

W26: The Overactive Pelvic Floor

Workshop Chair: Anna Padoa, Israel

08 October 2015 11:00 - 12:30

Start	End	Topic	Speakers
11:00	11:10	Definitions and pathophysiology of OPF.	Marc Beer-Gabel
11:10	11:30	Bladder pain syndromes, chronic pelvic pain, voiding dysfunction and OPF.	Mauro Cervigni
11:30	11:50	Sexual dysfunction and sexual pain disorders. Sexual abuse, other psychosocial factors and OPF.	Anna Padoa
11:50	12:10	Gastrointestinal co-morbidities and OPF.	Marc Beer-Gabel
12:10	12:30	Evaluating and understanding pelvic floor muscle overactivity - A physiotherapist perspective	Melanie Morin

Aims of course/workshop

This workshop will review the definition, etiology and pathophysiology of non-relaxing pelvic floor muscle tone, as well as discuss sexual function and past sexual experience in relation to the pelvic floor. Specific pelvic floor dysfunctions associated with pelvic floor over-activity will be reviewed. Special attention will be devoted to female genital pain, bladder pain and interstitial cystitis, sexual dysfunction related to pelvic pain, musculoskeletal aspects of pelvic floor over-activity, LUTS and voiding dysfunction, and anorectal disorders. Assessment of the pelvic floor will be addressed. Medical, psychosocial, and physical therapy treatment interventions will be discussed, with an emphasis on interdisciplinary management.

Learning Objectives

1. Identify specific pelvic floor dysfunctions associated with pelvic floor over-activity
2. Carry out initial assessment of pelvic floor overactivity patients and refer to appropriate specialists for further evaluation
3. Establish an initial treatment plan and referring pelvic floor overactivity patients to relevant therapy modalities

Definitions and Pathophysiology of Overactive Pelvic Floor

Marc Beer-Gabel, MD

Adapted from Chapter 1 in “The Overactive Pelvic Floor”; editors: Anna Padoa and Talli Rosenbaum, Springer Editions, in press: “Definition and Basic Etiology of the Overactive Pelvic Floor”. Author: Stephanie Thibault-Gagnon

Definition of “overactive pelvic floor muscles”

2005 report from the Pelvic Floor Clinical Assessment Group of the ICS:

“A condition in which the pelvic floor muscles do not relax, or may even contract when relaxation is functionally needed, for example during micturition or defecation”.

Pelvic Floor Muscle Function

The pelvic floor muscles work as a functional unit and normally contract simultaneously as a mass contraction.

Pelvic floor muscle types:

- 70% type I (slow twitch) – postural and supportive role
- 30% type II (fast twitch) - necessary for closure of these pelvic openings

Pelvic Floor Muscle Anatomy

- Superficial layer: superficial transverse perineal, bulbospongiosus, and ischioavernosus muscles.
- Intermediate layer: perineal membrane, or the urogenital diaphragm.
- Deep muscular layer: pelvic diaphragm, composed of the ischiococcygeus muscle and the levator ani muscle group.
- The endopelvic fascia provides muscle anchorage to the bony pelvis, thus proper functioning of muscles is highly dependent on the integrity ligamentous and fascial components.

Innervation of pelvic floor muscles

The somatic efferent (motor) nerve fibers:

- 2nd to 4th sacral nerve (S2-S4). Separate branches from the sacral plexus supply the levator ani (S3-S4) and coccygeus (S3-S4) directly, pudendal nerve (S2-S4).

The autonomic nervous system efferent (visceromotor) innervations (sympathetic and parasympathetic) to the pelvic floor:

- Sympathetic innervation: thoracolumbar region (T10-L2).
- Parasympathetic innervation: sacral spinal segments (S2-S4).

'Tonic' and 'phasic' pelvic floor muscle activity

- The striated urethral and anal sphincters and the levator ani (although not at all sites) demonstrate constant baseline activity, commonly referred to as 'tonic' activity

- The pelvic floor muscles also exhibit 'phasic' activity with stronger activation due to voluntary contraction or reflex activation, for example, in response to postural disturbances or increases in intra-abdominal pressure

Pathophysiology

- OPF implies a physical state of heightened activity within the pelvic floor muscles
- Co-morbid conditions involving different body systems can be present with symptomatic manifestations involving urinary, anorectal and/or sexual dysfunction, genital/pelvic pain and psycho-emotional distress
- Individuals with OPF: commonly found to present with pelvic floor muscle hypertonicity from other sources, most notably myofascial trigger points.
- Recognizing the various potential sources of pelvic floor muscle hypertonicity is particularly important for identifying the specific pelvic floor impairments affecting individuals with OPF, and designing tailored treatment interventions
- Important concept: **'Unnecessary' muscle tension**. a type of muscular activity that is unintentional and is the source of what is often referred to clinically as 'muscle tension'. This is amenable to voluntary control with training (e.g., through biofeedback assistance) and may arise from psychological distress or anxiety, overload from sustained contraction or repetitive activity, and/or inefficient use of muscles.

Etiologies of OPF

Chronic pelvic pain

OPF has been found to be a physical hallmark of several different conditions involving CPP, an “idiopathic pain disorder” (IPD) which has two primary pathways of vulnerability, both mediated by genetic and environmental/social factors, that underlie its development:

- Pain amplification
- Psychological distress.

Noxious stimuli occurring over a prolonged period of time cause up-regulation of nociceptive system function, which leads to dysregulations in both the peripheral and central mechanisms of sensory and pain processing.

Mechanisms responsible for the up-regulation of nociceptive nervous system components:

1. Peripheral sensitization:

- Sensitization of peripheral nociceptors
- Activation of ‘silent’ nociceptive afferents
- Conversion of myelinated afferents, such that they adopt the properties of nociceptive fibers

2. Central sensitization:

- Up-regulation (sensitization) of the dorsal horn of the spinal cord. Consequences of nociceptive system up-regulation:
 - ‘hyperalgesia’
 - ‘allodynia’
- Neuropathic reflexes:

- “neurogenic inflammation”.
- “viscero-visceral hyperalgesia”.

Psychological distress

- Psychological distress is thought to play a role in triggering and/or perpetuating CPP, and consequently OPF.
 - Fear-avoidance model (FAM) of chronic pain: a conceptual model that explains how negative pain-related cognitions and maladaptive behavioural responses contribute to the development and maintenance of chronic pain. The fear of pain, combined with pain-related anxiety and hypervigilance to pain, leads to defensive behaviours, notably muscular reactivity/contraction, in the presence of a painful stimulus or in the anticipation of pain.

Psychosocial and psychosexual disturbances

- A history of traumatic experience may lead to OPF, especially if the events occur repeatedly or if the person ‘re-lives’ the experiences through, for example, flashbacks or nightmares.
- The pelvic floor muscles are reactive to threatening images alone. These muscles demonstrate reactivity to experienced sexual trauma or abuse.

Abnormal behavior/pattern of pelvic floor muscle use

- Dysfunctional voiding and/or defecation can result from improper learning of these control mechanisms.

Direct trauma or pathology

- Both obstetric injury and pelvic surgical procedures, have been reported to result in painful and hypertonic pelvic floor muscles.
- This may be the consequence of inflammation and pain resulting from the trauma.
- Resulting anatomical disruptions within the pelvic floor, including tissue adhesions and imbalances between the right and left pelvic floor may lead to OPF.

Postural abnormalities

Any ongoing postural abnormality in the region of the spine, pelvis and/or lower extremities can contribute to the development of pelvic pain and/or OPF.

REFERENCES

- Messelink B, Benson T, Berghmans B, Bo K, Corcos J, Fowler C, et al. Standardization of terminology of pelvic floor muscle function and dysfunction: Report from the pelvic floor clinical assessment group of the international continence society. *Neurourol Urodyn.* 2005;24(4):374-80.
- Simons DG, Mense S. Understanding and measurement of muscle tone as related to clinical muscle pain. *Pain.* 1998;75(1):1-17.
- Chantraine A. Examination of the anal and urethral sphincters. . In: Desmedt JE, editor. *New developments in electromyography and clinical neurophysiology.* Basel: Karger; 1973. p. 421-32.

- Wright A. Neurophysiology of pain and pain modulation. In: Strong J, Unruh AM, Wright A, Baxter GD, editors. Pain: a textbook for therapists. London: Churchill Livingstone; 2002. p. 43-64.
- Alappattu MJ, Bishop MD. Psychological factors in chronic pelvic pain in women: relevance and application of the fear-avoidance model of pain. *Phys Ther.* 2011;91(10):1542-50.
- Ramakers MJ, van Lunsen RHW. Psychosocial influences. In: Carrière B, Feldt CM, editors. The pelvic floor. Stuttgart: Georg Thieme Verlag; 2006. p. 117-28.
- van der Velde J, Laan E, Everaerd W. Vaginismus, a component of a general defensive reaction. An investigation of pelvic floor muscle activity during exposure to emotion-inducing film excerpts in women with and without vaginismus. *Int Urogynecol J Pelvic Floor Dysfunct.* 2001;12(5):328-31.

Bladder Pain Syndromes, Chronic pelvic pain, voiding dysfunction and Overactive Pelvic Floor

Mauro Cervigni, MD

Adapted from Chapter 5 (Female Interstitial Cystitis/Bladder Pain Syndrome; authors: Mauro Cervigni, Andrea Morciano, Giuseppe Campagna) and Chapter 8 (Voiding Dysfunction; author: Asnat Groutz) in “The Overactive Pelvic Floor”; editors: Anna Padoa and Talli Rosenbaum, Springer Editions, in press.

Bladder Pain Syndrome/Interstitial Cystitis (BPS/IC)

Definition

The National Institute of Diabetes and Digestive Kidney Diseases (NIDDK) established a set of consensus criteria, which were developed to ensure the comparability of patients enrolled in clinical studies. These included:

- Hunner's ulcers
- any two of the following:
 - pain on bladder filling, relieved by emptying
 - suprapubic, pelvic, urethral, vaginal, or perineal pain for 9 months
 - glomerulations on endoscopy or upon hydrodistension under spinal or general anesthesia.

Epidemiology

680 per 100,000 (0.68%) for a probable BPS/IC diagnosis and 300 per 100,000 (0.3%) for a definite one.

Pathophysiology

- Mastocytosis
- Dysfunctional Bladder Epithelium
- Neurogenic Inflammation
- Reduced Vascularization
- Autoimmunity

IC/BPS and OPF

- Inflammation, pain or of pelvic visceral trauma may transfer noxious stimuli to the sacral cord, which can result in pelvic floor muscle dysfunction due to sacral nerve hypersensitivity and initiate a sacral cord wind-up effect.

- The “Guarding Reflex” is a visceromotoric reflex activated with the aim to increase the tone of the pelvic floor during routine daytime activity. The afferent autonomic bombardment occurring in BPS/IC patients may enhance and maintain such guarding reflex, resulting in pelvic floor overactivity.

Diagnosis

Disease type

Bladder diseases

Confusable diseases

Overactive bladder

Neurogenic bladder

Radiation cystitis

Bladder calculus

Bladder cancer

Prostate and urethral diseases

Benign prostatic hypertrophy

Prostate cancer

Urethral stenosis

Urethral diverticulum

Genitourinary infections

Bacterial cystitis

Urethritis

Prostatitis

Gynecologic diseases

Endometriosis

Uterine myoma

Vaginitis

Other conditions

Postmenopausal syndrome

Polyuria

Overactive pelvic floor

Symptoms and signs

Pain symptoms are often vague and poorly localized.

Pain is typically exacerbated by pelvic floor muscle activities like sexual intercourse or voiding.

The pressure arising from pelvic floor overactivity may be perceived as a need to void.

A lubricated cotton tip applicator is then gently used to evaluate for signs of allodynia and vulvodynia.

Active trigger points are often identified as exquisitely tender areas palpable as a small 3-6 mm nodule

Ancillary testing

- Electromyography
- Urodynamic testing
- Defecography

Treatment

- Behavioural modifications:
 - bladder training, diary-timed voiding
 - dietary changes
- Physical Therapy:

- Biofeedback and soft tissue massage
- Manual physical therapy to pelvic floor myofascial trigger points
- Modified Thiele intravaginal massage

Oral medications for treatment of BPS/IC

<i>Drug</i>	<i>RCT</i>	<i>Success (%)</i>
Amitriptyline; tricyclic antidepressant	Yes	42
Antibiotics	Yes	48
Cimetidine	Yes	65
Hydrocortisone	No	80
Ciclosporin	No	90
Hydroxyzine	Yes	31
l-Arginine	Yes	not effective
Nifedipine	No	87
Quercetin	No	92
Sodium pentosanpolysulfate	Yes	33
Suplatast tosilate	No	86

Intravesical medications for treatment of BPS/IC: results

<i>Drug</i>	<i>RCT</i>	<i>Success (%)</i>
-------------	------------	--------------------

DMSO	Yes	70
BCG	Yes	Conflicting data
Resiniferatoxin	Yes	No proven efficacy
Hyaluronic acid	Yes	No proven efficacy
Heparin	No	60
Chondroitin Sulfate	No	33
Lidocaine	No	65
PPS	Yes	Possible efficacy

Adapted from P. Hanno

Procedural interventions:

- Trigger point injections
- Botulinum Toxin
- Sacral nerve neuromodulation
- Laser resection, augmentation cystoplasty, cystolysis, cystectomy, and urinary diversion may be the ultimate option for refractory BPS/IC patients

REFERENCES

- Hanno PM, Landis JR, Matthews-Cook Y et al. The diagnosis of interstitial cystitis revisited: lessons learned from the National Institutes of Health Interstitial Cystitis Database Study. *J Urol* 1999;161:553–557.
- Leppilahti M, Sairanen J, Tammela TL et al. Prevalence of clinically confirmed interstitial cystitis in women: a population based study in Finland. *J Urol* 2005;174:581–583
- Butrick CW: Pelvic floor Hypertonic disorders: identification and amangement. *Obstet Gynecol Clin North Am* 2009; 36: 707-722
- Hanno P, Baranowski A, Fall M et al. Painful bladder syndrome (including interstitial cystitis). In: Abrams P, Cardozo L, Khoury S, Wein A (eds) *Incontinence*. Health Publication Ltd, Plymouth, 2005, pp 1457–1520.
- Sand PK. Chronic pain syndromes of gynecologic origin. *J Reprod Med* 2004;49:230–234.

Voiding dysfunction of functional origin

Prevalence

- Data are scarce.
- Previous studies reported 2-25% prevalence rates among women referred for evaluation of LUTS

Diagnosis

- No standard definitions.
- Pressure-flow study: an objective urodynamic examination considered to be the best method to assess the voiding phase of the micturition cycle.
- Uroflowmetry is a composite measure of the interaction between the pressure generated by the detrusor and the resistance offered by the urethra.
- EMG
- Video-urodynamic testing

Functional bladder outlet obstruction syndromes

Hinman syndrome

- Initially described in children with a non-neurogenic, neurogenic bladder. Presentation: increased daytime frequency, urgency, urinary incontinence, recurrent urinary tract infections, or occasionally, encopresis.
- Signs of obstructive uropathy, such as trabeculated bladder, elevated postvoid residual urine volume, hydronephrosis, and vesicoureteral reflux, in the absence of any identifiable neurological or obstructive abnormality.
- Usually acquired after toilet training, reaches its peak of destructiveness in late childhood, and tends to resolve after puberty.
- Groutz et al suggested the term “learned voiding dysfunction” and used the following clinical and urodynamic criteria to establish the diagnosis:
 - 1) a suggestive clinical history, i.e.: LUTS and difficulty in voiding in public places, or during uroflowmetry/urodynamics, having to concentrate, relax, touch genitalia, listen to running water, etc.
 - 2) intermittent "free" uroflow pattern
 - 3) exclusion of neurological disorders, or anatomical causes of bladder outlet obstruction
 - 4) demonstration of typical external urethral sphincter contractions during micturition with either needle EMG, or fluoroscopic visualization of the urethra during voiding

- Contrary to children, in whom the main subjective hallmarks of the syndrome are urinary incontinence and recurrent urinary tract infections, adult patients present mainly with obstructive and/or irritative symptoms, while urinary incontinence is less prominent

Treatment

- Suggestion and bladder retraining, bladder drill, and biofeedback
- Pharmacologic therapy to treat detrusor overactivity, obtain striated muscle relaxation, or to inhibit contraction of the α -adrenergic innervated bladder neck.
- Uroflowmetry biofeedback
- Botulinum toxin urethral sphincter injection to restore bladder emptying in patients with voiding dysfunction has also been used with some success, but data are limited
- Sacral neuromodulation for LUTS

Although potentially successful in more than 80% of cases, the treatment can span 6 weeks to several years with occasional relapses, requiring cooperation and determination on the part of the child and her/his family

Fowler's syndrome

- Described as voiding dysfunction due to abnormal EMG activity of the urethral sphincter among young women, 64% of whom also had polycystic ovaries
- The nature of the EMG activity was such that it suggested a muscle membrane disorder and, therefore, a primary disorder of the sphincter relaxation rather than inappropriately timed sphincter activity that occurs in neurogenic detrusor-sphincter dyssynergia
- Other investigators reported increased urethral pressure profile and increased sphincter volume on ultrasound in these patients [45]. It was therefore speculated that the abnormal activity in the urethral rhabdosphincter leads to hypertrophy of these myofibers
- Urinary retention in patients with Fowler's syndrome is unlikely to resolve without treatment, and sacral neuromodulation is the only intervention that has been demonstrated to restore voiding

REFERENCES

- Groutz A, Blaivas JG, Pies C, Sassone AM. Learned voiding dysfunction (non-neurogenic, neurogenic bladder) among adults. *Neurourol Urodynam* 2001; 20:259-268.
- Hinman F Jr. Nonneurogenic neurogenic bladder (The Hinman syndrome)-15 years later. *J Urol* 1986; 136:769-777.
- Fowler CJ, Christmas TJ, Chapple CR, et al. Abnormal electromyographic activity of the urethral sphincter, voiding dysfunction, and polycystic ovaries: a new syndrome? *BMJ* 1988; 297: 1436.

Sexual Dysfunction and psychosocial factors related to Overactive Pelvic Floor

Anna Padoa, MD

Adapted from Chapter 2 (Overactive Pelvic Floor: Female Sexual Function; authors: Ellen Laan and Rik Van Lunsen) and Chapter 4 (Female Genital Pain and Penetration Disorders; author: Ahinoam Lev-Sagie) in “The Overactive Pelvic Floor”; editors: Anna Padoa and Talli Rosenbaum, Springer Editions, in press.

Female Genital Pain and Penetration Disorders

- Up to 16% of the female population is diagnosed with vulvar pain syndromes, also known as vulvodynia, defined as genital pain in the absence of an identifiable cause
- Often, there is a significant delay in diagnosis, with a reported mean time of two years and evaluation by up to 15 physicians prior to correct diagnosis.

Table 1: Differential Diagnosis of Vulvovaginal Pain

Vulvar pain related to a specific disorder

Infectious

- Candidiasis
- Trichomoniasis
- Herpes simplex virus
- Group A streptococcus
 - (Bacterial vaginosis causes discharge and malodor, but only little or no inflammation and therefore it rarely cause pain.)

Inflammatory conditions and dermatoses

- Vulvar contact dermatitis
- Lichen sclerosus
- Lichen planus
- Desquamative inflammatory vaginitis
- Immunobullous disorders

Hormonal

- Vulvovaginal atrophy (estrogen deficiency)

Neoplastic

- Vulvar intraepithelial neoplasia –VIN
- Paget disease
- Squamous cell carcinoma

Neurologic

- Post-herpetic neuralgia
- Multiple sclerosis
- Spinal nerve compression

Systemic disorders

- Behcet's syndrome
- Crohn's disease

Vulvar pain not related to a specific disorder- Vulvodynia

1. Generalized
 1. Provoked (sexual, nonsexual, or both)
 2. Unprovoked
 3. Mixed (provoked and unprovoked)
2. Localized (vestibulodynia – previously known as vulvar vestibulitis, clitorodynia, hemivulvodynia, etc.)

1. Provoked (sexual, nonsexual, or both)
2. Unprovoked
3. Mixed (provoked and unprovoked)

Genital pain syndromes: provoked vestibulodynia and generalized unprovoked vulvodynia

PVD is the term describing a syndrome of provoked, localized allodynia of the vestibule of the vulva, not explained by another condition, and lasting more than 3 months

In **GVD** the patient reports a continuous, unpleasant sensation of pain, usually described as burning, stinging, irritating, itching, or a feeling of rawness. Most often the pain is diffuse, without clear borders.

Goldstein classifies PVD into groups, based on history and examination findings:

- 1) Hormonally mediated PVD
 - a. The pain began while taking hormonal contraceptive or other medications that affect hormones, after removal of ovaries, breastfeeding or menopause
 - b. low free testosterone
 - c. dryness, decreased libido, and decreased arousal
 - d. The entire vestibule is tender and vestibular mucosa is often dry and thin.
 - e. Treatment includes stopping hormonal contraception and application of topical estradiol (with or without testosterone) to the vestibule.
- 2) Hypertonic pelvic muscle dysfunction

- a. PF muscles become tight and tender.
- b. Patients often have other symptoms suggesting PF overactivity
- c. Predisposing factors, such as musculoskeletal disorders or anxiety
- d. The pain is much worse at 4-8 o'clock position of the vestibule with minimal or no pain in the upper vestibule.
- e. Treatment includes PF physiotherapy, with an optional addition of muscle relaxants (valium suppositories), Botulinum toxin injections and cognitive behavioral therapy.

3) Neuroproliferative PVD

- a. increased number of nociceptors in the vestibular mucosa.
- b. congenital and acquired forms. With acquired neuroproliferative PVD, the pain may begin after a severe allergic reaction or vaginitis
- c. tenderness of the entire vestibule.
- d. Treatments include topical anaesthetics, antidepressants, antiseizure drugs, capsaicin cream and a surgical procedure, termed "vulvar vestibulectomy".

REFERENCES

- Bachmann GA, Rosen R, Pinn VW, Utian WH, Ayers C, Basson R, et al. Vulvodynia: a state-of-the-art consensus on definitions, diagnosis and management. *The Journal of reproductive medicine*. 2006. p. 447–56.
- Haefner HK, Collins ME, Davis GD, Edwards L, Foster DC, Dee E, et al. The Vulvodynia Guideline. 2005;1–12.

- Goldstein A. Moving beyond the diagnosis of vestibulodynia. A holiday wish list. 2009;3227-9.
- Burrows LJ, Goldstein AT. The Treatment of Vestibulodynia with Topical Estradiol and Testosterone. Sex Med. 2013;1(1):30-3.
- Haefner HK. Report of the International Society for the Study of Vulvovaginal Disease terminology and classification of vulvodynia. J Low Genit Tract Dis . 2007;11(1):48-9.

- Overactive Pelvic Floor and Female Sexual Function

The involvement of pelvic floor muscles in sexual arousal and orgasm

- Masters and Johnson described voluntary and involuntary pelvic floor contractions during sexual arousal in both genders.
- Involvement of the levator ani in vaginal elongation, uterine elevation and vaginal muscle contractions was mainly described in terms of facilitation of male genital response, resulting from penile thrusting.

Does pelvic floor muscle training enhance sexual arousal and orgasm?

- Contrary to the promising findings of the early studies and contrary to common opinion, women who do not have a low tone pelvic floor and who seek to enhance sexual arousal and more frequent orgasms, have not much to gain from pelvic floor muscle training.
- Actually, a relaxed pelvic floor and mindful attention to sexual stimuli and bodily sensations seems a more effective means of enhancing sexual arousal and orgasm

Sexual function in women with pelvic floor dysfunction

- A relaxed or low tone pelvic floor is associated with better sexual function.
- That involuntary and rhythmic smooth muscle pelvic floor contractions contribute to the peak sensation of pleasure during orgasm does not imply that sexual arousal and orgasmic pleasure are enhanced by high tonus of the voluntary, striated muscles of the pelvic floor.

Pelvic floor overactivity and sexual arousal in women with sexual pain

- Apparently, genital response in women with dyspareunia is not impaired
- Genital response was found to be impaired by fear of pain

- Fear of pain may result not only in inhibited sexual arousal but also in increased pelvic floor activity, as part of a defensive reaction
- The pelvic floor musculature, like other muscle groups, is indirectly innervated by the limbic system and therefore highly reactive to emotional stimuli and states.

Provoked Vestibulodynia and Vaginismus

- PVD, as described earlier, is the most common form of superficial dyspareunia in premenopausal women
- Vaginismus, described in the DSMIV-TR as recurrent or persistent involuntary spasm of the musculature of the outer third of the vagina interfering with intercourse, may be characterized by high tone pelvic floor, chronically or in situations of attempted penetration (by any object).

Vaginismus and dyspareunia have been integrated in DSM 5 as genito-pelvic pain/penetration disorder.

Suggested vicious-cycle mechanisms:

- Increased pelvic muscle activity may be associated with reduced blood flow to the vagina.
- According to that hypothesis, in women with PVD the combination of increased pelvic floor muscle activity and lack of lubrication during intercourse results in friction between penis and vulvar skin, resulting in pain and possibly in tissue damage or irritation of the skin.
- Or, besides making vaginal entry more difficult, increased pelvic muscle activity may result in muscle pain, reduced blood flow to the vulva and vagina, and consequently, as a result of fear of pain, in reduced lubrication.

Pelvic floor overactivity as an emotional response

- The pelvic floor is involved in emotional processing. In cases of actual or imminent physical or mental pain the pelvic floor muscles will involuntarily, and often unconsciously, contract.
- For women who had been sexually abused in the past, the pattern of activity in the pelvic floor was different than for women without such

experiences: even consensual sexual situations can be experienced as threatening, and generate a protective pelvic floor response.

- Sexual difficulties in individuals with PTSD occur because the hormonal and neural circuit activation that normally leads to positively valenced sexual arousal and activity is already overactive in PTSD, possibly through reduced anterior cingulate activity, but leads to anxiety, fear, and other PTSD symptoms, such that sexual arousal signals impending threat rather than pleasure.
- PTSD: a major mediator in the relationship between the experience of rape and adverse health outcomes and a direct predictor of sexual problems.
- PTSD (as an anxiety disorder) may manifest itself in an OPF, as part of a generalized protective defense mechanism, which in turn might act as a mediator in the relation between rape and sexual problems.

Pelvic floor overactivity and attachment

- In recent years, attachment processes and attachment styles are increasingly acknowledged as important determinants of sexual problems in intimate relationships:
 - Securely attached individuals' beliefs about self and others and their effective emotion regulation strategies allow them to approach sexuality in a relaxed state of mind, and can enjoy sex for the pleasures involved.
 - Insecurely attached individuals may use sex to fulfill their attachment needs, leading to sexual experiences tarnished with anxiety, making it difficult to relax and enjoy sex.

REFERENCES

- van Lunsen R, Ramakers M. The hyperactive pelvic floor syndrome (HPFS): psychosomatic and psycho-sexual aspects of hyperactive pelvic floor

disorders with comorbidity of urogynecological, gastrointestinal and sexual symptomatology. *Acta Endoscopica* 2002;32:275–85

- Reissing ED, Binik YM, Khalife S, Cohen D, Amsel R. Vaginal spasm, pain, and behavior: An empirical investigation of the diagnosis of vaginismus. *Arch Sex Behav* 2004;33:5–17.
- Yehuda R, Lehrner A, and Rosenbaum TY. PTSD and sexual dysfunction in men and women. *J Sex Med* in press; doi: 10.1111/jsm.12856.
- Dewitte M. (2014) Different Perspectives on the Sex-Attachment Link: Towards an Emotion-Motivational Account. *J Sex Res.* 2012;49:105-24.
- Binik Y, Bergeron S, Khalife S. Dyspareunia. In: Leiblum S, Rosen RC, eds. *Principles and practices of sex therapy.* New York: Guilford Press; 2009:154–80.

Gastrointestinal co-morbidities and OPF

Marc Beer Gabel, MD

Adapted from Chapter 9 in “The Overactive Pelvic Floor”; editors: Anna Padoa and Talli Rosenbaum, Springer Editions, in press: “Overactive Pelvic Floor: Gastrointestinal morbidities”. Author: Marc Beer Gabel

Defecation:

- Synchronization of the autonomic and somatic neural functions of the pelvic floor muscles is essential in order to allow normal defecation
- Neural control:
 - The foregut is innervated by the vagal system
 - the hindgut by the sacral parasympathetic nerves

- the levator ani by sacral nerves
- the internal anal sphincter by nerves originating from L4 level
- the external anal sphincter by the pudendal nerve. Only the latter is volitional.

Defecation disorders are a common cause and effect of overactive pelvic floor and may be acquired in early childhood.

- Toilet training is the beginning of the cognitive control of continence.
- If the need to defecate is not properly perceived, recognized or accepted, continence may be affected.
- Children may learn to use a withholding mechanism and abstain from defecation, leading to inappropriate activation of the neuronal circuitry linking the rectal nerves to the sacral plexus, the spine and the brain, resulting in future abnormal behavior.
- 50% of constipated children contract the external anal sphincter during defecation.
- 95% of children with idiopathic constipation have impaired rectal sensation and weakening of rectal contraction during distension, which contributes to impaired rectal evacuation.
- One-third of children with idiopathic constipation continued to report severe complaints of constipation beyond puberty.

Constipation, pain and associated pelvic floor complaints

- In half of children with acute abdominal pain constipation is considered to be the cause of the pain.

- Prolonged contraction of muscles activate locally the free ends of afferent nerves fibers of the group III (thin myelinated fibers) and group IV (non-myelinated fibers), which transmit pain.
- ‘Cross talk’ between pelvic organs and pelvic muscular layers can enhance dysfunction and pain. Many patients have more than one underlying cause for their pain.
- Persistent levator ani contraction, which perpetuates the dysfunction, can be the result of:
 1. skeletal imbalance
 2. poor learned defecation habits
 3. a chronic visceral injury such as the neuroinflammation seen in irritable bowel syndrome (IBS) or interstitial cystitis
 4. a guarding reflex
- Constipated children frequently complain about urinary dysfunction, such as urinary tract infection (UTI) in 11% of the cases and urinary incontinence in 63% of the cases .
- The association of urinary dysfunction with constipation is supported by the observation that resolution of fecal retention leads to the disappearance of daytime urinary incontinence in 89% of the cases and of UTI in 100% of the children. (14).

Gastroenterological functional symptoms and sexual abuse

- There is a significant association between early sexual abuse, and gastroenterological functional symptoms (15).
- Patients with a history of sexual abuse were more likely to complain of both constipation and diarrhea.
- Anismus, a condition characterized by anal muscle contraction, was more frequent in sexual abuse survivors, suggesting a perturbation of pelvic floor function (16).

- In studies on sexually abused children, gastrointestinal disorders met the diagnostic criteria for somatization disorder, presenting with hypervigilance, anxiety and psychiatric disorders.
- These patients have poor quality of life due to health-related issues, utilize the health care system more often and report more pain (17).

Typical GI complaints in OPF patients

1. Constipation :with obstructive defecation, a sensation of incomplete rectal evacuation or anal blockage and a change of behavior with excessive straining to defecate and/or rectal digitation
2. Pain: during or after rectal evacuation, caused by tension of the hypertonic pelvic muscles. Cramping pain may be related to dyssynergic defecation and excessive abdominal straining.

Rome III classification of functional anorectal and pelvic pain:

- Chronic proctalgia
 - Levator ani syndrome
 - Unspecified functional anorectal pain
- Proctalgia fugax.

Pain can be also evoked by other pathologies, which should be excluded by a thorough examination, such as:

- Strained hypertonic pelvic floor muscles related to anal fissure.
- Prolapse of internal hemorrhoids
- Rectal mucosal prolapse with a recto-anal intussusception
- Overt rectal prolapse

Clinical examination

- Inspection of the perineum during contraction and relaxation
- Digital anal examination, to assess relaxation or contraction of the perineal muscles.
- Evaluation of tenderness on pelvic floor muscle palpation

Pelvic floor investigations

- Anorectal manometry and Rectal balloon evacuation test (BET)
- Imaging
 3. X-ray defecography
 4. Trans-perineal ultrasound
 5. MR defecography

Treatment

- Patient education and reassurance
- Nutritional management, treatment for constipation
- Biofeedback
- Electrical Stimulation
- Botulinum A Toxin
- Local analgesic treatment
- Oral Medications for chronic pain
- Sacral Nerve Neuromodulation (SNM)

REFERENCES

- Loening-Baucke V. Chronic constipation in children. *Gastroenterology* .1993Nov; 105(5):1557-64.

- Leroi AM, Bernier C, Watier A, Hémond M, Goupil G, Black R, Denis P, Devroede G Prevalence of sexual abuse among patients with functional disorders of the lower gastrointestinal tract. *Int J Colorectal Dis.* 1995; 10(4):200-6.
- Bharucha AE, Wald A, Enck P, Rao S. Functional Anorectal Disorders. *Gastroenterology* 2006;130:1510–1518.
- Rao SS, Welcher KD, Leistikov JS. Obstructive defecation : a failure of rectoanal coordination. *Am J Gastroenterol* .1998;93:1042-1052.
- Wald A, Bharucha AE, Cosman BC, Whitehead WE. ACG Clinical Guideline: Management of Benign Anorectal Disorders *Am J Gastroenterol* 2014; 109:1141–1157.
- Beer-Gabel M, Teshler M, Barzilai N, Lurie Y, Malnick S, Bass D, Zbar A. Dynamic transperineal ultrasound in the diagnosis of pelvic floor disorders: pilot study. *Dis Colon Rectum.* 2002 Feb;45(2):239-45; discussion 245-8.
- Wald A: Functional anorectal and pelvic pain .*Gastroenterol Clin North Am* 2001;30:243-51.

Workshop # 26, 11:00-12:30, Thursday October 8th 2015

The Overactive Pelvic Floor

Topic: Evaluating and understanding pelvic floor muscle overactivity - A physiotherapist's perspective

Mélanie Morin, PT, PhD
Associate Professor and Researcher
Research Center of the Centre Hospitalier Universitaire de Sherbrooke (CHUS)
School of Rehabilitation
Faculty of Medicine and Health Sciences
University of Sherbrooke, CANADA
Melanie.m.morin@usherbrooke.ca

Aims of this topic:

1. To present the current assessment tools for evaluating pelvic floor muscle (PFM) function and discuss these in light of muscle physiology. The psychometric properties of these tools will also be discussed;
2. To discuss the implication of PFMs in the pathophysiological mechanisms of pain syndromes and their role in bladder, bowel and sexual dysfunctions;
3. To present the findings in the literature on the effect of PFM physiotherapy on PFM function.

It has been recognized that PFM overactivity plays a crucial role in several conditions such as bladder and bowel elimination disorders, genital/pelvic pain syndromes and sexual dysfunctions and thus, an objective assessment is important to better understand the ongoing pathophysiological processes and hence orient physiotherapy treatment accordingly.

Muscle physiology

Terminology associated with PFM tone lacks standardisation and muscle physiology associated with muscle tone is often not well understood. General muscle tone, sometimes referred to as muscle tension, can be defined as the resistance provided by the muscle when pressure or a stretch is applied. Simons

and Mense [1] explained that general muscle tone in normally innervated skeletal muscles is composed of a passive and an active component [1]. The passive component consists of the viscoelastic properties of the muscle tissue related to several structures [2]: 1- the extensibility of actin-myosin cross-bridges; 2- non-contractile cytoskeleton proteins and 3- conjunctive tissues surrounding the muscle. The active component, consists of physiological contracture (more commonly defined as trigger point (TP)), electrogenic spasms (which include unintentional muscle contraction with or without pain that can be brought to voluntary control), and normal electrogenic contraction (involves resting activity in normally relaxed muscle and myotatic reflex during stretching). Only electrogenic spasms and normal electrogenic contractions involve electrical current propagating along muscle fibers that can be recorded by electromyography (EMG). It has been reported that electrogenic spasms may be related to psychological distress (e.g. anxiety), muscle overload or overuse (due for instance to inadequate posture) and inefficient uses (e.g. failure to fully relax after contraction) [1]. It should be pointed out that the presence of resting activity in normally relaxed muscle is controversial. Studies in skeletal muscle failed to find evidence of resting EMG [3, 4]. However, the PFMs may be an exception as Deindl et al. [5] suggested that some part of the levator ani may present sustained motor unit firing at rest.

Palpation: All components of general muscle tone evaluation are measured when using palpation. Different scales have been developed to evaluate PFM tone [6-12] and relaxation ability [8, 10, 13-15]. Women with vulvodynia for instance showed higher tonicity and lower flexibility [12, 16]. Although palpation remains a subjective tool, the ability to detect myofascial trigger points represents an important advantage over the other assessment techniques [17]. The assessment of trigger points is important in a pain condition and was demonstrated to play a key role in chronic pelvic pain in both men and women [18-26]. TP assessment has been deemed relevant not only because it is

associated with pain but it has also been reported that TP can provoke heightened PFM tone (related to electrogenic spasms) [18].

Electromyography (EMG): EMG measurement is basically the recording of the electrical current travelling along the muscle fibers. Viscoelastic properties and physiological contractures are not detectable using EMG. EMG signals represent electrogenic contraction and spasm. Hence, only a portion of general muscle tone is assessed. The role of the electrogenic component in the pathophysiology of vulvodynia has been shown by comparing women with pelvic/vulvar pain to asymptomatic controls [8, 27-29]. However, the results are controversial because some studies found a non-significant difference between the two groups [30-32]. This highlights the hypothesis that, in some women, the involvement of heightened PFM tone is not explained by an electrogenic cause. Such divergences may be explained by the various degrees of reliability found in the literature as well as some confounding factors (e.g. contact between the electrodes and the mucosa, vaginal lubrication, thickness of the vaginal tissue and cross-talk) interfering with measurements.

Ultrasound: Transperineal ultrasonography has been used to assess PFM morphology and function in men and women with various urological and gynecological conditions using transperineal and transabdominal methods [33-37]. PFM morphometry differs in women with vulvodynia and men with chronic pelvic pain in comparison to controls, suggesting an increase in general muscle tone [35, 38]. Transperineal ultrasound offers a great advantage in women with pain because it is a pain-free procedure (no vaginal insertion is required). Therefore, it was shown that these impairments are not limited to a protective defense reaction to the painful assessment but are rather present chronically. In addition to elevated PFM tone, findings also suggest a lower PFM strength in women with pain [35]. Furthermore, transperineal ultrasound was found to be useful in diagnosing rectoanal dyssynergy as compared to defecography in men [39] and women [40] with symptoms of obstructed defecation.

Manometry: Vaginal resting pressure and maximal pressure during PFM voluntary contraction have shown acceptable reliability [41, 42]. Recommendations have been formulated to ensure the validity of the pressure measurement [43]: 1-performing vaginal palpation before using the perineometer to make sure the patient is able to correctly contract her PFMs; 2-observing the cranial movement of the vaginal probe during measurement of the muscle contraction and 3-not considering the contractions associated with the Valsalva maneuver or retroversion of the hip [44, 45]. Naess et al. (56) showed that women with provoked vestibulodynia had significantly higher vaginal resting pressure compared to controls. In agreement with the hypothesis that elevated PFM tone may not always be explained by an electrogenic cause, these authors' results were not corroborated by EMG, as they found a non-significant difference in resting activity between the two groups.

Intravaginal dynamometry: Several PFM dynamometers have been developed to assess PFM function in women [46-63]. The Montreal dynamometer has been used in women with vulvodynia and a higher general PFM tone, lower strength, lower speed of contraction have been found compared to asymptomatic controls [64]. This methodology offers the advantage of assessing the PFM during a dynamic stretch.

Myotonometer: The MyotonPro™ is an instrument that has been used in skeletal muscle of the limbs to assess muscle tone [65-67]. This technology has been used recently for PFM assessment by applying pressures externally on the perineum [68, 69]. Davidson et al. [68, 69] showed that women with vulvodynia had higher PFM tone (stiffness) compared to asymptomatic controls.

Overall, the available evidence in women and men with conditions related to an overactive pelvic floor suggests an elevated global PFM tone (measured by ultrasound, dynamometry and manometry), TPs (measured by palpation and palpometer), increased viscoelastic properties (dynamometer and EMG) and for

some patients, elevated tone explained by electrogenic causes (evaluated by EMG). Empirical findings also indicate that the assessment of PFMs should not be limited to tone since the contractile properties (strength, speed of contraction, control and endurance) were also shown to be altered.

Impact of physiotherapy on PFMs in chronic pelvic pain

Physiotherapy interventions include different modalities such as education, manual therapy, biofeedback, electrical stimulation and dilation technique [70]. Effectiveness of physiotherapy has been shown in many studies to reduce pain [12, 71-75]. Significant effects on muscle function have been reported [12, 72]. It should be pointed out that these modalities may predominantly target one of the two components of PFM tone (active vs passive). For instance, biofeedback specifically addresses electrogenic spasms defined as unintentional muscle contraction that can be brought to voluntary control. The patients can thus learn how to properly relax their PFMs and gain control. Moreover, it has been shown that manual therapy for releasing TPs results in PFM relaxation as measured by the reduction in resting EMG activity (i.e. reduction of the electrogenic spasms) [18]. Muscle stretching may address reduced flexibility associated with the viscoelastic properties of the tissue.

This presentation will draw upon these references:

1. Simons, D.G. and S. Mense, *Understanding and measurement of muscle tone as related to clinical muscle pain*. Pain, 1998. **75**(1): p. 1-17.
2. Gajdosik, R.L., *Passive extensibility of skeletal muscle: review of the literature with clinical implications*. Clin Biomech, 2001. **16**: p. 87-101.
3. Basmajian, J.V., *New views on muscular tone and relaxation*. Can Med Assoc, 1957. **77**: p. 203-205.
4. Clemmesen, S., *Some studies on muscle tone*. Proc R Soc Med, 1951. **44**(8): p. 637-46.
5. Deindl, F.M., et al., *Activity patterns of pubococcygeal muscles in nulliparous continent women*. Br J Urol, 1993. **72**(1): p. 46-51.
6. Devreese, A., et al., *Clinical evaluation of pelvic floor muscle function in continent and incontinent women*. Neurourol Urodyn, 2004. **23**(3): p. 190-7.
7. Dietz, H.P. and K.L. Shek, *The quantification of levator muscle resting tone by digital assessment*. Int Urogynecol J Pelvic Floor Dysfunct, 2008. **19**(11): p. 1489-93.
8. Loving, S., et al., *Pelvic floor muscle dysfunctions are prevalent in female chronic pelvic pain: A cross-sectional population-based study*. Eur J Pain, 2014.
9. Lamont, J.A., *Vaginismus*. Am J Obstet Gynecol, 1978. **131**(6): p. 633-6.
10. Reissing, E.D., et al., *Pelvic floor muscle functioning in women with vulvar vestibulitis syndrome*. J Psychosom Obstet Gynaecol, 2005. **26**(2): p. 107-13.
11. Boyles, S.H., et al., *Validating a clinical measure of levator hiatus size*. Am J Obstet Gynecol, 2007. **196**(2): p. 174 e1-4.
12. Gentilcore-Saulnier, E., et al., *Pelvic Floor Muscle Assessment Outcomes in Women With and Without Provoked Vestibulodynia and the Impact of a Physical Therapy Program*. J Sex Med, 2010. **7**: p. 1003-1022.
13. Messelink, B., et al., *Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence Society*. Neurourol Urodyn, 2005. **24**(4): p. 374-80.
14. Slieker-ten Hove, M.C., et al., *Face validity and reliability of the first digital assessment scheme of pelvic floor muscle function conform the new standardized terminology of the International Continence Society*. Neurourol Urodyn, 2009. **28**(4): p. 295-300.
15. De Ridder, D., et al., *Clinical assessment of pelvic floor dysfunction in multiple sclerosis: urodynamic and neurological correlates*. Neurourol Urodyn, 1998. **17**(5): p. 537-42.
16. Reissing, E.D., et al., *Vaginal spasm, pain, and behavior: an empirical investigation of the diagnosis of vaginismus*. Arch Sex Behav, 2004. **33**(1): p. 5-17.
17. Simons, D.G., J.G. Travell, and L.S. Simons, *Travell and Simons Myofascial Pain and Dysfunction: The Trigger Point Manual*. 2e édition ed. Vol. 1. 1999, Baltimore: Williams & Wilkins.
18. Weiss, J.M., *Pelvic floor myofascial trigger points: manual therapy for interstitial cystitis and the urgency-frequency syndrome*. J Urol, 2001. **166**(6): p. 2226-31.
19. Montenegro, M.L., et al., *Importance of pelvic muscle tenderness evaluation in women with chronic pelvic pain*. Pain Med, 2010. **11**(2): p. 224-8.
20. Fitzgerald, C.M., et al., *Pelvic floor muscle examination in female chronic pelvic pain*. J Reprod Med, 2011. **56**(3-4): p. 117-22.
21. Bassaly, R., et al., *Myofascial pain and pelvic floor dysfunction in patients with interstitial cystitis*. Int Urogynecol J, 2011. **22**(4): p. 413-8.
22. Doggweiler-Wiygul, R., *Urologic myofascial pain syndromes*. Curr Pain Headache Rep, 2004. **8**(6): p. 445-51.

23. Itza, F., et al., *[Myofascial pain syndrome in the pelvic floor: a common urological condition]*. *Actas Urol Esp*, 2010. **34**(4): p. 318-26.
24. Tu, F.F., et al., *Physical therapy evaluation of patients with chronic pelvic pain: a controlled study*. *Am J Obstet Gynecol*, 2008. **198**(3): p. 272 e1-7.
25. Shoskes, D.A., et al., *Muscle tenderness in men with chronic prostatitis/chronic pelvic pain syndrome: the chronic prostatitis cohort study*. *J Urol*, 2008. **179**(2): p. 556-60.
26. Anderson, R.U., et al., *Painful myofascial trigger points and pain sites in men with chronic prostatitis/chronic pelvic pain syndrome*. *J Urol*, 2009. **182**(6): p. 2753-8.
27. Glazer, H.I., et al., *Electromyographic comparisons of the pelvic floor in women with dysesthetic vulvodynia and asymptomatic women*. *J Reprod Med*, 1998. **43**(11): p. 959-62.
28. Frasson, E., et al., *Central nervous system abnormalities in vaginismus*. *Clin Neurophysiol*, 2009. **120**(1): p. 117-22.
29. White, G., M. Jantos, and H. Glazer, *Establishing the diagnosis of vulvar vestibulitis*. *J Reprod Med*, 1997. **42**(3): p. 157-60.
30. Reissing, E.D., et al., *Vaginal spasm, pain, and behavior: an empirical investigation of the diagnosis of vaginismus*. *Archives of Sexual Behavior*, 2004. **33**(1): p. 5-17.
31. Engman, M., H. Lindehammar, and B. Wijma, *Surface electromyography diagnostics in women with partial vaginismus with or without vulvar vestibulitis and in asymptomatic women*. *J Psychosom Obstet Gynaecol*, 2004. **25**(3-4): p. 281-94.
32. Naess, I. and K. Bo, *Can a pelvic floor muscle contraction reduce vaginal resting pressure and resting EMG activity?* *Neurourol Urodyn*, 2013. **32**(8): p. Abstract 102.
33. Nahon, I., et al., *Assessing muscle function of the male pelvic floor using real time ultrasound*. *Neurourol Urodyn*, 2011. **30**(7): p. 1329-32.
34. Davis, S.N., et al., *Use of pelvic floor ultrasound to assess pelvic floor muscle function in Urological Chronic Pelvic Pain Syndrome in men*. *J Sex Med*, 2011. **8**(11): p. 3173-80.
35. Morin, M., et al., *Morphometry of the pelvic floor muscles in women with and without provoked vestibulodynia using 4D ultrasound*. *J Sex Med*, 2014. **11**(3): p. 776-85.
36. Braekken, I.H., et al., *Morphological changes after pelvic floor muscle training measured by 3-dimensional ultrasonography: a randomized controlled trial*. *Obstet Gynecol*, 2010. **115**(2 Pt 1): p. 317-24.
37. Hilde, G., et al., *Continence and pelvic floor status in nulliparous women at midterm pregnancy*. *Int Urogynecol J*, 2012. **23**(9): p. 1257-63.
38. Davis, S.N., et al., *Use of pelvic floor ultrasound to assess pelvic floor muscle function in urological chronic pelvic pain syndrome in men*. *Journal of Sexual Medicine*, 2011. **8**(11): p. 3173-3180.
39. Viscardi, A., C. Ratto, and A. Parello, *Dynamic transperineal ultrasound in the workup of men with obstructed defecation: a pilot study*. *Dis Colon Rectum*, 2012. **55**(9): p. 976-82.
40. Martellucci, J. and G. Naldini, *Clinical relevance of transperineal ultrasound compared with evacuation proctography for the evaluation of patients with obstructed defaecation*. *Colorectal Dis*, 2011. **13**(10): p. 1167-72.
41. Frawley, H.C., et al., *Reliability of pelvic floor muscle strength assessment using different test positions and tools*. *Neurourol Urodyn*, 2006. **25**(3): p. 236-42.
42. Hundley, A.F., J.M. Wu, and A.G. Visco, *A comparison of perineometer to brink score for assessment of pelvic floor muscle strength*. *Am J Obstet Gynecol*, 2005. **192**(5): p. 1583-91.
43. Bo, K. and M. Sherburn, *Evaluation of female pelvic-floor muscle function and strength*. *Phys Ther*, 2005. **85**(3): p. 269-82.

44. Bo, K., et al., *Pelvic floor muscle exercises for the treatment of female stress urinary incontinence : II. Validity of vaginal pressure measurements of pelvic floor muscle strength and the necessity of supplementary methods for control of correct contraction.* Neurourol Urodyn, 1990. **9**: p. 479-487.
45. Bump, R.C., et al., *The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction.* Am J Obstet Gynecol, 1996. **175**(1): p. 10-7.
46. Caufriez, M., *Postpartum. Rééducation urodynamique. Approche globale et technique analytique*, in *Book chapter:21993*, Collection Maïte: Brussels, Belgium. p. 36-44.
47. Caufriez, M., *Thérapies manuelles et instrumentales en urogynécologie.* Brussels, Belgium ed1998.
48. Rowe, P. *A new system for the measurement of pelvic floor muscle strength in urinary incontinence.* in *In 12th International Congress of the World Confederation for Physical Therapy Abstract book.* 1995.
49. Sampselle, C.M., et al., *Effect of pelvic muscle exercise on transient incontinence during pregnancy and after birth.* Obstetrics & Gynecology, 1998. **91**(3): p. 406-412.
50. Howard, D., et al., *Racial differences in the structure and function of the stress urinary continence mechanism.* Obstetrics & Gynecology, 2000. **95**(5): p. 713-717.
51. Ashton-Miller, J.A., J.O.L. DeLancey, and D.N. Warwick, *Method and apparatus for measuring properties of the pelvic floor muscles*, US patent 6, 232 B1, Editor 2002.
52. DeLancey, J.O., et al., *Comparison of levator ani muscle defects and function in women with and without pelvic organ prolapse.* Obstet Gynecol, 2007. **109**(2 Pt 1): p. 295-302.
53. Dumoulin, C., D. Bourbonnais, and M.C. Lemieux, *Development of a dynamometer for measuring the isometric force of the pelvic floor musculature.* Neurourol Urodyn, 2003. **22**(7): p. 648-53.
54. Verelst, M. and G. Leivseth, *Are fatigue and disturbances in pre-programmed activity of pelvic floor muscles associated with female stress urinary incontinence?* Neurourol Urodyn, 2004. **23**(2): p. 143-7.
55. Verelst, M. and G. Leivseth, *Force-length relationship in the pelvic floor muscles under transverse vaginal distension: a method study in healthy women.* Neurourol Urodyn, 2004. **23**(7): p. 662-7.
56. Verelst, M. and G. Leivseth, *Force and stiffness of the pelvic floor as function of muscle length: A comparison between women with and without stress urinary incontinence.* Neurourol Urodyn, 2007. **26**(6): p. 852-7.
57. Constantinou, C.E. and S. Omata, *Direction sensitive sensor probe for the evaluation of voluntary and reflex pelvic floor contractions.* Neurourol Urodyn, 2007. **26**(3): p. 386-91.
58. Constantinou, C.E., et al., *Evaluation of the dynamic responses of female pelvic floor using a novel vaginal probe.* Ann N Y Acad Sci, 2007. **1101**: p. 297-315.
59. Romero-Culleres, G., et al., *Reliability and validity of a new vaginal dynamometer to measure pelvic floor muscle strength in women with urinary incontinence.* Neurourol Urodyn, 2013. **32**(658-659).
60. Parezanovic-Ilic, K., et al., *[Muscle strength measurement of pelvic floor in women by vaginal dynamometer].* Srp Arh Celok Lek, 2009. **137**(9-10): p. 511-7.
61. Nunes, F.R., et al., *Reliability of bidirectional and variable-opening equipment for the measurement of pelvic floor muscle strength.* PM R, 2011. **3**(1): p. 21-6.
62. Kruger, J., et al., *Test-retest reliability of an instrumented elastometer for measuring passive stiffness of the levator ani muscle.* Neurourol Urodyn, 2011. **30**(6): p. 865-867.
63. Kruger, J.A., et al., *An automated hand-held elastometer for quantifying the passive stiffness of the levator ani muscle in women.* Neurourol Urodyn, 2013.

64. Morin, M., et al., *Dynamometric assessment of the pelvic floor muscle function in women with and without provoked vestibulodynia*. *Neurourol Urodyn*, 2010. **29**: p. 1140-1141.
65. Agyapong-Badu, S., et al., *Anterior thigh composition measured using ultrasound imaging to quantify relative thickness of muscle and non-contractile tissue: a potential biomarker for musculoskeletal health*. *Physiol Meas*, 2014. **35**(10): p. 2165-2176.
66. Aird, L., D. Samuel, and M. Stokes, *Quadriceps muscle tone, elasticity and stiffness in older males: reliability and symmetry using the MyotonPRO*. *Arch Gerontol Geriatr*, 2012. **55**(2): p. e31-9.
67. Bailey, L., et al., *Parameters representing muscle tone, elasticity and stiffness of biceps brachii in healthy older males: Symmetry and within-session reliability using the MyotonPRO*. *J Neurol Disord*, 2013. **1**(16).
68. Davidson, M.J., *Perineal muscle stiffness in women with and without vulvodynia*, in *Melbourne School of Health sciences 2014*, The University of Melbourne: Melbourne, Australia. p. 207.
69. Davidson, M., A. Bryant, and H. Frawley, *Perineal muscle stiffness in women with and without vulvodynia: Reliability of measurement and differences in muscle stiffness*. *Neurourol Urodyn*, 2014. **33**(6): p. Abstract 46.
70. Hartmann, D., M.J. Strauhal, and C.A. Nelson, *Treatment of women in the United States with localized, provoked vulvodynia: practice survey of women's health physical therapists*. *J Reprod Med*, 2007. **52**(1): p. 48-52.
71. .
72. Bergeron, S., et al., *Physical therapy for vulvar vestibulitis syndrome: a retrospective study*. *J Sex Marital Ther*, 2002. **28**(3): p. 183-92.
73. Fisher, K.A., *Management of dyspareunia and associated levator ani muscle overactivity*. *Phys Ther*, 2007. **87**(7): p. 935-41.
74. Holland, A., *Case report: physical therapy intervention for dyspareunia*. *Journal of the Section on Women's Health*, 2003. **27**(1): p. 18-20.
75. Downey, P. and I. Frederick, *Physical therapy treatment for vulvar vestibulitis: a case report*. *Journal of Women's Health Physical Therapy*, 2006. **30**(1): p. 16-19.



Notes