

Dyspareunia: clinical approach in the perimenopause

Alessandra Graziottin MD

Director, Center of Gynaecology and Medical Sexology

H. San Raffaele Resnati, Milano, Italy

Abstract

Dyspareunia has long been considered to be psychogenic. Opposite, it has solid biological bases, that need to be addressed in a more integrated and patient-centered perspective. The new International Classification on Female Sexual Disorders allocates the etiologic subtypes of “Organic, Psychogenic, Mixed, Unknown” in the group of Sexual Pain Disorders, that includes Vaginismus, Dyspareunia and Non-coital Sexual Pain Disorders. According to this classification, this review analyzes the many different medical (“organic”) factors that may cause pain and/or sum-up with psychogenic (psychosexual) and relational factors contributing to pain during intercourse, with special attention to perimenopausal patients. Biological factors include hormonal, inflammatories, muscular, iatrogenic, neurologic, vascular, connective and immunitary causes. Psychosexual factors -vaginismus, loss of libido, arousal disorders and sexual pain related disorders- may frequently overlap. Couple problems may finally contribute to dyspareunia when they cause loss of libido and poor arousal, with consequent vaginal dryness. A preliminary clinical approach aiming at integrating different biological and psychosexual etiologies in a comprehensive perspective is discussed. It encourages a clinical update on the psychobiology of the experience of sexual pain, hopefully leading to more satisfying and effective treatment.

Introduction

The gynaecologist is increasingly asked to address his/her patients sexual complaints. This is mandatory when the woman complains of pain during intercourse, clinically known as “Dyspareunia”, from the ancient Greek, meaning “difficult mating”. No other physician has the same competence in evaluating all the potential biological causes of dyspareunia, particularly during and after the menopause, when endocrine, dystrophic and age correlated etiologies become prominent, as the woman ages (1).

According to the DSM IV (2), dyspareunia defines: “A) recurrent or persistent genital pain associated with intercourse; B) the disturbance causes considerable distress and interpersonal difficulties; C) the disturbance is not exclusively due to vaginismus or lack of lubrication, nor is it due to another disturbance on the Axis I, nor to the direct physiological effects of a substance or of a general medical condition.”

Unfortunately, the DSM IV classification arbitrarily eliminates the major biological factors of coital pain (3-7), that is mandatory to address if the symptom is to be etiologically cured. The exclusion of “a general medical condition” keeps out of the diagnostic field, among others: the menopause, with the sexual hormones loss that is the most frequent condition leading to vaginal dryness and coital pain in the middle age (3-5); diabetes, with the reduced lubrication secondary to the microangiopathy, and vascular diseases (atherosclerosis, secondary to hypercholesterolemia, smoking, age)(6); urological conditions increasingly associated with sexual disorders as the woman ages (7). Vascular diseases, in women, (as in men!), are still underdiagnosed causes of arousal disorders, one of the major physiopathologic contributors to vaginal dryness and sexual pain.

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The new International Consensus Conference on Female Sexual Disorders (FSD) (8), has changed the classification as follows: a general category of “Sexual Pain Disorders”, that includes Dyspareunia, Vaginismus and a new entry, “Non-coital Sexual Pain Disorders”, conceptualized as “recurrent or persistent genital pain induced by non coital sexual stimulation”. As in the previous DSM IV classification, sub-types of Lifelong vs Acquired and Generalized vs Situational are maintained, but again, a new entry helps to describe a comprehensive diagnosis. The third sub-type, “Etiologic origin”, includes four possibilities: “*Organic*, Psychogenic, Mixed, Unknown”, finally stressing the importance of biological factors and therefore encouraging a more balanced, integrated diagnostic approach between biologic and psychogenic causes (Tab 1).

Sexual comorbidity is frequently reported in women, well addressing both the physiologic interdependence of different aspects of the sexual response (desire, arousal, orgasm, satisfaction) *and* the interplay between biological, psychosexual and relational factors in female sexuality (1,8,9) (Fig.1)

Prevalence of dyspareunia

10-15% percent of fertile, coitally active women and up to 3-33% of perimenopausal women complain of various degrees of dyspareunia (1,9) This variation in clinical reporting depends on a number of factors: type of population considered (general vs gynaecological vs sexological clinics, specialized in the medical approach to female sexual disorders); selection biases; quality of attention the clinician pays to such a complaint and to its solution; more important, the quality of listening that creates or not the openness of the clinician to the disclosure and discussion of sexual complaints (1).

Communication on sexual matters

It's difficult to provide an effective intervention, if there is no mention of a problem! Female sexual disorders have been (and still are) the great absent(s) from the clinical consultation, both for the lack of a formal training in sexual medicine in most of the Medical Schools and for the uneasiness many physicians feel when dealing with an extremely sensitive matter (1,3,10,11). Many physician are also afraid of the “waist of time” that opening a sexual issue may imply. A dedicated time indeed, that can be rewarded with a higher satisfaction in the doctor-patient relationship, especially when the physician is able to give the appropriate answer to improve or, hopefully, solve the sexual problem he/she is listening to and caring of. The physician open to treat FSD should (1,4,10,12,13):

- 1) gradually learn to be comfortable in asking questions about sexuality and responding to issues that arise from the questioning;
- 2) be able to maintain a sensitive, non judgemental approach;
- 3) use precise questions within the limits imposed by tact, being aware that the older cohorts of women may feel uneasy or even unrespected with the open language that may be in play with the younger cohorts;
- 4) be sensitive to the optimal time to ask the most emotionally charged questions, maybe preferring open questions in the first visit (how do you feel? how are things at home? how's your sexual life? did you notice any change in your sexual interest since you are menopausal?..)
- 5) pay attention to the non verbal language (feeling tone, face mimics, posture...) that may be more informative about the emotional relevance of an issue than the simple meaning of words;
- 6) assure the patient of the complete confidentiality of the issues discussed;
- 7) update regularly, as the field of female sexuality is finally having an outburst of interest both in the basic and clinical research.

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With a serene matter-of-fact attitude the physician will help the patient relax and become matter-of-fact too (10).

Aetiology of dyspareunia in the perimenopause

Sexual pain may be perceived in the introital area (*introital or superficial dyspareunia*), at mid-vagina (*mid-vaginal dyspareunia*), that share many etiological factors and are clinically considered together, or deep in the pelvis (*deep dyspareunia*) (1,13). Aetiology may be *multifactorial* –biological, psychosexual and relational- and *multisystemic* (13-17), as different systems –endocrine, vascular, muscular, immunitary, neurologic and pain-related- may contribute to the final perception of sexual pain (1,18,19).

Focusing on *biological* etiologies, *introital and midvaginal dyspareunia* which worsen in the perimenopause may be caused by:

- a) an *hormonal aetiology* (1,3,4,15-17), which tops the list of factors (Tab.2) as vaginal dryness is related to progressive (in premenopause) or definitive loss (in postmenopause) of adequate concentration of vaginal estrogens. Important to be mentioned, vaginal estrogen concentration may be insufficient in spite of still present periods (3,17). Data from the Yale Midlife Study indicated that 77% of women reported loss of sex drive, 58% had vaginal dryness and 39% suffered from dyspareunia (17). This study also showed a relationship between serum estradiol level and sex problem. With an arbitrary cut-off at 50 pg/ml, significantly more menopausal women reported problems of vaginal dryness, dyspareunia and pain compared with women whose estradiol levels were above 50 pg/ml (17). Different estrogenic receptor distribution may account for a different tissue sensitivity and different symptoms vulnerability in menopausal women (18). Topical estrogens, to improve vaginal dryness, may be necessary in different clinical conditions: when the patient is prescribed extra-low contraceptive pills (with 15 micrograms of etynil-estradiol) in the premenopause, as on average 18-22% of this patient may complain of vaginal dryness during this treatment; when she is on low dose HRT (Tab.3); when she is menopausal and not on HRT.
- b) *vaginitis* and *vulvitis* may be a frequent cofactor of acquired dyspareunia also in the perimenopausal years (19-24). The routine examination of *vaginal pH*, normally under 4.5, that usually rises in hypoestrogenic conditions, may immediately give a biologic feedback about the vaginal environment and related ecosystem (17,24). Gardnerella infections are more likely at pH above 5, whilst saprophytic pathogens from colonic origin (*Escherichia Coli*, *Enterococcus foecalis*) are usually in play at even higher pH level, when the vagina is deprived of the majority of the Lactobacilli due to the loss of estrogens and increased pH (24). When past Vulvar Vestibulitis Syndrome (VVS) -the most frequent biological aetiology of dyspareunia in the fertile age - is reported (1,19,20) special attention should be paid to diagnose potential recurrences, especially when sexually active women are on HRT (1,19). Candida infection may as well be exacerbated when the woman starts HRT (1).
- c) *vulvar dystrophy*, which is increasingly credited to be a full thickness disorder, with a progressive reduction of all the tissue components, not limited to the epiphenomenon of the mucocutaneous lichen sclerosus (25), probably on a genetic vulnerability accelerating hormone receptor loss. Tarcan et al (26) proved that there is an age dependent reduction of more than 50% of the cavernosal smooth muscle components from the first to the sixth decade of life, this being the basis of the increasingly reported difficulty in getting genitally aroused (“my clitoris is dead”) up to orgasm.
- d) *iatrogenic* factors, that may be responsible for an acquired dyspareunia in the perimenopause: overzealous colporrhaphies and other pelvic surgeries (27-29) may be responsible for an anatomic narrowing of the introital area that may be incompatible with the intercourse. Questions about ongoing sexual activity should be asked *before* every type of pelvic surgery, even in very old patients, to prevent further comorbidity due to the wrong assumption

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that the lady was too old for having sex. Radiotherapy and or radical surgery for cervical cancer may as well cause a reduced vaginal receptivity because of the shortening and the retraction of the vagina (29). With the exception of adenocarcinoma of the cervix (which are hormone dependent) there is no contraindication in treating patients cured of a cervical squamous carcinoma with topical estrogens, soon after surgery, more so if radiotherapy is prescribed (29). This will reduce the vaginal retraction, maintain a better mucosal and vascular trophism and, if combined with a timely rehabilitation of the pelvic floor, will help to maintain coital receptivity in these unfortunate women. Last but not least, as postmenopausal and elderly women are usually on multipharmacologic treatment, sexual side-effects of drugs contributing to dyspareunia through libido and arousal disorders should be considered (30)

- e) *muscular factor* – namely a tighten of the pelvic floor- which may become relevant in chronic dyspareunia, when the persistence of pain may cause a secondary defensive contraction of the levator ani, till a frank myalgia (1, 31-33). This may become an added cause of mid-vaginal dyspareunia and/or of *post-coital cystitis (1)*: pain is the most powerful reflex inhibitors of the perivaginal (and periurethral) arousal (1,19), increasing the vulnerability of the urethra and of the trigonal area, deprived of the protective effect of estrogen, to the further mechanical trauma of an intercourse without lubrication and without the protective vascular congestion of the periurethral vessel, which have a cavernosal structure in the lower third (34) .
- f) *vascular factors, like smoking, atherosclerosis, hypertension and dismetabolic disorders like diabetes with microangiopathy (and neuropathy)* that may all contribute to genital arousal disorders (1,6,) -in women as well as in men-, with vaginal dryness leading to dyspareunia.
- g) *pain related disorders*: most recent and exciting findings in dyspareunia, when caused by chronic VVS, indicate a specific pathology within the pain system, with an increased pain input from the introital area, with an histologically proven increase in pain fibers (35-37), and a systemic lowering of the pain threshold (38). This addresses the shift from *nociceptive pain*, when pain “simply” indicates an ongoing damage with tissue inflammation, to *neuropathic pain*, when pain is “produced” and/or exacerbated within the pain system (39-41).
- h) least frequent biological factors are *neurologic diseases* including pudendal nerve entrapment syndrome (28,42,43), leading to dyspareunia, which may appear many years after coccygeal-sacral physical trauma or pelvic surgery; the *Sjogren's Syndrome (44)*, an autoimmunitary disease where antibodies attack components of exocrine gland system; *physical genital trauma and/or sexual abuse (45)*, infrequent but very traumatic in dystrophic genital condition.

Deep dyspareunia may be more frequently caused by:

- a) *pelvic endometriosis (46)*, deep dyspareunia being the emerging symptom, recurring in case of cyclic HRT. In women with previous endometriosis, and with conserved uterus, low-dose continuous combined HRT should be the first choice to avoid bleeding and recurrence of pain;
- b) *pelvic inflammatory disease (PID) (47)* which may cause lateral, deep pelvic pain. Once considered a disease of youth , PID is now to be thought of as modified sex habits may expose women of all ages to PID when they have non protected sex with new partners.
- c) *pelvic varicocele (1)*, which is increasingly diagnosed as cause of deep dyspareunia.
- d) *levator ani myalgia*: deep pelvic pain may also be secondary to trigger points stimulation at the levator ani level, when the muscle is intensely myalgic (1,31-33).

How to diagnose dyspareunia

When a woman seeks clinical help because of coital pain during intercourse in the perimenopause, the clinical approach should start with a few general questions (TAB.4) helping to focus on the leading sexual complaint and sexual co-

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morbidity. In this paper, questions and answers will be presented in greater detail, to offer a meaningful clinical reasoning to be used when the patient complains of dyspareunia. A very careful clinical examination will complete the first level diagnostic evaluation.

Clinical interview

- 1) *When did you notice that intercourse was becoming painful? Did you always (“lifelong”) suffer from pain during intercourse, or is pain recent (“acquired“)?*

When lifelong, dyspareunia is usually caused by vaginismus (1) and/or coexisting, life-long female sexual disorders, like low libido (1, 4,8,11-13) and arousal disorders (13,15,16) of psychosexual aetiology. Lifelong dyspareunia may worsen till a frank avoidance of sex around the menopause because of the hormonal loss. If pain prevents penetration, a severe vaginismus is probably revealed (1), occasionally during the first gynaecologic visit around the menopause, when a menorrhagia or other acute symptoms make it necessary to be visited.

- 2) *If pain is recent (“acquired“), do you also suffer from vaginal dryness during intercourse? And/or from vaginitis? Do you suffer from cystitis-like symptoms, 24 to 72 hours after intercourse?*

Hormonal loss (3-5,13-19), altered vaginal ecosystem (24) and a tighten pelvic floor (1,31-33) may concur to the clinical picture. All factors should be addressed for a full recovery.

- 3) *Do you always feel pain during intercourse (general) or is it limited to some situations (situational)? In the latter case, which condition or situation precipitate it? In case of stable relationship, how is the quality of intimacy and couple satisfaction, besides sex? Does the partner suffer of any sexual problem: low libido, premature ejaculation, erectile deficit? Situational dyspareunia should focus the interview on the quality of the relationship (48,49), frustrated intimacy needs (50) and love-sickness, potential conflicts (51), areas of disappointment, anger (49) and/or context-dependent worsening and/or precipitating factors. It also should open a window on partner's sexuality (48), and on the potential collusion dynamics that may contribute to maintain the symptom. Dennerstein et Al (48), in their 8 years prospective studies on women during the menopausal transition, found that “feelings for partner” and “partner's health and sexual problems” were the strongest predictors of sexual changes across the menopause, whilst the two symptoms more frequently recorded were loss of libido and arousal disorders. “Symptom inducer and symptom carrier” scripts should also be gently explored (49, 51).*

- 4) *If you avoid intercourse, is your sexual experience still pleasurable and satisfying or not? How is your sexual drive? Is arousal easy? Do you usually get clitoral orgasm? Do you notice disappearance of lubrication when you try intercourse? These questions help to focus on the quality of sexual response, besides intercourse, and of intervening negative feedbacks on the sexual circuit (Fig.1). The presence of a dysfunctional sexual response, leading to sexual co-morbidity, may suggest a mixed aetiology, if organic factors are present as well. Worsening of the sexual response, after the onset of dyspareunia, addresses specifically the potent inhibitory effect of fear of pain on physical arousal (1,19,29).*

- 5) *How intense is the pain you feel? Focusing on the intensity and characteristics of pain is a relatively new approach in addressing dyspareunia issues (1,19,20,38). Analogic scales, or other traditional quantitative or qualitative measures of pain and pain disorders appear to be very useful for description and classification, particularly when vulvar vestibulitis is the leading aetiology of dyspareunia, as increasing input of pain signals (35-37,) and a lowered central pain threshold are increasingly recognized as specific aspect of this disease.*

- 6) *When do you feel pain? Before, during or after intercourse? Pain before intercourse suggests a phobic attitude toward penetration and/or the presence of a vestibulitis, if previous tentatives of intercourse have caused a mechanic micro trauma of the introital mucosa (1,19). Pain during intercourse is more frequently reported*

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(20,52,53). This information, combined with the following -"where does it hurt?"- proves to be the most predictive of the organicity of pain (53) Pain *after* intercourse: if introital, again it suggests vestibulitis (in the fertile women or in post menopausal women on HRT) because of the postcoital worsening irritation, hypertonic pelvic floor (the so called levator ani myalgia) and or vulvovaginal dystrophy in the peri-post-menopause (1,19).

- 7) *Where does it hurt? At the beginning of the vagina, in the mid vagina or deep in the vagina?* Meana et al (52) observed that *location of the pain* and *its onset* within an episode of intercourse were the strongest predictors of presence and type of organicity. Psychosocial variables such as situational factors, relationship adjustment and history of sexual abuse had no predictive value and women with dyspareunia did not differ significantly from controls on these measures. (52,53) A different diagnostic - and cultural - approach, namely to consider dyspareunia as a *sexual pain disorder*, and not "simply" a sexual disorder, is necessary to correctly address the many different issues covered by this comprehensive and yet elusive word (38,52).

Physical examination: inspection

Observation of the patient while she is waiting for the visit, the degree of her muscular tension, the presence of neurovegetative signs, avoidant or contracted postural attitudes should all be observed and reported in the clinical chart (1). Careful inspection of the patient and of her external genitalia may help to focus the diagnosis in case of superficial dyspareunia:

- a) neurovegetative response (sweating, blushing, tachycardia...) suggestive of phobic attitude to coitus and often associated to vaginismus (1);
- b) defensive general posture, suggesting phobic aversion to coitus and/or long lasting persisting pain; muscular postural changes that may become secondary sources of pain; (19, 31-33)
- c) defensive contraction of the perineal muscle, with reduced distance fourchette-anus (1); signs of inflammation, suggestive of vulvovaginitis 7,19 or vestibulitis (19-23) Three signs will suggest the latter: variable reddening of the vestibular area (the one between clitoris, inner face of labia minora and fourchette); acute pain when touching at 5 and 7, looking at the introitus as a clock face, and defensive contraction of the perineal muscle at tentative insertion (mimicking what happens at intercourse) (1,19,52-54);
- d) signs of vulvovaginal dystrophy in peri-post menopausal women (1,3,19,25)
- e) retracted, painful scars of previous pelvic surgeries (27-29); scars of vulvar traumas;
- f) congested clitoris, in case of associated clitoralgia (19);
- g) Bartholinitis; painful surgical outcome of surgery on Bartholin glands (27); inflamed Nuck's cyst (rare);
- i) haemorrhoids, suggestive of colonic and/or pelvic floor dysfunction (27) that may be associated to repeated vaginitis and/or cystitis from colonic saprophytes (24), frequently associated to dyspareunia in the post-menopause;

Physical examination: gynaecologic examination with semiology of pain

Accurate, delicate and respectful physical examination may indicate one or more specifically painful points, mimicking the pain the woman experiences during intercourse, in 90% of cases (52,54). "*Where does it hurt?*": this is the critical question. While gently and competently exploring all the possible sources of pain, the physician will not only build-up a careful "*pain map*" (1), based on solid anatomy and logic physiopathology. He/she will also make a gesture of attention that is of outmost psychological value. After months or years of being told that "pain is only in her mind" such a careful evaluation will create a very positive and trustful doctor-patient relationship (1). If the partner is present in the office, and if the woman agrees to that, explaining to him (or her) all the physical findings that might cause pain during the

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exploration (and likely so, during penetration), is useful and positive also for the couple relationship. When the partner sees that there is a physical problem, with a medical diagnosis and a reasonably effective treatment, and that “she is not just refusing sex and/or him”, he/she becomes much more understanding and collaborative.

The visit might reveal and/or confirm:

- a) acute pain at 5 and 7 (vestibulitis) (Q tip test) (19, 20,29,52-54)
- b) tender and/or trigger points on retracted scars, and/or on superficial perineal muscles (mostly on the bulbocavernosus muscle) (1,31-33);
- c) congested, painful clitoris in case of associated clitoralgia (19);
- d) a narrow introitus after surgery (colporrhaphy, colpoperineorrhaphy) (27-29): retraction, pain, mucocutaneous and myofascial trigger points, vaginal anatomic outcome (introital calibre, vaginal length and post-repair elasticity), and pelvic floor muscles' defensive hypertonus are frequent cofactors in introital and mid-vaginal dyspareunia after perineal surgery. (31-33)
- e) a dry, dystrophic vagina (13,15-18);
- f) spasm of the perivaginal muscles, with tender and/or trigger points in the mid-vagina, at the insertion of the levator ani on the spine, usually different in the two halves of the muscle (1,31-33). Trigger points on the levator ani may cause referred deep pelvic pain and mimic deep dyspareunia. Sometimes the muscle presents a strong asymmetric spasm, suggestive of both local muscle pathologies and/or asymmetric spasms secondary to pelvic traumas (1). Posterior palpation of the sacrum and coccyges *per vaginam* may confirm the presence of selective contractions or tension causing acute pain when touched.
- g) acute provoked pain at bimanual deep exploration. Posterior pain when causing tension to the uterosacral ligaments may be suggestive of endometriosis (1,46), of which deep dyspareunia may be the emerging symptom, recurring in case of cyclic HRT. In women with previous endometrioses, and with conserved uterus, low-dose continuous combined HRT should be the first choice to avoid bleeding; lateral, deep pelvic pain may be more frequently caused by PID (47) ; sometimes by pelvic varicocele (55); anterior pain, in the trigonal area, is more frequently present when dyspareunia is associated with post-coital cystitis, urgency and frequency (1). As mentioned before, deep pelvic pain may also be secondary to trigger points stimulation at the levator ani level (31-33).

Clinical interview and competent physical examination will contribute to an accurate etiologic diagnosis: organic, psychogenic, mixed or unknown. This latter group will be dramatically reduced if the classical medical steps in approaching pain disorders are respected and if a competent attitude to diagnose FSD is integrated with them.

Treatment

An integrated diagnosis between medical and psychosexual factors is preliminary to the effective treatment of dyspareunia, that requires physiopathologically oriented therapy of the organic factors and adequate direction of individual and couple psychosexual issues.

Medical treatment of dyspareunia includes:

- topical hormonal treatment, which are the first choice to reduce pain caused by inadequate hormonal dependent genital arousal, particularly during and after the menopause :
 - 1) testosterone propionate powder 2% in vaseline jelly is to be applied to the external genitalia, daily or every other day, in very small quantity. This local treatment is effective in increasing the clitoral arousal, sensitivity and responsiveness. The same therapy applied to the vulva will relieve entry dyspareunia due to vulvar dystrophy. Testosterone is avidly absorbed from the peripheral tissues and if used frequently or for prolonged

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periods should be monitored by regular testosterone assays, to avoid supraphysiologic plasmatic levels and related side-effects (3);

2) vaginal estrogens may offer a rapid relief when dyspareunia is more dependent on vaginal dystrophy (1,13,15-18). Topical estrogens may be the first line treatment in women who cannot or do not want a systemic HRT. They should be prescribed, even in the premenopause or during systemic HRT when symptoms and/or signs of vaginal dystrophy are present (24). 17 beta estradiol vaginal tablet may significantly improve atrophic vaginitis and related symptoms with better compliance profile that the more powerful conjugated equine estrogen vaginal cream (56);

- systemic HRT is indicated in case of sexual co-morbidity (loss of libido and poor central arousal concomitant to the menopause) (1,5, 12, 15,16, 57-60) and/or when other menopausal symptoms are complained of (3,5,17,29,48). Estroandrogens are the treatment of choice when sexual comorbidity is reported, either in form of injection or subcutaneous implant (5,57-59). Testosterone patches are still being investigated (61). Among the available oral treatments, tibolone, thanks to its androgenic action, seems to offer a very positive effect both on libido and central arousal and on peripheral genital arousal and orgasm, thus improving different dimensions of the sexual response and easing the sexual recovery when other organic etiologies of dyspareunia have been appropriately treated (60). Progestins with androgenic properties like noretisterone do again better, on the sexual response, than the non androgenic ones. In tailoring treatment, the wish of the woman to maintain a good sexuality, or improving a fading one, should be considered for the optimal HRT choice. Two recent papers, on either tibolone (62) and oral HRT containing noretisterone (NETA) (63), suggest a significant positive effect on muscle trophism and motor competence. This impact could be beneficial also from the sexual point of view, both for the increased general well being, that improves the vital energy and the sense of fitness, and specifically for the trophism of the pelvic floor, so important in the female sexual function, although specific studies on this special aspects have not been performed so far. Systemic and local HRT is to be considered to reduce dyspareunia and improve the quality of life and overall sexuality of patients treated for genital cancer , with the exception of adenocarcinoma of the endometrium and of the cervix (29, 64);
- rehabilitation of the pelvic floor muscles, defensively contracted in chronic coital pain, is of specific importance in the treatment of dyspareunia (1,65,66). Stretching and relaxation of contracted muscles, self massage with medicated oil (Saint John's wart) (1) or electromyographic biofeedback (65-66), when available, may all relieve the muscle tension, reducing mid-vaginal dyspareunia and limiting the source of referred pain. In a prospective controlled study (54) cognitive behavioural therapy, electromyographic biofeedback and vestibulectomy all proved to offer significant improvement in dyspareunia associated with VVS, by modifying different etiological factors concurring to pain. Special attention to systemic muscular involvement as part of general defensive reaction should be paid when dyspareunia is associated to vaginismus (67);
- electroanalgesia is to be recommended when introital hyperalgesia is due to chronic Vulvar vestibulitis syndrome (VVS) (63). Vestibulectomy, in fashion when the physiopathology of introital pain was not well understood, in being abandoned in favour of more conservative antalgic treatments (39,65-66);
- antalgic treatment (39, 68), with systemic and local treatments is to be reserved to severe dyspareunia, usually associated with VVS or neurologic pain, when all previous treatment have failed. Systemic treatment may include tricyclics, like amitriptyline (68), aimed at modulating the serotonin and epinephrine imbalance associated with persisting pain, and anticonvulsants, like gabapentin (39), aimed at raising the threshold for the amount of stimuli needed for nerves to fire, thus raising the central pain threshold. Presacral anaesthetic block of the ganglion impar

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(every 3 wks up to four months) has recently been proposed (39) as an effective conservative second line treatment when all previous treatment have failed;

- coexistent general medical conditions (vascular, dismetabolic, neurologic, immunitary) should be addressed as well to reduce the multisystemic aetiology of dyspareunia (1).

Conclusions

Pain is rarely purely psychogenic. Dyspareunia makes no exception. As all the pain syndromes, usually it has one or multiple biological etiologic factors. It deserves careful clinical attention, as it is the common emerging symptom of a variety of medical conditions that should be recognized and treated accordingly. Psychosexual factors, mostly low libido, lifelong or acquired, because of the persisting pain, and arousal disorders, as well as life-long or secondary to the inhibitory effect of pain, should be addressed in parallel, in order to give a comprehensive, integrated and more effective treatment. Psychodynamic issues, both personal and/or related to couple relationships should be adequately addressed if present.

Interdisciplinary approach, with the contribution of different experts – gynaecologist, sexologist, psychiatrist, pain therapist – is needed to give dyspareunia its full meaning, in respect of the individual complexity of sex pain experience. Gynaecologists should enrich their clinical competence with a more active knowledge of the biological basis of the female sexual disorders that are rooted in their specialistic domain, with an increasing competence in the physiopathology of sexual -and pelvic- pain. They are indeed the physicians more likely to offer the first line best comprehensive diagnosis and treatment of sexual pain disorders.

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DRAFT COPY – PERSONAL USE ONLY**TABLE 1. Classification of Sexual Pain Disorders**

Dyspareunia: the recurrent or persistent genital pain associated with sexual intercourse.

Vaginismus: the recurrent or persistent involuntary spasm of the musculature of the outer third of the vagina that interferes with vaginal penetration, which causes personal distress.

Non coital sexual pain disorders: recurrent or persistent genital pain induced by non coital sexual stimulation

Subtyping further differentiates the diagnosis of FSD according to:

- a) the temporal onset: lifelong versus acquired
- b) the context-dependent dynamic: generalized versus situational
- c) the aetiology: organic, psychogenic, mixed, unknown

Adapted from Basson et Al. 2000 (8)

TABLE 2. Short form of a sexual history in perimenopause

- How do you feel?
- How's your sex life? Is it satisfying for you?
- If your sex life is dissatisfying, what's the main complaint you have?
- Do you feel a loss of sex drive (are you still interested in sex)?
- Do you feel pain during or after the intercourse?
- Do you feel an arousal/lubrication difficulty with vaginal dryness?
- Do you have increasing difficulties in getting orgasm?
- If you have one or more of these disorders, did they appear or worsen after the menopause (or were they lifelong)?
- Do you have a stable relationship?
- If yes, how's your couple relationship? And your partner general and sexual health?
- Do you feel that your sexual problem is more dependent on physical or couple problem?
- Are you personally interested in improving your sex life?

TABLE 3. Comparison of biological activity of different estrogens used on ERT

ESTROGEN DOSING	LOW	INTERMEDIATE	HIGH
17beta estradiol	0.5 mg	1 mg	2 mg
Esterified Estrogens	0.3 mg	0,625 mg	1,25 mg
CEE	0,3 mg	0,625 mg	0,9 mg
E2 - patch	25 mcg	50 mcg	100mcg

TABLE 4. Leading biological aetiology of dyspareunia in perimenopause

INTROITAL & MIDVAGINAL	DEEP
- sex hormone loss	- endometriosis
- vulvovaginitis and vestibulitis	- PID
- vulvar dystrophy	- pelvic varicocele
- iatrogenic	- referred pain
- muscular	
- vascular	
- neurologic	
- connective/immunitary	
- traumatic	
- pain disorders	

FIG. 1 Impact of dyspareunia on different dimensions of the sexual response

This circular model, formulated by the presenting author, 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 physiopathologic point of view;
- 2) potential negative or positive feedback mechanisms operating in sexual function;
- 3) the *direct* inhibiting effect of dyspareunia on genital arousal and vaginal receptivity and the *indirect* inhibiting effect on orgasm, satisfaction and libido, with close interplay between biological and psychosexual factors.

