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Chapter Eleven

SEXUAL PAIN DISORDERS

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Dyspareunia (coital pain) is a very sensitive issue for women. Painful intercourse directly impacts sexual intimacy in women's relationships and so affects an emotionally charged aspect of their lives (Binik, in press; Foster, 2001; Graziottin, 2004). Talking with patients about sexual pain disorders requires sensitivity, empathy, and a willingness to acknowledge that the pain is rooted in a biological problem (Graziottin, 2001a, 2006b). This approach can be the basis of a very rewarding clinician-patient relationship and is the prerequisite for an effective therapeutic alliance. This chapter will describe the different types of sexual pain disorders (including vaginismus) in women and their prevalence, classification, etiology, clinical evaluation, and treatment.

DEFINITIONS

According to the Second International Consensus Conference Definitions of Women's Sexual Dysfunction, dyspareunia and vaginismus are defined as follows (Basson et al., 2003; also see appendix, volume 4).

- *Dyspareunia:* Persistent or recurrent pain with attempted or complete vaginal entry or penile-vaginal intercourse.
- *Vaginismus:* The persistent or recurrent difficulties of the woman to allow vaginal entry of a penis, a finger, 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.

PREVALENCE

Dyspareunia is reported by 12–15 percent of coitally active women (Laumann, Paik, & Rosen, 1999) and in up to 45.3 percent of postmenopausal women (Graziottin, 2004). Vaginismus may cause pain in one-half to one percent of coitally active women (personal clinical evaluation), but reliable data are lacking. In spite of their high prevalence, scientific interest in dyspareunia and other sexual pain disorders has been limited until recently (Friedrich, 1987; Graziottin, 2001a, 2003b, 2004; Meana, Binik, Khalife, & Cohen, 1997; Plaut, Graziottin, & Heaton, 2004; Graziottin, 2005).

Causes of sexual pain are closely related to the various reproductive phases of a woman's life (e.g., adolescence, childbearing years, perimenopause, and postmenopause). Treatment, in turn, depends on the cause of the pain. The etiology, diagnosis, and treatment of dyspareunia are discussed in detail below, but we would like to begin by presenting a few different clinical scenarios, all involving dyspareunia.

CASE HISTORIES

Four clinical case studies will illustrate how sexual pain can affect a woman's life and how an early diagnosis can definitely change the woman's and couple's sexual experience for the better. All names in the following case stories have been changed.

CASE 1

Marion is an 18-year-old girl. Her presenting problem is pain during intercourse: "I had pain from the very first time, two years ago, but felt it was normal. I love my boyfriend, and he adores me. We have been together for almost three years now. Contrary to my expectations, the pain I had did not fade away. Actually, it became worse and worse. Now, when I try to make love, I feel that my vagina is tight and dry. I cannot get aroused anymore because I'm afraid of pain. Even my sexual desire is going away. My family physician prescribed the contraceptive pill for me, suggesting that perhaps I was afraid of getting pregnant and did not relax. The pain didn't change. The day after our last intercourse, I developed a bladder infection. Everything is going wrong with me. Why is sex so painful for me, while all the other girls enjoy it a lot? I'm afraid I will never be able to have a normal sexual life."

Clinical examination: The clinical history revealed two predisposing factors for dyspareunia, one precipitating factor, intercourse; and several maintaining factors (including systemic anxiety).

When asked, Marion reported the following: (1) she could not use tampons during menstruation because she could not insert them ("I felt I had a wall there"); (2) since she was a girl, she was afraid of feeling pain at intercourse, in spite of longing for a closer intimacy with her boyfriend; and (3) she was not abused or harassed. These observations suggest that she could have a hyperactive pelvic floor and lifelong vaginismus, not severe enough to prevent intercourse but sufficient to predispose her to dyspareunia.

At clinical examination, her body language indicated a systemic anxiety and fear of being examined. She could not relax in spite of her physician's reassuring manner. Exquisite pain was elicited at five and seven o'clock (when viewing the vaginal opening as a clock face with the anus at six o'clock), between the hymen remnants and the introitus vaginae (the opening of the vagina). Tender points were elicited where the levator ani muscle inserts at the ischiatic spine, bilaterally.

Diagnosis: Lifelong dyspareunia, comorbid with vulvar vestibulitis and a tightened, myalgic pelvic floor. Lifelong vaginismus could be the predisposing factor. (Both vaginismus and vulvar vestibulitis will be described below.) Acquired genital arousal disorder and acquired loss of sexual desire were the comorbid female sexual disorders (FSD).

Comment: Comorbidity among FSDs and between FSD and medical conditions is frequent in women. A careful clinical history is essential to identify *predisposing, precipitating,* and *maintaining* factors, which may be biological, psychosexual, or relational. In this case the natural history of the current complaint had vaginismus as a likely predisposing factor, while intercourse was the precipitating factor. Maintaining factors were biological (vulvar vestibulitis) and psychosexual (systemic fear and anxiety about the pain). Acquired desire and arousal disorders increased the vulnerability of the introital mucosa to mechanical trauma due to lack of lubrication in response to sexual stimulation. The postcoital cystitis may have been precipitated by the mechanical trauma of having intercourse with a dry (nonaroused) vagina and a tightened pelvic floor. When adequately investigated, comorbidity between lower urinary tract symptoms and dyspareunia is frequent.

CASE 2

Laura is a 32-year-old woman. She reports a satisfying sexual life since her first intercourse, at age 16. She enjoys sex but "she gets rapidly bored with the same man," so she has had a number of partners. She has consistently used condoms as a contraceptive. No sexually transmitted infections are reported. Her last gynecological examination was performed three years before, with a normal record and normal pap smear.

She consults for a worsening, deep dyspareunia that appeared in the last two years. Periods are reported as having become progressively more painful since her adolescence. They are now incapacitating so that she has to take two days off work to stay home because of her "cramping" periods.

Clinical examination: She had a specific tenderness at the location of the uterosacral ligaments, with acutely elicited pain when these ligaments were palpated. An ovarian cyst of apricot size was present on the left side. The blood sample of the specific marker CA-125 was elevated (56 IU/mL). The ultrasound confirmed an ovarian cyst 4.8 centimeters in diameter, on the left, suggestive of endometriosis.

Diagnosis: Acquired deep dyspareunia due to pelvic endometriosis.

Comment: The incapacitating dysmenorrhea is a key symptom of endometriosis. Unfortunately, it is often neglected or ignored until more serious conditions develop, such as ovarian endometrioma, deep dyspareunia, or infertility. Besides endometriosis, which is the most frequent etiology of deep dyspareunia, pelvic inflammatory diseases should be considered as the second leading cause of deep dyspareunia.

CASE 3

Carol is 35 years old. She had her first child one year ago. Delivery was reported as very traumatic. A painful episiotomy was performed. Owing to the resulting infection, Carol "could not even walk for a week." Carol is still nursing her beautiful child, "who fortunately did not suffer from that awful delivery." Everything seemed to get better. "Unfortunately, when my husband and I tried to have intercourse for the first time four months ago, I felt a terrible aching pain. I shouted so loud that he lost his erection. We tried three other times, but my pain was excruciating. What can I do now? I have no more desire as I'm afraid of pain. Yes, I'm still without periods."

Clinical examination: Her episiotomy scar was tense and retracted. The vaginal pH was 6.0, suggesting vaginal dryness caused by the lack of estrogens due to continued breast-feeding. This could be a concomitant etiological factor.

Diagnosis: Acquired dyspareunia, acquired genital arousal disorder, and acquired loss of sexual desire.

Comment: The postpartum period is a difficult transitional phase for the woman and the couple. Besides making the adjustment to meeting their infant's needs, the couple has to face a major reassessment of their erotic intimacy. The most frequent complaint is vaginal dryness, which correlates with a genital arousal disorder. Comorbidity with low desire is common, while dyspareunia is more frequent when an episiotomy results in significant scarring.

CASE 4

Paula is a 58-year-old woman. She has been very happily married for 34 years and has three children. She is also the proud grandmother of two girls. She had no specific complaints at menopause, besides a few hot flashes, so she decided not to use hormone therapy. She has developed progressive vaginal dryness. In the last year, intercourse has become painful. "I love my husband, and I do not want to make him feel rejected. He feels that if I'm dry, I have no more desire for him. Well, yes, I do not have the drive I was used to, but I'd still enjoy our intimacy were it not for that pain that is becoming worse and worse. By the way, I often suffer from vaginitis with the same germ, *Escherichia coli*."

Clinical examination: External genitalia presented with vulvar dystrophy. The vaginal pH was 7.0, which suggested vaginal atrophy because of the persistent lack of estrogens.

Diagnosis: Acquired genital arousal disorder with acquired dyspareunia. Comorbidity with recurrent vaginitis from saprophytic pathogens *(E. coli)*.

Comment: Vaginal dryness is the second most frequent sexual complaint during the postmenopausal years, after loss of desire. Comorbidity with recurrent vaginitis and cystitis is frequent. Comorbidity between genital arousal disorder and dyspareunia is also frequent when the atrophic vaginitis is complicated by vulvar dystrophy, which contributes to introital dyspareunia.

These four different cases illustrate that pain during intercourse can present in many different ways, depending on (among other factors) where in her reproductive cycle the woman is. We will now turn to a more detailed description of the various forms of dyspareunia.

CLASSIFICATION

Sexual pain was included in the classification of FSDs established in 1998 during the First Consensus Conference (Basson et al., 2000) and then revised during the Second Consensus Conference in 2003 (Basson et al., 2003). Before 1998, sexual pain was viewed as a psychological concern, as evidenced by its inclusion in the World Health Organization's *International Statistical Classification of Disease and Related Health Problems–10* (ICD-10) and the American Psychological Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV). (The evolution of various classification systems for sexual disorders, including sexual pain, is outlined in appendix, volume 4.)

We now know that pain is almost never psychogenic, except for pain from grieving. Physical pain has a prominent biological basis and can be characterized as nociceptive or neuropathic. When the pain is a signal of impending or current tissue damage from which the body should withdraw to protect itself from further damage, it is defined as "nociceptive" (Bonica, 1990). When pain becomes a disease per se, that is, it is generated within the nerves and nervous centers, it is called "neuropathic" (Bonica, 1990; Vincenti & Graziottin, 2004; Woolf, 1993).

Pain is a complex, perceived experience, involving psychological as well as relational components, each of which may become increasingly important as the pain becomes chronic (Bonica, 1990; Vincenti & Graziottin, 2004; Woolf, 1993). The comorbidity between depression and pain, for example, is receiving increasing attention from researchers and clinicians. Persistently invalidating pain in a member of the couple or of the family can modify couple and family dynamics. This is another issue deserving clinical and therapeutic attention if the complexity of pain is to be addressed in an effective, multimodal approach.

ETIOLOGY

As the case histories presented earlier in the chapter demonstrate, the etiology of dyspareunia is complex (Brotto, Basson, & Gehring, 2003; Graziottin, 2001b, 2003a, 2003b, 2004, 2005, 2006a; Pukall, Lahaie, & Binik, 2005). Coital pain can be understood as the pain-dominated perception of a mosaic of interacting factors. Sexual pain disorders can therefore be considered as (1) multifactorial, (2) multisystemic, and (3) complex, as follows.

1. *Multifactorial:* Biological, psychosexual, and relational factors can coexist in a woman complaining of coital pain. Over time, these different factors may act as predisposing to, precipitating, or maintaining sexual pain disorders. The multifactorial nature of dyspareunia implies the need for a "dynamic" diagnosis, considering both the natural history of the sexual complaint and interacting factors.

2. *Multisystemic:* Sexual function involves the nervous, endocrine, vascular, muscular, and immunological systems and the integrity of the vaginal ecosystem. Thus the pathophysiology of coital pain also involves these various biological systems.
3. *Complex:* The overall experience of satisfying sexual function is greater than the sum of each physical or emotional factor. Similarly, the experience of coital pain is greater than the simple peripheral tissue damage that may initially trigger the nociceptive component (experienced through pain receptors) of coital pain.

When the clinician aims at understanding the etiology of dyspareunia, he or she should first distinguish between (1) introital/midvaginal pain and (2) deep pain. Each type of pain typically involves different sets of contributing factors. A woman complaining of sexual pain will usually describe the pain in very different ways when it is a pain at the introitus (vaginal opening) as compared to a deep pain. Pain at the introitus will create difficulties with vaginal penetration, while deep pain usually becomes worse during the thrusting phase of intercourse.

INTROITAL/MIDVAGINAL PAIN

This type of pain has several possible etiologies: inflammatory, hormonal, muscular, iatrogenic, neurological, immunological, vascular, and anatomical. Each of these will be described below.

I. *Inflammatory etiology:* Inflammation is one of the leading factors contributing to vulvar vestibulitis, which is considered to be the most frequent cause of chronic introital dyspareunia in women of reproductive age (Abramov, Wolman, & David, 1994; Friedrich, 1987; Graziottin, 2003b, 2004, 2005; Harlow, Wise, & Stewart, 2001; Harlow & Stewart, 2003; Pukall et al., 2005). According to Friedrich (1987), the diagnostic triad of vulvar vestibulitis includes the following: (1) severe pain on vestibular touch or attempted vaginal entry (the vestibule is the area around the vaginal opening); (2) exquisite tenderness to Q-tip (cotton swab) palpation of the introital area (mostly at five o'clock and seven o'clock, when looking at the introitus as a clock face); and (3) vestibular erythema of variable intensity.

The causes of vulvar vestibulitis are not straightforward. From a pathophysiological point of view, vulvar vestibulitis can involve (1) the immune system, (2) the nervous system (both peripheral and central), and (3) the muscular system.

A. *The up-regulation of the introital immune system,* that is, an increase in the number of local mast cells within the superficial mucosal layers (Bornstein et al., 2002; Bornstein, Goldsmith, & Sabo, 2004), and an increase in degranulation, with release of various inflammatory molecules (kinines, P substance, etc.). These changes cause vasodilatation with reddening of the vestibular mucosa, swelling (edema), and increased local temperature and burning (Bohm-Starke, Hilliges, & Falconer, 1999; Bornstein et al., 2004; Buhling

et al., 2006; Graziottin, 2004, 2005, 2006a; Plaut et al., 2004). The mast cells are the cellular mediators of the inflammatory involvement of the vestibular mucosa. Their up-regulation may be triggered by infectious, chemical, physical, immunological, neurogenic, or microtraumatic factors (Theoharides, Kempuraj, & Sant, 2001). Thus a very heterogeneous set of stimuli may all contribute to the chronic inflammation of the vestibular mucosa.

B. *The up-regulation of the nervous system:*

- At the *peripheral* level the local up-regulated mast cells overproduce nerve growth factor, which induces the proliferation of local pain fibers and their growth toward the most superficial layers of the introital mucosa (Bornstein et al., 2002, 2004). These anatomical changes are the morphological correlates of two key clinical symptoms of vulvar vestibulitis: the *hyperalgesia,* i.e., the perception of severe pain even at mild vestibular touch; and *allodynia,* i.e., the shift from tactile to burning pain, which is associated with neuropathic pain (Bohm-Starke et al., 1999; Graziottin, 2004, 2005, 2006a). Neurogenic pathways may also contribute to the local signs of inflammation, which are *vasodilatation* and consequent local vestibular erythema, which may be triggered by the release of calcitonin gene-related peptide from C nociceptors (Bohm-Starke et al., 1999). This neurogenic vasodilatation may coexist with the inflammatory vasodilatation due to histamine release by the mast cells and indicates the complex multisystemic pathophysiology underlying and contributing to introital pain.
- At the *central* level the reduction of the central pain threshold and the progressive switch from neuroceptive to neuropathic pain[1] (Bohm-Starke et al., 1999; Brotto et al., 2003; Buhling et al., 2006; Graziottin, 2003b) may explain why some women seem to be more susceptible to experiencing vaginal pain. Recent data seem to suggest that women vulnerable to chronic pain syndrome have a genetic predisposition to a higher production of inflammatory substances, such as interleukin, kinines, tumor necrosis factor alpha, and others, by the mast cells. Mast cells are inflammatory cells that produce these substances and release them within the tissues in response to agonist agents, such as bacteria, viruses, mechanical traumas, and allergic substances.

C. *The hyperactivity of the local muscular system:* The hyperactivity of the levator ani muscle may be primary (as in lifelong vaginismus contributing to vulvar vestibulitis) or may be acquired (in response to acquired introital pain associated with vulvar vestibulitis) (Graziottin, 2006a).

II. *Hormonal etiology:* There are several ways in which a lack of sexual hormones (estrogen and androgens) may contribute to the development of dyspareunia. (1) Estrogen or androgen deficiency may cause vulva-vaginal atrophy/dystrophy (atrophy is more severe than dystrophy), which can cause vaginal dryness. (2) Lack of these hormones may reduce the perivaginal and periurethral vascular congestive response (Graziottin, 2003a, 2003b, 2004, 2005; see chapters 3 and 8 of this volume). An inadequate vascular response,

secondary to poor genital arousal, may increase the vulnerability of the introital and bladder mucosa to coital microtraumas and acquired inflammatory response of the damaged tissue. (3) Clitoral and cavernosal bulb congestion may also be affected since androgens are permitting factors for nitric oxide (NO), a powerful vasodilating neurotransmitter in both men and women. Inadequate cavernosal arousal may contribute to vestibular or bladder pain (Graziottin, 2001a, 2003b, 2004, 2005; Plaut et al., 2004). (4) Hypoestrogenic conditions, such as menopause, breast-feeding amenorrhea, and hypothalamic amenorrhea, may cause an increase in vaginal pH value from 4.0 to 7.0. This reduction in acidity facilitates vaginal or bladder infections from intestinal bacteria (*Escherichia coli, Enterococcus faecalis,* etc.).

III. *Muscular etiology:* Pelvic floor disorders of the hyperactive type are key causes of introital dyspareunia. In particular, the hyperactivity of the levator ani muscle can be a preceding factor for vulvar vestibulitis and comorbid with vaginismus of a mild degree (Abramov et al., 1994; Graziottin, 2003b, 2004, 2006a), or it can be secondary to the introital pain due, for instance, to postcoital cystitis. (Note that pain elicited by digital pressure during the physical examination can be localized at the insertion of the levator ani at the ischiatic spine level (the so-called tender point) or referred to the surrounding areas (trigger point). Typically, it does not have a dermatomeric distribution. This means that it does not follow the pattern of distribution of nerves as determined by their embryonic origins.

Hyperactivity of the pelvic floor may be the following.

A. *Lifelong,* such as in primary vaginismus and associated obstructive constipation (Graziottin, Bottanelli, & Bertolasi, 2004);
B. *Acquired and triggered by gynecological factors,* such as vulvar vestibulitis, *or by sexual factors,* such as dyspareunia caused by inflammatory conditions, such as recurrent *Candida* infections, or from persistently poor genital arousal leading to vaginal dryness and introital pain;
C. *Acquired and triggered by nongynecological, nonsexual causes,* such as urological factors and anorectal problems. Urological factors can range from irritable bladder symptoms to urge incontinence, when tightening of the pelvic floor may be secondary to the aim of reinforcing the ability to control the bladder (Graziottin, 2006x). Anorectal problems include anismus, hemorrhoids, rhagades, and chronic constipation (Wesselmann, Burnett, & Heinberg, 1997).

IV. *Iatrogenic etiology:* "Iatrogenic" means that the physician or surgeon causes the pain. Vaginal pain can be iatrogenic when the dyspareunia is complained of after episiotomy, after perineal or vaginal surgery (anterior or posterior colporraphy), or after radical surgery for cervical cancer. Vaginal radiotherapy following cancer treatment may cause vaginal fibrosis, stenosis, and progressive damage of vaginal vessels, contributing to genital arousal disorders and both introital and deep dyspareunia.

Neurological damage may also be a cause of deep dyspareunia. This etiology may complicate radical hysterectomy or vaginal surgery. The so-called pudendal nerve entrapment syndrome may cause clitoralgia and introital dyspareunia (Shafik, 1998).

"Light" oral contraception may cause relative hypoestrogenism, resulting in dyspareunia.

V. *Neurological etiology:* This type of etiology is likely when neurogenic pain is in play; when pain has prominent neuropathic characteristics; or when neurodegenerative diseases, such as multiple sclerosis, are complicated by pain syndromes.

VI. *Immunological etiology:* The immune system may be involved in the etiology in two circumstances. One circumstance is when the mast cells are up-regulated, as exemplified in chronic inflammation. The other circumstance is when autoantibodies are secreted against the exocrine glands, causing Sjögren's syndrome, or against the vulvar connective tissue, contributing to lichen sclerosus.

VII. *Vascular etiology:* Vascular factors can be associated with coronary heart disease, diabetes, and smoking. These factors can contribute to genital arousal disorders, which may in turn lead to vaginal dryness and dyspareunia.

VIII. *Anatomical etiology:* Anatomical barriers such as a fibrous hymen, vaginal agenesis, or vaginal transversal septi can physically prevent penetration and make efforts to penetrate very painful.

DEEP DYSPAREUNIA

Deep dyspareunia may be caused by endometriosis, pelvic inflammatory disease (PID), pelvic varicocele, iatrogenic causes, neurological causes, referred abdominal pain, and chronic pelvic pain. Each of these factors is briefly discussed below.

IX. *Endometriosis:* This disease is due to the presence and growth of endometrial tissue outside the uterus. (The endometrium is the innermost lining of the uterus.) Just like the endometrium, this transplanted tissue proliferates under the influence of estrogen, matures under the influence of progesterone, and sheds at menstruation. These cyclical changes and shedding irritate the surrounding tissues, causing inflammation and pain. Endometriosis is the leading cause of deep dyspareunia, chronic pelvic pain, and infertility.

X. *Pelvic inflammatory disease (PID):* PID is most frequently caused by two sexually transmitted infections: *Chlamydia trachomatis,* the leading etiological agent, and gonorrhea. The endometrial and tubal damage resulting from infection may cause acute or chronic pelvic pain, deep dyspareunia, and infertility.

XI. *Pelvic varicocele:* It is caused by bulging ovarian veins, with an unusual preference for the left ovary. Frequently diagnosed with transvaginal ultrasound, it may occasionally cause deep dyspareunia. Its exact etiological role in deep dyspareunia is, however, still controversial.

XII. *Iatrogenic etiology:* Deep dyspareunia may be caused by surgery, that is, when radical hysterectomy must be performed to treat invasive cervical cancer. This surgery may require shortening of the vagina to prevent the spreading of a tumor to local tissue. Vaginal radiotherapy may further complicate the narrowing of the tissue, with vaginal fibrosis, stenosis, and progressive damage of vaginal vessels contributing to genital arousal disorders and both introital and deep dyspareunia. (Graziottin 2001b)

XIII. *Neurological etiology:* Neurological damage may be a cause of deep dyspareunia. This etiology may complicate radical hysterectomy or vaginal surgery. The so-called pudendal nerve entrapment syndrome may contribute to deep dyspareunia, besides causing clitoralgia and introital dyspareunia.

XIV. *Referred abdominal pain:* Myalgic muscles may cause referred pain. Trigger points on the levator ani, at the insertion of the muscle to the ischiatic spine, may cause referred pain, which characteristically has no dermatomeric irradiation (see above).

XV. *Chronic pelvic pain (CPP):* This complex pain syndrome may be rooted in different organs and tissues. CPP is a cyclic or noncyclic pain of six months or more in duration that localizes to the anatomical pelvis, severe enough to cause functional disability that requires medical or surgical treatment.

CLINICAL EVALUATION

The clinical evaluation of dyspareunia consists of (1) taking a thorough clinical history and (2) performing a physical examination. Whenever sexual pain disorders are being diagnosed, the following subtypes should be specified:

- lifelong versus acquired, focusing on time-related characteristics;
- generalized versus situational, according to the context, physical or relational, where it may be experienced;
- organic, psychogenic, mixed, or unknown, focusing on the etiology;
- highly distressing, moderately distressing, or nondistressing, according to how intensely the woman experiences her coital pain.

Clinical History

Focusing on the presenting symptom—dyspareunia—and keeping the sensitivity of the issue in mind, the key questions can be summarized as follows (Graziottin, 2003a, 2003b, 2005; Plaut et al., 2004).

1. *Where does it hurt?* The answer to this question, together with the answers to the following questions, has proven to be the most predictive of the origin of pain. (See the section on pain maps.)

2. *When does it hurt?* Pain can be lifelong or acquired; pain may be perceived before intercourse (phobic attitude toward penetration, usually associated with vaginismus or vulvar vestibulitis or vulvodynia (Abramov et al., 1994; Graziottin, 2005)); at the beginning/during/at complete penetration (see the section on pain maps); or after intercourse (arousal disorder associated with levator ani myalgia (muscle pain) and vulvar vestibulitis).
3. *How long does it hurt?* An association with vulvar vestibulitis can be suspected if pain is still present after the end of intercourse and if it lasts for two to four days.
4. *Do you feel other accompanying symptoms?*[2] Possible accompanying symptoms include urinary symptoms; vaginal dryness (coexisting eye and mouth dryness should suggest Sjögren's syndrome (Graziottin, 2003a, 2003b, 2006a)); vulvar itchiness/burning/dryness, which may suggest the presence of vulvar lichen sclerosus, which may worsen introital dyspareunia (Graziottin, 2004); pain or paresthesias in the genitals and pelvic areas; or cystitis 24–72 hours after intercourse.
5. *How intense is the pain you feel?* The pain can be evaluated with a self-administered analogical scale. The patient records a diary of pain, mirroring the menstrual cycle phases if the woman is of reproductive age (i.e., starting every page with the first day of her cycle, with the date on the *x* axis and the 24 hours of the day on the *y* axis). Pain intensity can be portrayed with three colors, that is, zero is white; 1–3 is yellow; 4–7 is red, 8–10 is black. This would (1) improve the recording and understanding of pain flares before, during, and after her cycle; (2) describe the circadian rhythm of pain, to improve the diagnosis of etiology and contributors of pain (typically, nociceptive pain tends to persist during sleep, while neuropathic pain is markedly reduced or disappears during sleep); (3) suggest a better tailoring of the analgesic treatment; and (4) make the recording of the impact of treatment on pain perception more accurate, with a quick glance at the diary being adequate to understand the results (Graziottin, 2005, 2006a).

Physical Examination

This should include an evaluation of the following.

1. *The external genitalia:* The health practitioner should give special attention to signs of vulvar/vestibular inflammation or vulvar dystrophy.
2. *The pain map:* The location of pain and the nature of its onset within an episode of intercourse are the strongest predictors of the presence and type of organicity (Graziottin, 2001a, 2003b, 2004; Meana et al., 1997; Plaut et al., 2004). An accurate clinical examination may exactly reproduce the site and characteristics of pain in 90 percent of patients. Unfortunately, the majority of gynecologists and sexual health providers are not trained in the diagnostic evaluation of pain maps. An inadequate clinical examination may fail to reveal the biological components of coital pain, facilitating an erroneous diagnosis of a psychological problem, for example, making claims that the pain is all in the patient's head or is psychogenic. Omitting the pain map will thus (1) perpetuate ignorance of the physical contributors to dyspareunia, facilitating the shift from nociceptive to neuropathic pain; (2) worsen a sense of helplessness and inadequacy in the affected woman; and (3) contribute to depression and negative relational dynamics (Graziottin, 2001a, 2005, 2006a; Graziottin & Brotto, 2004).

Pain Differentiation

The examining clinician should be able to differentiate among the following.

Introital Dyspareunia

Introital dyspareunia may be caused by poor arousal, mild vaginismus, vulvar vestibulitis, vulvar dystrophy, painful outcome of vulvar physical therapies, perineal surgery (episitomy, episiorraphy, colporraphy, posterior perineorraphy), pudendal nerve entrapment syndrome or pudendal neuralgia, and Sjögren's syndrome. Vulvar vestibulitis is the primary etiology of chronic dyspareunia in women of reproductive age. The second leading etiology of dyspareunia in women of reproductive age is the postpartum pain associated with poor episiotomy outcome and vaginal dryness secondary to the hypoestrogenic state when the woman is breast-feeding (Buhling et al., 2006; Glazener, 1997). Usually, introital and midvaginal dyspareunia are coexisting and referred to by the woman as "pain at the beginning of intercourse."

Midvaginal Pain

Lateral This type of pain is most frequently due to levator ani myalgia (Alvarez, & Rockwell, 2002; Graziottin, 2001a, 2003b, 2004, 2006a; Plaut et al., 2004). Early diagnosis of a hyperactive pelvic floor in adolescents may reduce their risk of vulvodynia and dyspareunia later in life (see case 1) (Graziottin, 2005).

Anterior This pain is associated with urologic factors, including the bladder and urethral damage associated with postcoital cystitis; recurrent cystitis; irritable bladder symptoms, that is, frequency, urgency, and urge incontinence; and when the contraction of the pelvic floor may be secondary to the aim of reinforcing the ability to control the bladder (Graziottin, 2005; see also chapter 9, volume 4, describing how the urethra passes through the pelvic floor muscles; contraction of the pelvic floor muscles participates in bladder control).

Posterior This particular type of pain is variably associated with anorectal problems (anismus, hemorrhoids, rhagades, obstructive constipation) (Graziottin et al., 2004; Graziottin, 2006a; Wesselmann et al., 1997).

Deep Vaginal Pain

Deep vaginal pain is caused most frequently by endometriosis (see case 2) and PID and less frequently by pelvic radiotherapy or vaginal radical surgery. Varicocele, adhesions, referred abdominal pain from myalgic muscles, and abdominal cutaneous nerve entrapment syndrome are still controversial causes of deep dyspareunia, which should nevertheless be considered in the differential diagnosis (Graziottin, 2003a,b, 2006a).

As summarized in Table 11.1, vaginal pH offers an easy to evaluate test of vaginal ecosystems which is easily shared with the woman. Other signs of chronic pelvic pain need to be considered.

Comorbidity with Vaginismus

In patients with vaginismus (Table 11.2) the diagnosis and prognosis may be made based on three variables (Abramov et al., 1994; Bergeron, Khalife, & Pagidas, 2001; Glazer, Rodke, & Swencionis, 1995; Graziottin, 2003b, 2005 Lamont, 1978; McKay, Kaufman, & Doctor, 2001; Pukall et al., 2005; Reissing, Binik, & Khalifé, 1999; van der Velde, Laan, & Everaerd, 2001):

- intensity of the phobic attitude toward penetration (mild, moderate, severe);
- intensity of the pelvic floor hypertonicity;
- coexisting personal or relational psychosexual problems.

Mild cases of vaginismus may contribute to lifelong dyspareunia (see case 1). Intercourse is usually possible in these mild cases, but with a defensively contracted pelvic floor and poor genital arousal, which contributes to mechanical

Table 11.1
Physical Examination Evaluations

1. The external genitalia: color and condition, with attention to signs of inflammation or dystrophy
2. The pain map: vulvar, introital, midvaginal, deep
3. Pelvic floor tonus and condition (muscular tonus, strength, and performance)
4. Vaginal pH
5. Myogenic referred pain
6. Poor outcomes of pelvic or perineal surgery (episiotomy) or radiotherapy
7. Other signs of pelvic or abdominal tenderness associated with chronic pelvic pain

Table 11.2
Severity of Vaginismus

Grade	Description of patient behavior during the clinical examination
I	Spasm of the levator ani, which disappears with patient's reassurance
II	Spasm of the levator ani, which persists during the gynecological, urological, and proctological examination
III	Spasm of the levator ani and buttock's tension at any tentative or gynecological examination
IV	Mild/moderate neurovegetative arousal, spasm of the levator ani, dorsal arching, thighs adduction, defense and retraction
XO	Extreme defense and severe neurovegetative arousal, with refusal of the gynecological examination

Source: Modified from Lamont (1978).

introital microabrasions (Graziottin, 2006a). Severe vaginismus prevents any kind of vaginal penetration. It is the leading female cause of unconsummated marriages (see chapter 9, volume 4).

DIFFERENT REPRODUCTIVE STAGES

The etiology and hence the treatment of dyspareunia varies according to the reproductive stages of a woman's life. Women of reproductive age complaining of dyspareunia often are affected by vulvar vestibulitis, hyperactivity of the levator ani muscle, vaginismus, and relative hypoestrogenism due to "light" oral contraception (15 micrograms of ethynilestradiol) (Caruso et al., 2004) or prolonged amenorrhea (breast-feeding; anovulatory cycles, especially in adolescents; hypothalamic amenorrhea) (Graziottin, 2001, 2003b, 2006a).

After menopause, the leading cause of dyspareunia is hypoestrogenism with subsequent vaginal dystrophy or atrophy (Graziottin, 2003a, 2006a). The progressive involution of the external genitalia is called "vulvar dystrophy." It is caused by aging or loss of androgens or autoantibodies (the latter are more likely to play a role in the special type of vulvar dystrophy called "lichen sclerosus," which causes vulvar dryness and itching). This may contribute to introital dyspareunia, potentiating the negative effects of vaginal dystrophy.

On a clinically relevant note, lichen sclerosus can be present also in children, adolescents, and young women. In sexually active adults it may cause not only entry dyspareunia but also genital arousal difficulties and loss of desire. This comorbidity demands a full genital examination, even in young patients complaining of low/loss of desire.

Hypoestrogenic conditions may cause an incremental increase in vaginal pH from 4.0 to 7.0. This decline in acidity alters the vaginal ecosystem, with the resulting loss of protective microorganisms, and facilitates vaginal or bladder infections from saprophytic germs of colonic origin (*Escherichia coli, Enterococcus faecalis,* etc.) (Gorodeski, 2005; Reid & Burton, 2002; Raz, 2001).

TREATMENT

Sexual pain disorders can be successfully treated if their complex etiologies are appropriately diagnosed and addressed, with a focused attention to the biological components, which until recently have been almost systematically neglected (Graziottin, 2001a, 2003b, 2006a). Treatment of sexual pain disorders should be individually tailored with a multimodal approach, addressing predisposing, precipitating, and maintaining factors as well as biological, psychosexual, and relational factors (Graziottin, 2006b). It often differs according

to which stage of the reproductive cycle a woman is in. Please note that all treatment suggestions in the following are recommendations only. Any type of medical treatment needs to be considered and prescribed by a qualified health provider.

Treatment of Dyspareunia in Adolescents

In adolescents, dyspareunia can be caused by genital arousal disorders with vaginal dryness, secondary to either psychosexual conditions, such as low desire and or inadequate mental arousal, or biological factors, such as inadequate vaginal estrogenic tissue levels due to "light" oral contraception, anovulatory cycles, or hypothalamic amenorrhea. Topical vaginal estrogen treatments (creams, tablets, or vaginal suppositories) are considered as the first choice when dyspareunia is associated with genital arousal disorders and hypoestrogenism (Graziottin, 2003a, 2003b, 2004, 2005; Simunic et al., 2003). Pelvic floor physical therapy (described in chapter 9, volume 4) is recommended when there is hypertonicity of pelvic floor muscles (including vaginismus) or when the pelvic floor responds with inverted command; the latter happens when the woman, instead of relaxing her pelvic floor muscles, contracts them, thus making penetration impossible or extremely painful.

Dyspareunia Associated with Vulvar Vestibulitis

General Recommendations

Appropriate lifestyle changes are the beginning of the healing process. Topical vaginal treatment should be avoided in the acute phase of the disorder as the local hyperregulated mast cells increase the likelihood of local allergic reactions (Graziottin, 2006b). Any associated medical condition, inclusive of depression and anxiety, should be treated. Intercourse, and other precipitating factors, should be avoided until the burning pain has disappeared (Graziottin , 2006b).

Noncoital intimacy should be recommended, with the exception of the subset of women reporting a worsening of symptoms even with mild arousal without genital stimulation. Glucose- and yeast-containing foods should be reduced to avoid recurrence of yeast infections (Graziottin, 2003a). Rubbing of hard tissues, like jeans, cycling, or riding, should be avoided to reduce vulvar trauma. Vulvar hygiene with water or diluted, low-pH (3.5–4.5) soap (Graziottin, 2003a) once or twice a day may reduce the irritant effect of soaps and the disruption of the physiologic protective mucosal layer of secretions.

Specific Medical Treatment of Infections Considered as Predisposing or Precipitating Factors

Chronic Mycosis This infection is present in up to 58.1 percent of vulvar vestibulitis patients versus a 5–8 percent prevalence in the general population

(Sobel et al., 2004) as a consequence of recurrent previous treatments with antibiotics. This high prevalence suggests the need for preventive treatment with antimycotic to avoid *Candida* recurrences when microabrasions of the introital mucosa are provoked by intercourse in nonaroused conditions or when pain blocks lubrication through an automatic neurovegetative reflex. Minimal mucosal lesions may activate even a small quantity of *Candida* spores living in the vaginal ecosystem which are otherwise silent. Treatment may include the following (note that these are only listed for informational purposes; any medication needs to be prescribed by a health provider):

- itroconazole (200 milligrams orally per day for three days, every two weeks for three months, then once a month for three months);
- fluconazole (150 milligrams once a week). This has been proven to give a significant reduction of *Candida* recurrences in patients with chronic candidacies: 90.8 percent at 6 months and 42.9 percent at 12 months (Sobel et al., 2004). The partner should be treated for the first month to avoid reinfection.

Gardnerella or Haemophilus's Infections These infections are generally associated with a vaginal pH of 5.0 or more. The use of boric acid's vaginal tablets (300 milligrams) once a day per 10 days a month demonstrated to be useful to reduce its recurrence (Graziottin & Brotto, 2004). Vaginal vitamin C tablets have a similar effect.

Pharmacologic Modulation of Mast Cells' Hyperreactivity

As described above, inflammatory causes of vulvar vestibulitis are due to an up-regulation of the mast cells. This can be treated in the following ways:

1. by reducing the agonist/triggering factors which enhance degranulation, such as vulvar-vaginal infections and microabrasions of the introital mucosa (from intercourse with a dry vagina or from inappropriate lifestyles), and by avoiding allergens/chemical irritants (see general recommendations);
2. by using drugs such as amitriptyline (Mariani, 2002; Graziottin, 2006b; Graziottin & Brotto, 2004; Reissing, Binik, & Khalifé, 2003), which, besides their central actions, have been shown to reduce mast cell activity, or by using a topical degranulation antagonist (aliamide).

Physical Therapy of the Muscular System

Hyperactivity of the pelvic floor, which may precede vulvar vestibulitis when the predisposing factor is vaginismus or be acquired in response to genital pain (Mariani, 2002; Graziottin, 2003a, 2006a; Graziottin et al., 2004; Graziottin & Brotto, 2004; Plaut et al., 2004), should be treated in parallel to the pharmacologic treatment. This is further discussed in chapter 9, volume 4.

Physical therapy includes patient education about the role of pelvic floor musculature in the maintenance of vulvar-vaginal pain; manual, hands-on

techniques (self-massage, levator ani stretching, and physiotherapy) (Graziottin, 2006a; Reissing et al., 2003; Graziottin & Brotto, 2004); electromyographic vaginal biofeedback (Glazer et al., 1995); electrical stimulation (Nappi et al., 2003); and type A botulin toxin (Maria, Cadeddu, & Brisinda, 2005; Ghazizadah & Nikzed, 2004). Botulin A toxin, injected in the levator ani when the patient is able to accept the injection, is performed at the level of the centrum tendineum with an insulin needle (Maria et al., 2005; Ghazizadah & Nikzed, 2004). This is usually done by the physician, not the pelvic floor physical therapist. Both work together in tandem.

To improve therapy, the couple should avoid coital attempts until the pelvic floor is adequately relaxed and the woman is willing and able to accept intercourse (Plaut et al., 2004; Katz & Tabisel, 2004). The use of progressive vaginal dilators, combined with active voluntary relaxation of the pelvic floor, may facilitate the learning of a positive control on the feelings evoked by penetration. This also may modify the "unconscious geography of genitals": "where there was a wall, there is a door" (Graziottin, 2006a, 2006b; Plaut et al., 2004). The main goal of this therapy is to rehabilitate the pelvic floor by (1) increasing proprioceptive awareness of the musculature; (2) improving muscle discrimination and relaxation; (3) normalizing muscle tone, thus reducing a further source of pain, relieving the myalgic component; (4) increasing elasticity of the tissues at the vaginal opening as well as desensitizing the painful area; (5) decreasing fear of vaginal penetration; and (6) improving the vascular flow and related tissue oxigenation, reduced by the tight muscle (Plaut et al., 2004).

Therapies Targeting the Pain System

Pain should be treated according to its severity, chronicity, and the degree of the patient's distress.

Systemic Analgesia In the most severe cases, when a neuropathic component is present (Graziottin & Brotto, 2004; Vincenti & Graziottin, 2004; Edwards, Mason, & Phillips, 1997; Shanker & McAuley, 2005), an oral analgesia including tryciclic antidepressants (amitriptyline, with increasing doses from 10 to 60 milligrams per day) and anticonvulsivants (gabapentin, 300–900 milligrams per day, or pregabalin, 75 milligrams twice a day) may be successfully considered. A further 75 milligrams may be added in the evening in case of severe neuropathic pain, with a total dose of 225 milligrams per day. This results in a rise of the pain threshold. The parallel treatment of the myalgic pelvic floor contributes to reducing this component of pain (Graziottin, 2006b; Graziottin & Brotto, 2004).

Local Analgesia Treatment with local analgesia can take two forms.

- Electroanalgesia involves applying very mild electric waves to the painful area. This provides a sense of delicate tingling and results in a progressive reduction of pain (Nappi et al., 2003).

- Ganglion impar block,[3] recently proposed (Vincenti & Graziottin, 2004), can be used when the characteristics of pain become unbearable and other treatments have failed (Vincenti & Graziottin, 2006).

Surgical Therapy: Vestibulectomy

This technique is to be reserved as a last resort to treat chronic vulvar vestibulitis that is not responsive to the above conservative treatments. The rationale is to remove the mucosal tissue with associated nerve proliferation and hypersensibility. Perineoplasty to remove the hymeneal ring and adjacent five millimeters of tissue is the surgical treatment of choice. The incision is extended from about five millimeters beneath and lateral to the urethra to the posterior fourchette, which is the area around six o'clock when viewing the vaginal opening as a clock face. Healthy tissue from the vagina is mobilized to cover the defect. The success rate, in terms of pain reduction, is about 60–70 percent. However, 25 percent of patients report persistence or worsening of vulvar vestibulitis symptoms after vestibulectomy.

Dyspareunia Associated with Puerperium

Hypoestrogenism due to breast-feeding can be easily improved by using topical vaginal estrogen treatments (creams, vaginal tablets, or vaginal suppositories, with preference for products that have the lowest systemic absorption, such as tablets, so that they do not interfere with lactation) (Graziottin, 2003a, 2003b, 2005, 2006b; Simunic et al., 2003). In case of poor outcome of episiotomy or perineal tears, manual, hands-on techniques (self-massage and physiotherapy) (Graziottin, 2006b; Graziottin & Brotto, 2004; Reissing et al., 2003) could be useful. Therapeutic ultrasound, used to treat dyspareunia after childbirth, has been studied but has not provided any conclusive results (Hay-Smith, 2000).

Dyspareunia in Postmenopause

Dyspareunia is often comorbid with vaginal dryness due to both lack of estrogens and genital arousal disorders. Current treatment options are dependent on the diagnosis and include hormonal supplements, physical therapy, psychological counseling, medication changes, and sexual devices, such as the Eros device, which provides suction to the clitoris, thereby locally improving blood flow (Walsh & Berman, 2004), and clitoral vibrators to elicit normal genital lubrication response when mental arousal is still blocked by fear of experiencing pain.

Topical Vaginal Estrogen Treatments

These can be used in case of vaginal atrophy/dystrophy caused by hypoestrogenism. This is a very common condition among postmenopausal women.

Vaginal dryness can also be overcome by lubricants when topical estrogens cannot or will not be used (Graziottin, 2003a, 2003b, 2004, 2005; Simunic et al., 2003; Dennerstein, Dudley, Hopper, Guthrie, & Burger, 2000; Castelo-Branco, Cancelo, Villero, Nohales, & Julia, 2005; Coope, 1996). Vaginal lubricants are available over the counter in pharmacies and drug stores.

The vaginal pH is modulated by the estrogen tissue levels, and it contributes to the maintenance of the normal vaginal ecosystem and bladder condition. This explains why optimal hormonal therapy (systemic or at least topical/vaginal) may prevent vaginal infections and urological symptoms. Physiological vaginal pH can be restored by using natural adicidificants, such as vitamin C or boric acid tablets, both to be applied inside the vagina.

After surgery for breast cancer, topical vaginal estrogen therapies are not contraindicated. Additional therapies, such as hypericus oil, self-massage, and physiotherapy, may provide symptom reduction.

Systemic Hormone Treatments

Premature ovarian failure and iatrogenic menopause (ovariectomy, pelvic radio/chemotherapy) are associated with more aggressive decay of general health and sexual wellness (Graziottin & Basson, 2004). Symptoms include FSDs with low desire and poor genital arousal, contributing to entry dyspareunia. An appropriate hormonal treatment (estro-progestinic or natural progesterone) is useful, when not contraindicated, until the age of 51 (Skouby et al., 2005; Alexander et al., 2004).

There are several large, key, placebo-controlled international trials of tibolone currently underway, one of which aims to test the safety of tibolone (vs. placebo) in women with a history of breast cancer who are suffering from climacteric symptoms (Eden, 2005; Kenemans & Speroff, 2005).

When vulvar dystrophy or lichen sclerosus are associated with dyspareunia, topical vulvar treatments with either testosterone (one or two percent testosterone in Vaseline, oil, or petrolatum) or cortisone cream may reduce the coital pain. It should be noted that this is an area of intense research. To date, the only FDA-approved use of testosterone in women in the United States is estratest (esterified estrogens and methyltestosterone), prescribed for the treatment of hot flashes.

Comorbidity with Anorectal or Urologic Symptoms

Anorectal problems can be associated with hyperactivity of the levator ani muscle (see above) (Wesselmann et al., 1997). Urological factors may be caused by the concomitant hyperactivity of the pelvic floor and local hypoestrogenism. They can be reduced when topical vaginal estrogen treatment is combined with pelvic floor relaxation (Graziottin, 2003a, 2003b, 2004, 2005; Simunic et al., 2003).

Other less frequent etiologies require a specialist treatment that goes beyond the scope of this chapter.

Treatment of Vaginismus

Vaginismus should be treated with a multimodal approach, given its complex neurobiological, muscular, and psychosexual etiology. Particular attention has to be given to the patient's sexual education to reduce generalized anxiety and systemic arousal, to modulate negative affects (such as fear, disgust, or repulsion to touch, body image concerns, loss of self-esteem and self-confidence, and fear of abandonment by the partner), and to improve the control of the pelvic floor muscles.

Depending on the intensity of the phobic attitude, the general anxiety arousal may be reduced with pharmacologic treatment (low-dose selective serotonin reuptake inhibitor or SSRI, such as paroxetine, anxiolytic, such as alprazolam, or myorelaxant, such as diazepam) (Graziottin, 2006b; Plaut et al., 2004).

The muscular component can be addressed with multiple approaches (see the paragraph on the muscular treatment in vulvar vestibulitis patients). It is important to remind the patient to avoid intercourse until therapies have had good results, while active foreplay is encouraged.

For patients affected by vaginismus, gentle masturbation and self- or couple massage can be very useful. Teaching better control of pelvic floor muscles can improve voluntary relaxation. This involves suggesting exercises with vaginal dilators. During the exercise the woman or her partner inserts lubricated, contoured cylinders of increasing diameter into the vagina, while the woman actively relaxes the levator ani muscles, so that the vagina gradually accommodates to an object approximating the size of the erect penis (Plaut et al., 2004).

If possible, concurrent psychotherapy, sex therapy, or couples therapy should be recommended when significant psychodynamic or relationship issues are evident (Plaut et al., 2004; Katz & Tabisel, 2004; Leiblum, 2000). Psychosexual or behavioral therapy are the first-line treatments of lifelong dyspareunia associated with vaginismus (Graziottin, 2006b; Graziottin & Brotto, 2004; Leiblum, 2000). They 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 (Graziottin, 2006b; Plaut et al., 2004). In this latter group, comorbidity with sexual aversion disorder should be investigated and treated.

Treatment of endometriosis can be medical or surgical. Use of continuous hormonal contraceptives (without the seven or five days of interruption) or of noretisterone acetate (2.5 milligrams per day continuously) is associated with a significant reduction of dyspareunia, dysmenorrhea, dyschezia (straining with stools), and nonmenstrual pain (Vercellini et al., 2003). Surgery of endometriosis

may be associated with reduction of coital pain in 60 percent of cases. However, approximately 25 percent do get worse after surgery (Crosignani, Olive, Berqqvist, & Luciano, 2006).

PSYCHOSEXUAL APPROACH

The health practitioner should aim at understanding and treating individual lifelong or acquired personal psychological problems or FSD or couple issues that may contribute to the complex multifactorial pathophysiology of sexual pain disorders (Graziottin, 2003a, 2006a). These include the following:

1. psychodynamic treatment in case of severe psychosexual problems, such as lifelong or acquired FSD or previous abuse, or of inadequate coping modalities;
2. short-term behavioral therapy (Graziottin, 2003a, 2006a, 2006b; Plaut et al., 2004), which is mandatory when vaginismus, erotic aversion, or sexual inhibition are present and when FSDs are secondary to sexual pain;
3. couples therapy, when relational factors are an issue in maintaining FSD after etiological sexual pain treatment—psychosexual support may be necessary to help the couple to (re)gain a satisfactory sexual intimacy and coital pleasure after months or years of frustration, disappointment, secondary FSD, and avoidance of intimacy (Plaut et al., 2004);
4. treatment of the partner when male factors (inclusive of an abusive/aggressive attitude) may contribute to the persistence of pain (Graziottin, 2003a, 2006b; Plaut et al., 2004).

REFERRAL RESOURCES

The multisystemic and multifactorial etiologies of sexual pain disorders require a professional multidisciplinary team. Appropriate referral is a key part of a successful treatment (Graziottin, 2006a). These professionals include medical practitioners with a special knowledge of sexuality (for sexual dysfunction in either partner), urologists or andrologists (when the male partner experiences erectile or ejaculatory problems that require medical intervention), oncologists (when hormonal treatment is considered for cancer survivors), psychiatrists (when depression and anxiety are associated with sexual dysfunction), sex therapists and counselors (to carry our psychosexual counseling), couples therapists (when relationship issues are primary contributors to the sexual dysfunction), individual psychotherapists (when personal psychodynamic issues are inhibiting sexual function), and physical therapists (when hyper- or hypotonicity of the pelvic floor is a contributing factor) (Plaut et al., 2004).

CONCLUSION

Pain is hardly ever psychogenic, and dyspareunia is no exception. Treatment of sexual pain disorders requires a comprehensive diagnosis of predisposing,

precipitating, and maintaining factors in their respective biological, psychosexual, and relational components. The biological component, historically the most neglected, needs to be fully appreciated in each patient with a careful history and competent physical exam as it is often key to complete sexual healing.

A multimodal approach is rewarding in terms of positive outcomes, thus offering women and couples the possibility of improving their sex lives.

NOTES

1. When pain is a symptom indicating impending or current damage to the body, it is a friend and is defined as "nociceptive." When pain becomes chronic, independent from the original etiology, as it is generated by the pain fibers and nerve centers themselves, it becomes an enemy and is called "neuropathic." Persistent chronic inflammation with overproduction of nerve growth factors and other neurotrophins by the hyperactive mast cell is one of the key factors contributing to the shift of pain from nociceptive to neuropathic.
2. See Wesselmann et al. (1997) for a review of the comorbidity between dyspareunia and anorectal and/or urologic symptoms. Unfortunately, these symptoms are often not included in the clinical history, resulting in ignorance of pelvic comorbidities.
3. Ganglion impar block is the anthalgic block of the ganglion of Walter, the last impar ganglion of the sympathetic chain. This ganglion is the main peripheral station where all the pain fibers from the genitals arrive to get to the medulla. With this local analgesia a progressive percentage of pain signals are blocked before they even enter the spinal cord.

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