

Chapter 25

Sexual pain disorders: dyspareunia and vaginismus

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Introduction

Pain is almost never “psychogenic,” except for pain from grieving. Pain has biological basis, when it is the alerting signal of an impending or current tissue damage from which the body should withdraw it is defined as “nociceptive” [1]. When pain becomes a disease per se, i.e. it is generated within the nerves and nervous centers, it is called “neuropathic” [1-3]. It is a complex perceptive experience, involving psychological and relational meanings, which may become increasingly important with the chronicity of pain [1-3].

Sexual pain disorders – dyspareunia and vaginismus – are very sensitive issues, as the pain involves emotionally charged behaviors: sexual intimacy and vaginal intercourse [4-6]. Most patients have been denied for years that their pain was real and feel enormously relieved when they finally meet a clinician who trusts their symptoms and commits him/herself to a thorough understanding of the complex etiology of their sexual pain.

Talking with patients about sexual pain disorders requires special attention to the sensitivity of the issue and an empathic attitude to the biological “truth” of pain [7]. This is the basis of a very rewarding clinician-patient relationship and is the basis of an effective therapeutic alliance.

Definition

Dyspareunia defines the persistent or recurrent pain with attempted or complete vaginal entry and/or penile vaginal intercourse.[8].

Vaginismus indicates the persistent or recurrent difficulties of the woman to allow vaginal entry of a penis, a finger, and/or any object, despite the woman’s expressed wish to do so. There is often (phobic) avoidance and anticipation/fear/experience of pain, along with variable involuntary pelvic muscle contraction. Structural or other physical abnormalities must be ruled out/addressed [8].

Although there is a longstanding tradition to distinguish female sexual pain disorders into vaginismus and (superficial) dyspareunia, recent research has demonstrated persistent problems with the sensitivity and specificity of the differential diagnosis of these two phenomena.

Both complaints may comprise, to a smaller or larger extent [5-7,9-10]:

- a. problems with muscle tension (voluntary, involuntary, limited to vaginal sphincter, or extending to pelvic floor, adductor muscles, back, jaws or entire body);
- b. pain upon genital touching: superficially located at the vaginal entry, the vulvar vestibulum and/or the perineum; either event-related to the duration of genital touching/pressure, or more chronic, lasting for minutes/hours/days after termination of touching; ranging from unique association with genital touching during sexual activity to more general association with all

types of vulvar/vaginal/pelvic pressure (e.g., sitting, riding horse or bicycle, wearing tight trousers)

- c. fear of sexual pain (either specifically associated with genital touching/intercourse or more generalized fear of pain, or fear of sex).
- d. propensity for behavioral approach or avoidance. Despite painful experiences with genital touching/intercourse, a subgroup of women continues to be receptive to sexual partner initiatives or to self-initiate sexual interaction.

However, as no consensus has been reached so far in unifying the two entities, they will be kept separate according to the latest classification [8].

Prevalence

Various degrees of dyspareunia are reported by 12-15% of coitally active women [11-13], up to 45.3% of postmenopausal women [14]. Vaginismus may occur in 0.5–1% of fertile women, although precise estimates are lacking [7]. However, mild hyperactivity of the pelvic floor, that could coincide with grade I or II vaginismus, according to Lamont [15] [Tab1], may permit intercourse while causing coital pain [16,17].

Pathophysiology

Vaginal receptiveness is a prerequisite for intercourse, and requires anatomical and functional tissue integrity, both in resting and aroused states [16-19]. Normal trophism, both mucosal and cutaneous, adequate hormonal impregnation, lack of inflammation, particularly at the introitus, normal tonicity of the perivaginal muscles, vascular, connective and neurological integrity and normal immune response are all considered necessary to guarantee vaginal 'habitability' [6,7,15-17]. Vaginal receptiveness may be further modulated by psychosexual, mental and interpersonal factors, all of which may result in poor arousal with vaginal dryness [5,7, 8-11].

Fear of penetration, and a general muscular arousal secondary to anxiety, may cause a defensive contraction of the perivaginal muscles, leading to vaginismus [7,9,10]. This disorder may also be the clinical correlate of a primary neurodystonia of the pelvic floor, as recently demonstrated with needle electromyography [20]. It may be so severe as to prevent penetration completely [15,16]. Vaginismus is the leading cause of unconsummated marriages in women. Co-morbidity between lifelong vaginismus and dyspareunia, and other FSD is frequently reported (Fig. 1). The defensive pelvic floor contraction may also be secondary to genital pain, of whatever cause [21,22].

Dyspareunia is the common symptom of a variety of coital pain-causing disorders (Tab.2). Vulvar vestibulitis (VV), a subset of vulvodinia, is its leading cause in women of fertile age [6,7,11,12,16,17, 23,24]. The diagnostic triad is: 1) severe pain upon vestibular touch or attempted vaginal entry; 2) exquisite tenderness to cotton-swab palpation of the introital area (mostly at 5 and 7, when looking at the introitus as a clock face); 3) dyspareunia [23].

From the pathophysiologic point of view, vulvar vestibulitis involves the up-regulation of: a) the immunological system, ie of introital mast-cells (with hyperproduction of both inflammatory molecules and nerve growth factors (NGF) [25-27]; b) the pain system, with proliferation of local pain fibers induced by the NGF [27-28], which may contribute to the hyperalgesia and allodynia, associated with neuropathic pain, reported by VV patients [2,3,6]; c) hyperactivity of the levator ani, which can be antecedent to vulvar vestibulitis and comorbid with vaginismus of a mild degree

[6,16,24], or secondary to the introital pain. In either case, addressing the muscle component is a key part of the treatment [28-30].

Hyperactivity of the pelvic floor may be triggered by non-genital, non-sexual causes, such as urologic factors (urge incontinence, when tightening the pelvic floor may be secondary to the aim of reinforcing the ability to control the bladder) [17], or anorectal problems (anismus, haemorrhoids, rhagades) [22].

Medical ("organic") factors, which are often under-evaluated in the clinical setting, may cause pain and they may combine with psychogenic (psychosexual) factors contributing to pain during intercourse. They include hormonal/dystrophic, inflammatory, muscular, iatrogenic, neurological and/or post-traumatic, vascular, connective and immunological causes [6,7,9-11,16-22,27].

Co-morbidity with other sexual dysfunctions – loss of libido, arousal disorders, orgasmic difficulties and/or sexual pain related disorders – is frequently reported with persisting/chronic dyspareunia [16]. The second leading etiology of dyspareunia in the fertile age is the post-partum pain associated with poor episiorraphy outcome and vaginal dryness secondary to the hypoestrogenic state when the woman is breast feeding [31].

Clinical approach

In sexual pain disorders, the accurate clinical history and careful physical examination are essential for the diagnosis and prognosis. Location and characteristics of pain have been demonstrated to be the most significant predictors of the etiology of pain [16,18,19,21,32]. No instrumental exam has so far been demonstrated to be more informative than a carefully performed clinical examination.

Focusing on the presenting symptom – dyspareunia - and with the above mentioned attention to the sensitivity of the issue, key questions to obtain the most relevant informations can be summarized as follows [16-18,33]:

- Did you experience coital pain from the very beginning of your sexual life onwards (lifelong) or did you experience it after a period of normal (painless) sexual intercourse (acquired disorder)?
If lifelong, were you afraid of feeling pain before your first intercourse?
When lifelong, dyspareunia might usually be caused by mild/moderate vaginismus (which allows a painful penetration) and/or coexisting, life-long low libido and arousal disorders.
- If acquired, do you remember the situation or what happened when it started?
The answer can give information about the “natural history” of the current sexual complaint.
- *Where* does it hurt? At the beginning of the vagina, in the mid vagina or deep in the vagina?
Location of the pain and its onset within an episode of intercourse is the strongest predictor of presence and type of organicity [32].
 - Introital dyspareunia may be more frequently caused by poor arousal, mild vaginismus, vestibulitis, vulvar dystrophia, painful outcome of vulvar physical therapies, perineal surgery (episiorrhaphy, colporrhaphy, posterior perineorrhaphy), pudendal nerve entrapment syndrome and/or pudendal neuralgia, Sjogren syndrome [5-7, 9-12, 14-19] (see also the chapter on iatrogenic factors).
 - Mid vaginal pain, acutely evoked during physical examination by a gentle pressure on the sacro-spinous insertion of the levator ani muscle, is more frequently due to levator ani myalgia, the most frequently overlooked biological cause of dyspareunia [6,16,17,21].
 - Deep vaginal pain may be caused more frequently by endometriosis or pelvic inflammatory disease (PID) or by outcomes of pelvic radiotherapy or vaginal radical surgery.

Varicocele, adhesions, referred abdominal pain, and abdominal cutaneous nerve entrapment syndrome (ACNES) are less frequent and still controversial causes of deep dyspareunia, which should nevertheless be considered in the differential diagnosis [16,17].

- *When* do you feel pain? Before, during or after intercourse?
 - Pain before intercourse suggests a phobic attitude toward penetration, usually associated with vaginismus, and/or the presence of chronic vulvar vestibulitis, and/or vulvodinia [24,35].
 - Pain during intercourse is more frequently reported. This information, combined with the previous-"where does it hurt?"- is the most predictive of the organicity of pain [5-7, 9-12, 14-19].
 - Pain after intercourse indicates that mucosal damage was provoked during intercourse, possibly because of poor lubrication, concurring to vestibulitis, pain and defensive contraction of the pelvic floor [6,16-17].
- Do you feel other accompanying symptoms, vaginal dryness, pain or paresthesias in the genitals and pelvic areas? Or do you suffer from cystitis 24-72 hours after intercourse?
 - Vaginal dryness, either secondary to loss of estrogen and/or to poor genital arousal may coincide with/contribute to dyspareunia [6,16-19].
 - Clitoralgia and/or vulvodinia, spontaneous and/or worsening during sexual arousal may be associated with dyspareunia, hypertonic pelvic floor muscles, and or neurogenic pain (pudendal nerve entrapment syndrome).[36].
 - Post coital-cystitis should suggest a hypoestrogenic condition and/or the presence of hypertonic pelvic floor muscles: it should specifically be investigated in post-menopausal women who may benefit from topical estrogen treatment [37] and rehabilitation of the pelvic floor, aimed at relaxing the myalgic perivaginal muscles [28-30].
 - Vulvar pruritus, vulvar dryness and/or feeling of a burning vulva should be investigated, as they may suggest the presence of vulvar lichen sclerosus, which may worsen introital dyspareunia [19]. Neurogenic pain may cause not only dyspareunia but also clitoralgia. Eye and mouth dryness, when accompanying dyspareunia and vaginal dryness, should suggest Sjogren's syndrome, a connective and immunitary disease [16-19].
- How intense is the pain you feel? Focusing on the intensity and characteristics of pain is a relatively new approach in addressing dyspareunia [5-7,16,17]. A shift from nociceptic to neuropathic pain is typical of chronic dyspareunia, and treatment may require a systemic and local analgesic approach [3,6].

Key point

Suggest that patient record a *diary of pain*, mirroring the menstrual cycle phases if the woman is in her fertile age (ie, starting every page with the first day of her cycle, with the date on the x axis, and the 24 hours of the day in the y axis. Pain intensity could be reported with three colours: zero=white; 1 to 3=yellow; 4 to 7=red, 8 to 10 black).

This would: 1) improve the recording and understanding of pain flares before, during and/or after cycle, and the circadian rhythm of pain, to improve the diagnosis of etiology and contributors of pain,; 2) suggest a better tailoring of the analgesic treatment; 3) make more accurate the recording of the impact of treatment of pain perception, with an easy to catch perspective [36]. Typically, nociceptive pain persists at night, while neuropathic pain is significantly reduced or absent during sleep.

Clinical approach

The diagnostic work-up of dyspareunia should focus on:

- accurate *physical* examination, to describe:
 - ✓ the ‘pain map’, i.e. any site in the vulva, introitus, midvagina and deep vagina where pain can be elicited [16,18,19,40];
 - ✓ pelvic floor trophism (and vaginal pH), muscular tonus, strength and performance and myogenic and referred pain [16,21];
 - ✓ signs of inflammation (primarily vulvar vestibulitis) [6,16,23];
 - ✓ poor outcome of pelvic [41] or perineal surgery (episiotomy/rraphy) [31];
 - ✓ associated urogenital and rectal pain syndromes [22];
 - ✓ neuropathic pain, in vulvodynia or localized clitoralgia with no objective findings [3,5,7];
- psychosexual factors, poor arousal and coexisting vaginismus [6,7, 9, 10, 15, 24];
- relationship issues [7,42];
- hormonal profile, if clinically indicated, when dyspareunia is associated with vaginal dryness [16-18].

In patients with vaginismus, the diagnosis and prognosis may be made based on three variables:

- intensity of the phobic attitude (mild, moderate, severe) toward penetration [7,9,10,15,16];
- intensity of the pelvic floor hypertonicity (in four degree, according to Lamont [15,28-30];
- co-existing personal and/or relational psychosexual problems [6,7, 9, 10, 15, 24].

Principles of Treatment of Sexual Pain Disorders

Sexual co-morbidity is a key issue in sexual pain disorders. Dyspareunia and vaginismus, because of coital pain, directly inhibit genital arousal and vaginal receptivity. Indirectly, they may affect the (coital) orgasmic potential during the intercourse and impair physical and emotional satisfaction, causing loss of desire for and avoidance of sexual intimacy. [16,18,19, 21, 26,40,42].

DYSPAREUNIA may benefit from a well tailored clinical approach, based on a clear understanding of the pathophysiology of coital pain the patient is complaining of, with attention to predisposing, precipitating and maintaining factors in any of the systems potentially involved [Table 3]:

1) Medical Treatment:

- **Multimodal therapy :**

Vulvar vestibulitis (VV) should be treated with a combined, multimodal treatment aimed at **reducing**:

- a) the **up-regulation of mastcells**, both by reducing the agonist stimuli (such as candida infections, microabrasions of the introital mucosa because of intercourse with a dry vagina and/or a contracted pelvic floor, chemicals, allergens etc) that cause degranulation leading to chronic tissue inflammation, and/or with antagonist modulation of its hyper-reactivity, with amitryptiline or aliamide’s gel [6,42,44];
- b) the **up-regulation of pain system** secondary to both the proliferation of introital pain fibers [25-27] induced by the Nerve Growth Factor produced by the up-regulated mast-cells, and the lowered central pain threshold [45]. A thorough understanding of the pathophysiology of pain, in its nociceptive and neuropathic component, is essential. Anthalgic treatment should be prescribed: locally, with electroanalgesia [39] or, in severe cases, with the ganglion impar block [3];

systemically with tricyclic antidepressant, gabapentin or pregabalin in the most severe cases, with a neuropathic component [3,6,38,43];

c) the **up-regulation of the muscular response**, with hyperactivity of the pelvic floor, which may precede vulvar vestibulitis, when the predisposing factor is vaginismus [6,24] or be acquired in response to genital pain [6,20,33]. In controlled studies, electromyographic feed-back [28-30] has been demonstrated to significantly reduce pain in VV patients. Self-massage, pelvic floor stretching and physical therapy may also reduce the muscular component of coital pain [6,40,42]. When hyperactivity of the pelvic floor is elevated, treatment with Type A. botulin toxin has been proposed on the basis of its efficacy and safety profile in the pelvic floor disorders of the hyperactive type [46,47].

Individually tailored combinations of this approach are useful to treat introital dyspareunia with etiologies different from VV.

Key point: patients- and couples – should be required to abstain from vaginal intercourse and use other forms of sexual intimacy – during VV treatment, until introital pain and burning feelings have disappeared. This is essential to prevent the introital microabrasion caused by penetration when there is vaginal dryness, overall poor genital arousal and/or tightened pelvic floor that would maintain the upregulation of mast-cells, pelvic floor defensive hypertonus and peripheral pain fibers' proliferation [6].

Deep dyspareunia, secondary to endometriosis, pelvic inflammatory disease (PID), chronic pelvic pain and other less frequent etiologies requires a specialist treatment that goes beyond the scope of this chapter.

- **Topical hormones**

Vaginal estrogen treatment is the first choice (when no contraindications are present) when dyspareunia is associated with genital arousal disorders and hypoestrogenism [16-19,37] (see also the sub-chapter on arousal disorders). This biological contributor of dyspareunia in long lasting hypothalamic amenorrhea, puerperium, and postmenopause can be easily improved with this topical treatment [16-19,37]. Safety of hormonal topical treatment is discussed in the sub-chapter on Hormonal Therapy. Vulvar treatment with testosterone (1% or 2% testosterone in Vaseline, oil or petrolatum) may be considered when vulvar dystrophy and/or lichen sclerosus contribute to introital dyspareunia.

2) Psychosexual Treatment

- **Psychosexual and/or behavioural therapy:**

This is the first line treatment of *lifelong* dyspareunia associated with *vaginismus* [6,40,48]. It should be offered in parallel with a progressive rehabilitation of the pelvic floor and a pharmacologic treatment to modulate the intense systemic arousal in the subset of intensely phobic patients [33,40]. In this latter group, co-morbidity with sexual aversion disorder should be investigated and treated (see the sub-chapter on sexual aversion).

This contributes to the multimodal treatment of **lifelong dyspareunia**, which is reported in one third of VV patients [40]. Anxiety, fear of pain and sexual avoidant behaviours should be addressed as well. The shift from pain to pleasure is key from the sexual point of view. Sensitive and committed psychosexual support of the woman and the couple are mandatory.

VAGINISMUS may as well be treated with a multimodal approach, given its complex neurobiological, muscular and psychosexual etiology.

Pharmacologic therapy

- ✓ Depending on the intensity of the phobic attitude, the general anxiety arousal may be reduced with pharmacologic treatment. [33,40];
- ✓ Botulin A toxin, injected in the levator ani when the patient is able to accept the injection [46,47].

Psychosexual behavioural therapy

- ✓ Address underlying negative affects (fear, disgust, repulsion to touch, but also loss of self-esteem and self-confidence, body image concerns, fear of being abandoned by the partner) when reported [48];
- ✓ Teach how to command the pelvic floor muscles and to control the ability to do so with a mirror [40,49];
- ✓ Encourage self-contact, self-massage, self-awareness, through sexual education. If the woman has a current partner, encourage active sexplay, to maintain and/or increase libido, arousal and possibly clitoral orgasm, with specific prohibition of coital attempts until the pelvic floor is adequately relaxed and the woman is willing and able to accept intercourse [33,49];
- ✓ When good pelvic floor voluntary relaxation has been obtained, teach how to insert a dilator under pelvic floor relaxation [33,49];
- ✓ Discuss contraception, if the couple does not desire children at present [33];
- ✓ Encourage the sharing of control with the partner;
- ✓ Give permission for more intimate play, inserting of penis with the woman in control;
- ✓ Support the possible performance anxiety of the male partner with vasculogenic active drugs [33];
- ✓ Support the couple during the first attempts, as anxiety is frequent and may undermine the result if not adequately addressed, both emotionally and pharmacologically. PDE-5 inhibitors are useful when performance ED is reported [33];
- ✓ If possible, recommend concurrent psychotherapy, sex therapy, or couples therapy when significant psychodynamic or relationship issues are evident [33,48,49].

Conclusion

Pain is rarely purely psychogenic, and dyspareunia is no exception. Like all pain syndromes, it usually has one or more biological etiologic factors. Hyperactive pelvic floor disorders are a constant feature and co-morbidity with urological and/or proctological disorders is a frequent and yet neglected area to be explored for comprehensive treatment. Psychosexual and relationship factors, generally lifelong or acquired low sexual desire because of the persisting pain, and lifelong or acquired arousal disorders due to the inhibitory effect of pain, should be addressed in parallel, in order to provide comprehensive, integrated and effective treatment.

Vaginismus, which may contribute to lifelong dyspareunia, when mild/moderate, and may prevent intercourse, when severe, needs to be better understood in its complex neurobiological, muscular and psychosexual etiology and addressed as well with a multimodal approach.

Couple issues should be diagnosed and appropriate referral considered when the male partner presents with a concomitant Male Sexual Disorder.

References

1. Bonica J. Definitions and taxonomy of pain. In: Bonica J, ed. *The Management of Pain*. Lea & Febiger: Philadelphia, 1990.
2. Woolf C. The pathophysiology of peripheral neuropathic pain: abnormal peripheral input and abnormal central processing. *Acta Neurochir* 1993;Suppl 58:125-130.
3. Vincenti E, Graziottin A. Neuropathic pain in vulvar vestibulitis: Diagnosis and treatment. In: Guest, ed. *Female Sexual Dysfunction: Clinical Approach*. *Urodynamic* 2004,p. 112-6.
4. Foster D. Chronic vulval pain. In: MacLean AB, Stones RW, Thornton S, ed. *Pain in Obstetrics and Gynecology*. RCOG Press: London, 2001, pp 198-208.
5. Binik Y. Should dyspareunia be classified as a sexual dysfunction in DSM-V? A painful classification decision. *Arch Sex Behav* 2005(in press).
6. Graziottin A, Brotto LA. Vulvar vestibulitis syndrome: a clinical approach. *J Sex Marital Ther* 2004;30:125-39.
7. Pukall C, Lahaie M, Binik Y. Sexual pain disorders: Etiologic factors. In: Goldstein I, Meston CM, Davis S, Traish A, ed. *Women's Sexual Function and Dysfunction: Study, Diagnosis and Treatment*. Taylor and Francis: UK, 2005.
8. Basson R, Leiblum S, Brotto L. Revised definitions of women's sexual dysfunction. *J Sex Med* 2004;1:40-48.
9. Reissing E, Binik Y, Khalifé S. Does vaginismus exist? A critical review of the literature. *J Nerv Ment Dis* 1999;187:261-74.
10. 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;12:328-31.
11. Harlow B, Wise L, Stewart E. Prevalence and predictors of chronic lower genital tract discomfort. *Am J Obstet Gynecol* 2001;185:545-50.
12. Harlow B, Stewart B. A population-based assessment of chronic unexplained vulvar pain: have we underestimated the prevalence of vulvodynia? *J Am Med Womens Assoc* 2003;58:82-8.
13. Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. *Jama* 1999;281:537-44.
14. Oskai U, Baj N, Yalcin O. A study on urogenital complaints of postmenopausal women aged 50 and over. *Acta Obstet Gynecol Scand* 2005;84:72-8.
15. Lamont J. Vaginismus. *Am J Obstet Gynecol* 1978;131:632-636.
16. Graziottin A. Etiology and diagnosis of coital pain. *J Endocrinol Invest* 2003;26:115-21.
17. Graziottin A. Sexual pain disorders in adolescents. In: 12th World Congress of Human Reproduction, International Academy of Human Reproduction. 2005. Venice.
18. Graziottin A. Dyspareunia: clinical approach in the perimenopause. In: Studd J, ed. *The management of the Menopause*. 3th edition, The Parthenon Publishing Group: London, UK, 2003, pp 229-241.
19. Graziottin A. Sexuality in postmenopause and senium. In: Lauritzen C and Studd J, ed. *Current Management of the Menopause*, Martin Duniz: London, UK, 2004, pp 185-203.
20. Graziottin A, Bottanelli M, Bertolasi L. Vaginismus: a clinical and neurophysiological study. In: Guest, ed. *Female Sexual Dysfunction: Clinical Approach*. *Urodynamic* 2004, pp 117-121.
21. Alvarez D, Rockwell P. Trigger points: diagnosis and management. *American Family Physician* 2002;65:653-60.
22. Wesselmann U, Burnett AL, Heinberg LJ. The urogenital and rectal pain syndromes. *Pain* 1997;73:269-94.

23. Friedrich E. Vulvar vestibulitis syndrome. *J Reprod Med* 1987;32: 110-4.
24. Abramov L, Wolman I, David M. Vaginismus: An important factor in the evaluation and management of vulvar vestibulitis syndrome. *Gynecol Obstet Invest* 1994;38:194-197.
25. Bohm-Starke N, Hilliges M, Falconer C. Neurochemical characterization of the vestibular nerves in women with vulvar vestibulitis syndrome. *Gynecol Obstet Invest* 1999;48: 270-275.
26. Bohm-Starke N, Hilliges M, Blomgren B. Increased blood flow and erythema in posterior vestibular mucosa in vulvar vestibulitis. *Am J Obstet Gynecol* 2001;98: 1067-1074.
27. Bornstein J, Goldschmid N, Sabo E. Hyperinnervation and mast cell activation may be used as a histopathologic diagnostic criteria for vulvar vestibulitis. *Gynecol Obstet Invest* 2004;58:171-178.
28. Glazer H, Rodke G, Swencionis C. Treatment of vulvar vestibulitis syndrome with electromyographic feedback of pelvic floor musculature. *J Reprod Med*, 1995;40:283-290.
29. Bergeron S, Khalife S, Pagidas K. A randomized comparison of group cognitive-behavioural therapy surface electromyographic biofeedback and vestibulectomy in the treatment of dyspareunia resulting from VVS. *Pain* 2001;91:297-306.
30. McKay E, Kaufman R, Doctor U. Treating vulvar vestibulitis with electromyographic biofeedback of pelvic floor musculature. *J Reprod Med* 2001;46:337-342.
31. Glazener C. Sexual function after childbirth: women's experiences, persistent morbidity and lack of professional recognition. *Br J Obstet Gynaecol* 1997;104:330-5.
32. Meana M, et al. Dyspareunia: sexual dysfunction or pain syndrome? *J Nerv Ment Dis* 1997;185:561-9.
33. Plaut M, Graziottin A, Heaton J. *Sexual dysfunction*. Health Press, Oxford, UK, 2004.
34. Ferrero S, Esposito F, Abbamonte L. Quality of sex life in women with endometriosis and deep dyspareunia. *Fertil Steril* 2005.;83:573-9.
35. Graziottin A. Female sexual dysfunction. In: Bo K, Berghmans B, van Kampen M, Morkved S, ed. *Evidence Based Physiotherapy for the Pelvic Floor. Bridging Research and Clinical Practice*, Elsevier: Oxford, UK, 2005.
36. Shafik A. Pudendal canal syndrome as a cause of vulvodynia and its treatment by pudendal nerve decompression. *Eur J Obstet Gynecol Reprod Biol* 1998;80:215-20.
37. Simunic V, Banovic I, Ciglar S, Jeren L, Pavicic Baldani D, Sprem M. Local estrogen treatment in patients with urogenital symptoms. *Int J Gynecol Obstet* 2003;82:187-197.
38. Edwards L, Mason M, Phillips M. Childhood sexual and physical abuse: incidence in patients with vulvodynia. *J Reprod Med* 1997;42:135-9.
39. Nappi RE, Ferdeghini F, Abbiati I, Vercesi C, Farina C, Polatti F. Electrical stimulation (ES) in the management of sexual pain disorders. *J Sex Marital Ther* 2003;29(suppl 1):103-10.
40. Graziottin A. Treatment of sexual dysfunction, In: Bo K, Berghmans B, van Kampen M, Morkved S, ed. *Evidence Based Physiotherapy for the Pelvic Floor. Bridging Research and Clinical Practice*, Elsevier: Oxford, UK, 2005.
41. Graziottin A. Sexual function in women with gynecologic cancer: a review. *Italian Journal of Gynecology and Obstetrics* 2001;2:61-68.
42. Reissing E, Binik Y, Khalifé S. Etiological correlates of vaginismus: sexual and physical abuse, sexual knowledge, sexual self-schema, and relationship adjustment. *J Sex Marital Ther* 2003;29:47-59.
43. Shanker B, Mc Auley J. Pregabalin: a new neuromodulator with broad therapeutic indications. *Ann Pharmacother* 2005;39:2029-37.
44. Mariani L. Vulvar vestibulitis syndrome: an overview of non-surgical treatment. *Eur J Obstet Gynecol Reprod Biol* 2002;101:109-112.

45. Pukall CF, Binik YM, Khalife S, Amsel R, Abbott FV. Vestibular tactile and pain thresholds in women with vulvar vestibulitis syndrome. *Pain* 2002;96:163-75.
46. Maria G, Cadeddu F, Brisinda D. Management of bladder, prostatic and pelvic floor disorders with botulinum neurotoxin. *Curr Med Chem* 2005;12:247-65.
47. Ghazizadah S, Nikzed M. Botulinum toxin in the treatment of refractory vaginismus. *Obstet Gynecol* 2004;104:922-5.
48. Leiblum S. Vaginismus: a most perplexing problem. In: *Principles and Practice of Sex Therapy*. 3th edition, Guilford: New York, 2000, pp 181-204.
49. Katz D, Tabisel R. Management of vaginismus, vulvodynia and childhood sexual abuse. In: AP Bourcier, EJ McGuire, and P Abrams, ed. *Pelvic Floor Disorders*. Elsevier Saunders: Philadelphia, 2004, pp 360-369.

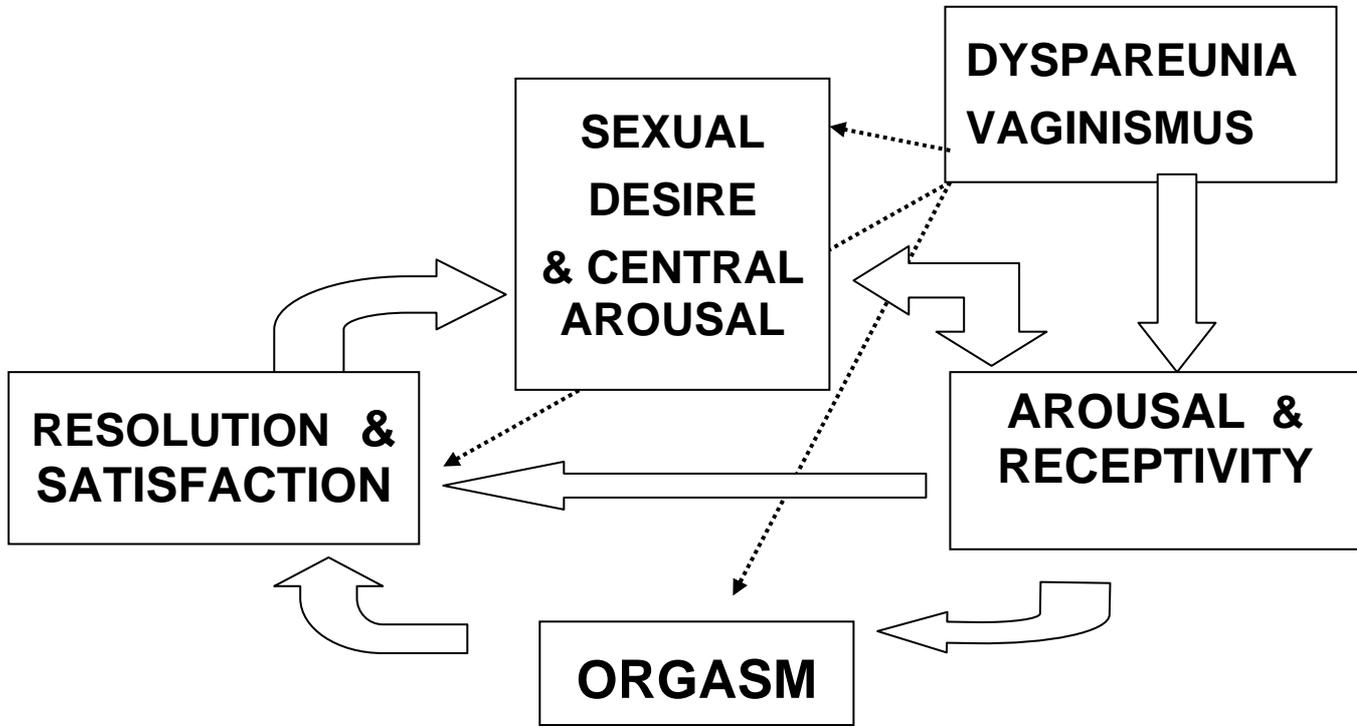
Tab. 1 Severity of Vaginismus

grades

- I Spasm of the elevator ani, which disappears with patient's reassurance**
- II Spasm of the elevator ani, which persists during the gynecologic/ urologic/ proctologic examination**
- III Spasm of the elevator ani and buttock's tension at any tentative or gynecologic examination**
- IV Mild neurovegetative arousal, spasm of the elevator, dorsal arching, thighs adduction, defense and retraction**
- XO Extreme defense neurovegetative arousal, with refusal of the gynecologic examination**

modified from Lamont J.A [15]

FIG 1 Circular model of female sexual function and the interfering role of sexual pain disorders



This simplified circular model contributes to the understanding of:

- 1) frequent overlapping of sexual symptoms reported in clinical practice (“co-morbidity”), as different dimensions of sexual response are correlated from a pathophysiologic point of view;
- 2) potential negative or positive feedback mechanisms operating in sexual function;
- 3) the *direct* inhibiting effect of dyspareunia and/or vaginismus on genital arousal and vaginal receptivity and the *indirect* inhibiting effect they may have on coital orgasm, satisfaction, sexual desire and central arousal, with close interplay between biological and psychosexual factors. Pelvic floor disorders (PFD) of the hyperactive type, causally related to sexual pain disorders as predisposing and/or maintaining factors (see chapter on Classification and etiology), may inhibit all the sexual response.

Modified from: Graziottin A. [18]

Tab. 2 Etiology of dyspareunia: different causes may overlap or be associated with coital pain with complex and dynamic pathophysiologic interplay.

A) Biological

a) superficial/introital and/or mid-vaginal dyspareunia

- **infectious: vulvitis, vulvar vestibulitis, vaginitis, cystitis**
- **inflammatory: with mastcell's up-regulation**
- **hormonal: vulvo-vaginal atrophy**
- **anatomical: fibrous hymen, vaginal agenesis, Rokitansky syndrome**
- **muscular: primary or secondary hyperactivity of levator ani muscle**
- **iatrogenic: poor outcome of genital or perineal surgery; pelvic radiotherapy**
- **neurologic, inclusive of neuropathic pain**
- **connective and immunitary: Sjogren's syndrome**
- **vascular**

b) deep dyspareunia

- **endometriosis**
- **pelvic inflammatory disease (PID)**
- **pelvic varicocele**
- **chronic pelvic pain and referred pain**
- **outcome of pelvic or endovaginal radiotherapy**
- **abdominal cutaneous nerve entrapment syndrome (ACNES)**

B)Psychosexual

- **co-morbidity with desire and /or arousal disorders, or vaginismus**
- **past sexual harassment and/or abuse**
- **affective disorders : depression and anxiety**
- **catastrophism as leading psychological coping modality**

C) Context or couple related

- **lack of emotional intimacy**
- **inadequate foreplay**
- **couple' conflicts; verbally, physically or sexually abusive partner**
- **poor anatomic compatibility (penis size and/or infantile female genitalia)**
- **sexual dissatisfaction and consequent inadequate arousal**

adapted from Graziottin, 2003 [16]

Tab 3 Treatment of dyspareunia

Medical

a) Inflammatory etiology (up-regulation of the mast-cells):

- Pharmacologic modulation of mastcells' hyper-reactivity
 - * with antidepressants: amitriptyline
 - * with lidocaine's topical gel
- Reduction of agonist factors causing the mast-cells' hyper-reactivity
 - * recurrent Candida or Gardnerella vaginitis
 - * microabrasions of the introital mucosa: from intercourse with a dry vagina
from inappropriate life-styles
 - *allergens/ chemical irritants
 - *physical agents
 - *neurogenic stimuli

b) Muscular etiology (up-regulation of the muscular system)

- Self-massage and levator ani stretching
- Physical therapy of the levator ani
- Electromyographic bio-feedback
- Type A Botulin Toxin

c) Neurologic etiology (up-regulation of the pain system)

- Systemic Analgesia
 - * amitriptyline
 - * gabapentin
 - * pregabalin
- Local Analgesia
 - * electroanalgesia
 - * ganglion impar block
- Surgical therapy: vestibulectomy (?)

d) Hormonal etiology

- Hormonal therapy
 - * local: vaginal estrogens
testosterone for the vulva
 - * systemic: with hormonal replacement therapies

Psychosexual

- Behavioral cognitive group therapy
- Individual Psychotherapy
- Couple Psychotherapy

modified from Graziottin, 2005 [42]