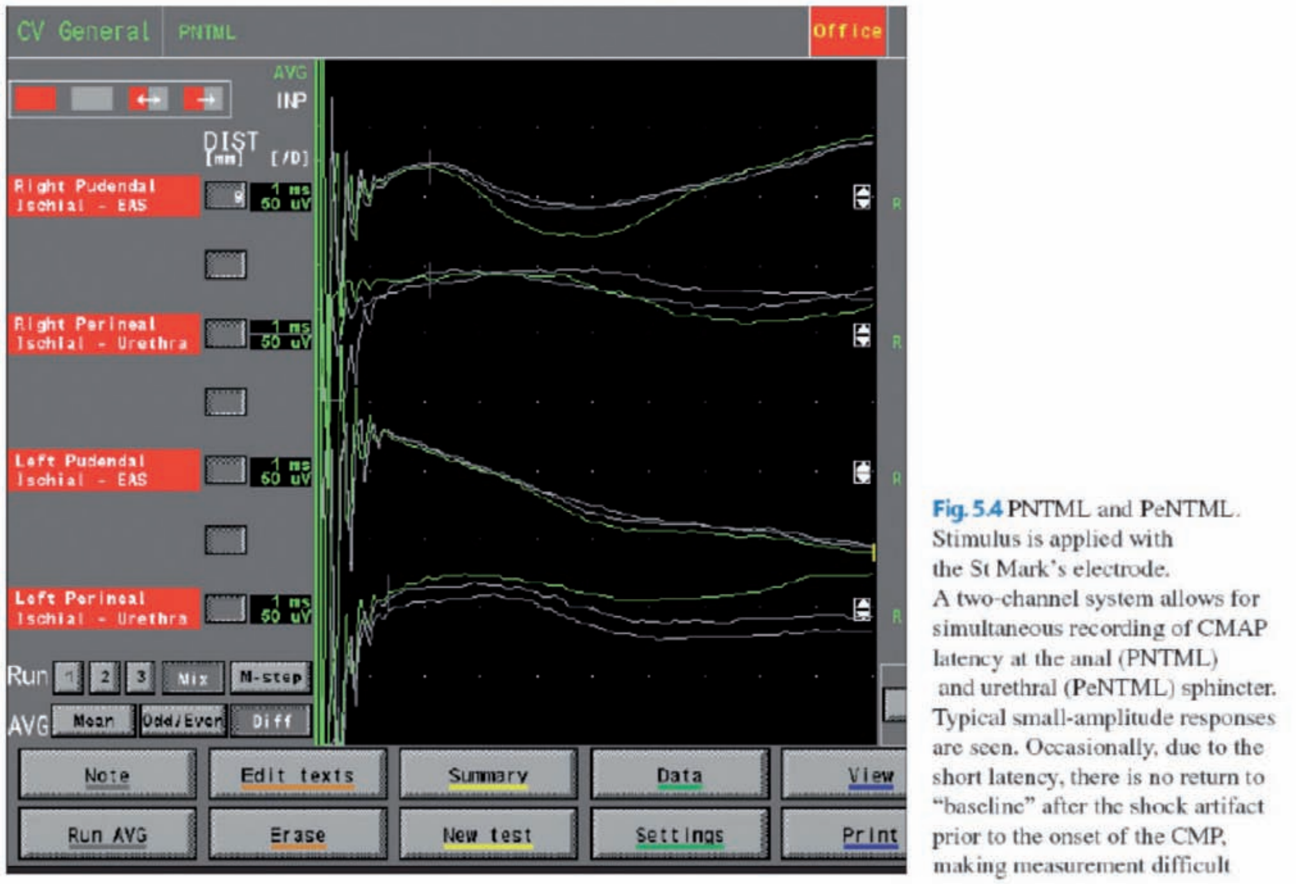



Figure 2. – Typical waveform from pudendal (PNTML) and perineal (PeNTML) nerve conduction studies with simultaneous recording of CMAP (compound muscle action potentials) latency at the anal and urethral sphincters.

The fifth chapter entitled "*Clinical Neurophysiology of the Pelvic Floor*" by W. Thomas Gregory and Kimberly Kenton focuses on electrodiagnostic testing of the pelvic floor, which is becoming increasingly common in clinical pelvic medicine and pelvic floor research. Clinically, it can be used with history, physical examination, and urodynamic testing to aid in the disgnosis of certain pelvic floor disorders and to determine if a central or peripheral neurologic problems exist. Electrodiagnostic testing is also emerging in studies investigating the etiology of pelvic floor disorders. A basic understaning of the principles and techiques used in electrodiagnostic medicine is essential for reconstructive pelvic surgeons. However, most pelvic surgeons will never have the skills or expertise to perform pelvic floor neurophysiologic testing. According to authors a multidisciplinary teams including urogynecologic, urologic and colorectal surgeons, phychatrist, neurologist, and physical therapists are imperative if we are going to improve our understanding of pelvic floor disorders and improve treatment outcomes.

Much of our current understanding of the etiology of pelvic floor disorders has come from both nerve conduction studies and EMG of the pelvic floor muscles in women with stress incontinence, fecal incontinence or pelvic organ prolapse. We understand that surgery can affect pelvic innervation, the relationship between vaginal childbirth and pudental neuropathy has been also confirmed. The degree of denervation and pelvic floor injury can be measured. Such measurements have some correlation with clinical outcomes, but further research refining techniques and estabilishing normative electrodiagnostic parameters for the urethral sphincter, anal sphincter, and levator ani are imperative. Pelvic floor electrodiagnostic studies may aid in the clinical diagnosis of some pelvic floor disorders and help to predict outcomes of incontinence surgery. However, further confirmatory studies are necessary.

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