Female sexual dysfunction

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ASSESSMENT

Women's sexuality has only recently emerged as a central concern after years of neglect in the medical world. The current challenge is to blend together the biological, psychosexual and context-related components of women's sexual response in a comprehensive and meaningful scenario (Basson et al 2000, 2004). In this perspective, the role of pelvic floor function and dysfunction is of the highest importance (Alvarez & Rockwell 2002, Bourcier et al 2004, Graziottin 2001a, 2005a).

Levator ani's tone, strength and performance is a major contributor to vaginal receptivity, vaginal responsiveness, coital competence and pleasure (for both partners), and for the orgasmic muscular response. Indirectly, pelvic floor disorders (PFD) may impair genital arousal and, through a negative feedback, may affect the potential for physical and emotional satisfaction, and for sexual desire and mental arousal, thus potentially affecting the whole of a woman's sexual response, particularly when coital pain is a disruptive factor (Fig. 9.26) (Graziottin 2000, 2001a, 2004a).

Hyperactivity of the pelvic floor is causally associated with sexual pain disorders, namely dyspareunia and vaginismus (Abramov et al 1994, Glazer et al 1995, Graziottin et al 2004a, 2005a, Harlow et al 2001; Harlow & Stewart 2003, Lamont 1978, McKay et al 2001) and overexertion of the pelvic floor muscles (PFM) may lead to myalgia and 'Kegel' dyspareunia (DeLancey et al 1993).

The pelvic floor is central in understanding how physiological events such as vaginal deliveries may modulate levator ani's sexual competence in a life span perspective (Baessler & Schuessler 2004, Glazener 1997). Pelvic floor disorders are a common denominator in urogenital, proctological and sexual comorbidities (Barlow et al 1997, Cardozo et al 1998, Graziottin et al 2001a, 2004a, Lauman et al 1999, Weiss 2001, Wesselmann et al 1997). Iatrogenic problems, consequent to urogenital surgery, may in parallel affect and impair both a woman's well-being and sexual response (Graziottin 2001b).

Last, but not least, new insights into the role of the hyperactivity of the pelvic floor in adolescence and, possibly, infanthood, as predictors of vulnerability to further sexual pain disorders (vaginismus and dyspareunia) and to vulvar vestibulitis/vulvodynia open a new preventive window for female sexual dysfunctions (FSD) (Chiozza & Graziottin 2004, Graziottin 2005a, Harlow et al 2001). Appropriate management of early hyperactivity of the pelvic floor could hopefully prevent the urogenital and sexual comorbidities that affect so many young lives.

In this book, dedicated to physical therapy for the pelvic floor, FSD is reviewed paying special attention to the genital components of women's sexual response in physiological and pathological conditions. However, the role of the biological and medical factors should always be considered in the appropriate psychosexual and sociocultural context.

THE COMPLEXITY OF WOMEN'S SEXUALITY

Women's sexuality is multifactorial, rooted in biological, psychosexual and context-related factors (Basson et al 2000, 2004, Binik et al 2002, Dennerstein 2004, Dennerstein et al 1999, Levin 2002, Graziottin 2004a, 2004b, Leiblum & Rosen 2000, Klausmann 2002, Plaut et al 2004, Segraves & Balon 2003), correlated to couple dynamics and family and sociocultural issues. It is multisystemic: in men and women, a physiologic response requires the integrity of the hormonal, vascular, nervous, muscular, connective and immune systems; this fact has been too often overlooked in women until recently (Bachmann et al 2002, Goldstein & Berman 1998, Graziottin 2000, 2004b, Graziottin & Brotto 2004, Levin 2002, Meston & Frolich 2000, O'Connell et al 1998, 2004, Pfaus & Everitt 1995).

Three major dimensions – female sexual identity, sexual function and sexual relationship – interact to give women's sexual health its full meaning or its problematic profile (Graziottin 2000, 2004a, Graziottin & Basson 2004). Women's sexuality is discontinuous throughout

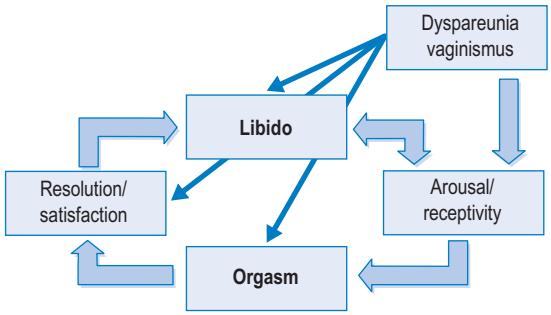

Fig. 9.26 Circular model of female sexual function and the interfering role of sexual pain disorders. This model contributes to the understanding of frequent overlapping of sexual symptoms reported in clinical practice (comorbidity) because different dimensions of sexual response are correlated from a pathophysiological point of view. Potential negative or positive feedback mechanisms operate in sexual function: dyspareunia and/or vaginismus has a direct inhibiting effect on genital arousal and vaginal receptivity and may have an indirect inhibiting effect on orgasm, satisfaction and libido, with close interplay between biological and psychosexual factors. Pelvic floor disorders of the hyperactive type causally related to sexual pain disorders may rapidly affect the sexual response. Modified from Graziottin 2000, with permission.

the life cycle and is dependent on biological (reproductive events) as well as personal, current contextual and relationship variables (Basson et al 2000, 2004).

FSD is age related, progressive and highly prevalent, affecting up to 20–43% of premenopausal women (Lauman et al 1999), and 48% of older women who are still sexually active in the late postmenopause (Dennerstein et al 2003, 2007, Graziottin & Koochaki 2003).

FSD may occur along a continuum from dissatisfaction (with potential integrity of the physiological response but emotional/affective frustration) to dysfunction (with or without pathological modifications), to severe pathology (Basson et al 2000, 2004). Pelvic floor disorders are among the most important and yet neglected medical contributors to FSD (Graziottin 2001a, 2005a, 2005b). However, sexual dissatisfaction, disinterest and even dysfunction may be appropriate for an 'antisexual' context (e.g. a partner affected by male sexual disorder or abusive) and they should not be labelled per se as 'diseases' or dysfunctions requiring medical treatment (Bancroft et al 2003).

FSD may occur with or without significant personal (and interpersonal) distress (Bancroft et al 2003, Graziottin & Koochaki 2003). Sociocultural factors may further modulate the perception, expression and complaining modality (i.e. the 'wording') of a sexual disorder. The meaning of sexual intimacy is a strong modulator of the sexual response and of the quality of satisfaction a woman experiences besides the simple adequacy of the physical response (Basson 2003, Kaplan 1979, Klausmann 2002, Levine 2003, Plaut et al 2004). The quality of feelings for the partner and the partner's health and sexual problems may further contribute to FSD (Dennerstein et al 2003, Dennerstein 2004). Sexual problems reported by women are not discrete and often co-occur, comorbidity being one of the leading characteristics of FSD (Basson et al 2000, 2004).

Co-morbidity between FSD and medical conditions (e.g. urological, gynaecological proctological, metabolic, cardiovascular and neurological) is increasingly recognized (Graziottin 2000, 2004a, 2004b, 2005b, Wesselmann et al 1997). For example, latent classes analysis of sexual dysfunctions by risk factors in women indicate that urinary tract symptoms have a RR = 4.02 (2.75–5.89) of being associated with arousal disorders and a RR = 7.61 (4.06–14.26) of being associated with sexual pain disorders according to the epidemiological survey of Laumann et al (1999), credited as being the best survey carried out so far. The attention dedicated to pelvic floor related comorbidities – both between FSD and between FSD and medical conditions – in this paper reflects the clinical relevance of this association, especially in the urogynaecological and proctological domain.

CLASSIFICATION OF FSD

Over the past decades, the classification of FSD has undergone intense scrutiny and revisions that mirrors the new understanding of its complex aetiology. Until a decade ago, the classification of FSD, which constitutes the frame of reference for an appropriate diagnosis, was focused almost entirely on its psychological and relational components. Indeed, FSD was included in the broader manual of 'psychiatric' disorders (American Psychiatric Association 1987, 2000). The first and second consensus conferences on FSD (Basson et al 2000, 2004) set out to define FSD with special attention to bringing together the current level of evidence with definitions to fit women's wording and experiences. The latest classification is shown in Box 9.3.

CLINICAL HISTORY

For a more accurate definition of the sexual symptoms, health care providers should also briefly investigate the

Box 9.3: Classification of female sexual disorders (From Basson et al 2004)

WOMEN'S SEXUAL INTEREST/DESIRE DISORDER
- Absent or diminished feelings of sexual interest or desire, absent sexual thoughts or fantasies and a lack of responsive desire. Motivations (here defined as reasons/incentives), for attempting to become sexually aroused are scarce or absent. The lack of interest is considered to be more than that due to a normative lessening with the life cycle and duration of a relationship.

SEXUAL AVERSION DISORDER
- Extreme anxiety and/or disgust at the anticipation of/ or attempt to have any sexual activity

SUBJECTIVE SEXUAL AROUSAL DISORDER
- Absence of or markedly diminished cognitive sexual arousal and sexual pleasure from any type of sexual stimulation. Vaginal lubrication or other signs of physical response still occur.

GENITAL SEXUAL AROUSAL DISORDER
- Complaints of absent or impaired genital sexual arousal. Self-report may include minimal vulvar swelling or vaginal lubrication from any type of sexual stimulation and reduced sexual sensations from caressing genitalia. Subjective sexual excitement still occurs from non-genital sexual stimuli.

COMBINED GENITAL AND SUBJECTIVE AROUSAL DISORDER
- Absence of or markedly diminished subjective sexual excitement and awareness of sexual pleasure from any type of sexual stimulation as well as complaints of absent or impaired genital sexual arousal (vulvar swelling, lubrication).

PERSISTENT SEXUAL AROUSAL DISORDER
- Spontaneous, intrusive and unwanted genital arousal (e.g. tingling, throbbing, pulsating) in the absence of sexual interest and desire. Any awareness of subjective arousal is typically but not invariably unpleasant. The arousal is unrelieved by one or more orgasms and the feelings of arousal persist for hours or days.

WOMEN'S ORGASMIC DISORDER
- Despite the self-report of high sexual arousal/ excitement, there is either lack of orgasm, markedly diminished intensity of orgasmic sensations or marked delay of orgasm from any kind of stimulation.

DYSPAREUNIA
- Persistent or recurrent pain with attempted or complete vaginal entry and/or penile vaginal intercourse.

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

so-called 'descriptors' of the disorders, as defined by the International Consensus Conferences held in 1998 and 2003 (Basson et al 2000, 2004). They include the following.

The aetiology of the disorder

The aetiology of the disorder is further detailed in predisposing, precipitating and maintaining factors (Box 9.4) (Graziottin 2003a, 2003b, Graziottin & Brotto 2004, Graziottin & Leiblum 2005). Each category includes biological, psychosexual and contextual causes.

Biological descriptors include hormonal dysfunctions, PFDs, cardiovascular problems, neurological conditions (particularly pain-related) (Binik 2005, Binik et al 2002), metabolic disorders (diabetes mellitus), affective disorders (depression and anxiety). All the medical conditions that may directly or indirectly affect sexuality through their multisystemic impact and/or the consequences of pharmacological, surgical and/or radiotherapy treatment should be considered in the differential diagnosis of potential contributors to the reported FSD. Loss of sexual hormones, consequent to natural or iatrogenic menopause is a major contributor to FSD (Dennerstein et al 2003, 2005). It can be addressed with appropriate hormonal replacement therapy (Bachmann et al 2002, Graziottin 2000, 2004, Graziottin & Basson 2004). Current medication use and substance abuse should be actively investigated (Segraves & Balon 2003).

Psychosexual descriptors refer to emotional/affective/psychic factors such as negative upbringing/ losses/trauma (physical, sexual, emotional) (Basson 2003, Edwards et al 1997, Rellini & Meston 2004), body

Box 9.4: Factors contributing to female sexual dysfunction (modified from Graziottin & Leiblum 2005)

PREDISPOSING FACTORS

Biological

- Endocrine disorders (hypoandrogenism, hypoestrogenism, hyperprolactinaemia)
- Menstrual cycle disorders/premenstrual syndrome
- Recurrent vulvovaginitis and/or cystitis
- Pelvic floor disorders: lifelong or acquired
- Drug treatments affecting hormones or menstrual cycle
- Contraceptive methods inappropriate for the woman and couple in that period of life
- Chronic diseases (diabetes mellitus, cardiovascular, neurological or psychiatric disease etc)
- Disorders associated with premature ovarian failure (POF): genetic, autoimmune
- Benign diseases (e.g. endometriosis) predisposing to iatrogenic menopause and dyspareunia
- Iatrogenic menopause: bilateral oophorectomy, chemotherapy, radiotherapy
- Persistent residual conditions (e.g. dyspareunia/ chronic pain associated with endometriosis)

Psychosexual

- Inadequate/delayed psychosexual development
- Borderline personality traits
- Previous negative sexual experiences: sexual coercion, violence, or abuse
- Body image issues/concerns
- Affective disorders (dysthymia, depression, anxiety)
- Inadequate coping strategies
- Inadequate sexual education (attitudes towards contraception and sexually transmitted diseases)
- Dissatisfaction with social/professional role(s)

Contextual

- Ethnic/religious/cultural messages, expectations, and constraints regarding sexuality
- Social ambivalences towards female sexuality, when separated from reproduction or marriage
- Negative social attitudes towards female contraception
- Low socioeconomic status/reduced access to medical care and facilities
- Support network

PRECIPITATING FACTORS

Biological

- Negative reproductive events (unwanted pregnancies, abortion, traumatic delivery with damage of the pelvic floor, child's problems, infertility)
- Postpartum depression
- Vulvovaginitis/sexually transmitted diseases
- Sexual pain disorders
- Age at menopause
  - premature ovarian failure (POF) – menopause before age 40
  - premature menopause – menopause between age 40 and 45
- Biological vs iatrogenic menopause (especially for premature menopause)
- Iatrogenic menopause
  - androgen (besides oestrogen) loss
  - associated disorder/disease
- Extent and severity of menopausal symptoms and impact on well-being
- Current disorders
- Current pharmacological treatment
- Substance abuse (mainly alcohol and opiates)

Psychosexual

- Loss of loving feelings toward partner
- Unpleasant/humiliating sexual encounters or experiences
- Affective disorders (depression, anxiety)
- Relationship of fertility loss to fulfilment of life goals

Contextual

- Relationship discord
- Life-stage stressors (e.g. child's diseases, divorce, separation, partner infidelity)
- Loss or death of close friends or family members
- Lack of access to medical/psychosocial treatment and facilities
- Economic difficulties

MAINTAINING FACTORS

Biological

- Diagnostic omissions: unaddressed predisposing/ precipitating biological aetiologies
- Untreated or inadequately treated comorbidities
  - physiatric: pelvic floor disorders
  - urologic: incontinence, lower urinary tract symptoms (LUTS), urogenital prolapse
  - proctologic: constipation, rhagades
  - metabolic: diabetes mellitus
  - psychiatric: depression, anxiety, phobias
- Pharmacological treatments
- Substance abuse
- Multisystemic changes associated with chronic disease or secondary to menopause

Box 9.4: Factors contributing to female sexual dysfunction—cont'd

  - hormonal
  - vascular
  - muscular
  - neurological
  - immunological
- Contraindications to hormone replacement therapy (HRT)
- Inadequacy of hormone replacement therapy in ameliorating menopause-associated biological symptoms

Psychosexual
- Low or loss of sexual self-confidence
- Performance anxiety
- Distress (personal, emotional, occupational, sexual)
- Diminished affection for or attraction to partner
- Unaddressed affective disorders (depression and/or anxiety)
- Negative perception of menopause-associated changes
- Body image concerns and increased body changes (wrinkles, body shape/weight, muscle tone)

Contextual
- Omission of menopause and female sexual dysfunction from provider's diagnostic and therapeutic approach
- Lack of access to adequate care
- Partner's general health or sexual problems or concerns
- Ongoing interpersonal conflict (with partner or others)
- Environmental constraints (lack of privacy, lack of time)

image issues (Graziottin 2006), binge eating disorders affecting self-esteem and self-confidence, attachment dynamics (secure, avoidant, anxious) (Clulow 2001) that may also modulate the level of trust in the relationship, the intensity of the commitment, and the confidence in loving and attitude towards affective and erotic intimacy.

Contextual descriptors include past and current significant relationships (Basson 2003, Leiblum & Rosen 2000), cultural/religious restrictions (Basson et al 2000, 2004), current interpersonal difficulties (Klausmann 2002, Liu 2003), partner's general health issues and/or sexual dysfunctions (Dennerstein et al 1999, 2003, 2004), inadequate stimulation and unsatisfactory sexual and emotional contexts (Levine 2003).

Generalized or situational?

Is the disorder generalized (with every partner and in every situation) or situational, specifically precipitated by partner-related or contextual factors, which should be specified (Basson et al 2000, 2004)? Situational problems usually rule out medical factors that tend to affect the sexual response with a more generalized effect (Graziottin 2004a, 2004b).

Lifelong or acquired?

Has the disorder been lifelong (from the very first sexual experience) or is it acquired after months or years of satisfying sexual intercourse? Asking the woman what in her opinion is causing the current FSD may offer useful insights into the aetiology of the disorder, particularly when it is acquired (Plaut et al 2004);

Level of distress

The level of distress indicates a mild, moderate, or severe impact of the FSD on personal life (Bancroft et al 2003, Dennerstein et al 2005, Graziottin & Koochaki 2003). Sexual distress should be distinguished from non-sexual distress and from depression. The degree of reported distress may have implications for the woman's motivation for therapy and for prognosis.

An interdisciplinary team is the most valuable resource for a patient-centred approach, both for diagnostic accuracy and tailored treatment. Key professional figures include a medical sexologist, gynaecologist, urologist, psychiatrist, endocrinologist, physiatrist, anaesthetist, neurologist, proctologist, dermatologist, psychotherapist (individual and couple), and physical therapist. Physical therapists are emerging as a key resource in addressing PFDs, which are finally receiving the attention they deserve as key biological factors in the aetiology of FSD.

WOMEN'S SEXUAL DESIRE/ INTEREST DISORDER

Hypoactive sexual desire disorder (HSDD) is the sexual dysfunction most frequently reported by women (Dennerstein et al 2003) The complaint of low desire becomes a sexual disorder when it causes severe personal distress to the woman. Population data indicate a prevalence of low desire in 32% of women between 18 and 59 years of age (Laumann et al 1999). A recent European survey of 2467 women, in France, UK, Germany and Italy indicates that the percentage of

women with low sexual desire is 19% in the age cohort from 20 to 49 years; 32% in the same age cohort in women who have experienced surgical menopause; 46% in postmenopausal women aged 50 to 70 years with natural menopause; and 48% in the same age cohort, after surgical menopause (Graziottin & Koochaki 2003).

The percentage of women distressed by their loss of desire and having a HSDD was 27% in premenopausal women and 28% after surgical menopause, in the age cohort 20–49 years, respectively; 11% in women with natural menopause; and 14% in those with surgical menopause aged 50 to 70 years (Graziottin & Koochaki 2003). The likelihood of HSDD increases with age, while the distress associated with the loss of desire is inversely correlated with age.

Surgical menopause secondary to bilateral oophorectomy has a specific damaging effect due to the loss of ovarian oestrogens and androgens. Ovaries contribute to more than 50% of total body androgens in the fertile age. A European survey on 1356 women indicated that women with surgical menopause had an odds ratio (OR) of 1.4 (CI = 1.1, 1.9; p = 0.02) of having low desire. Surgically menopausal women were more likely to have HSDD than premenopausal or naturally menopausal women (OR = 2.1; CI = 1.4, 3.4, p = 0.001). Sexual desire scores and sexual arousal, orgasm and sexual pleasure were highly correlated ($p < 0.001$). Women with HSDD were more likely to be dissatisfied with their sex life and their partner relationship than women with normal desire ($p < 0.001$) (Dennerstein et al 2005).

The leading biological aetiology of HSDD includes not only hormonal factors (low testosterone, low oestrogens, or high prolactin), but also depression and/or comorbidity with major diseases (see Box 9.4). Premature iatrogenic menopause is the most frequent cause of a biologically determined generalized loss of desire; the younger the woman, the higher the distress this loss causes to her (Dennerstein et al 2005, Graziottin & Basson 2004). Key questions to address women's desire disorders are summarized in Box 9.5. Unaddressed pain associated with sexual pain disorders, and causally related, among others, to hyperactivity of the pelvic floor up to a frank myalgia, is a frequently overlooked predisposing, precipitating and maintaining factor of acquired loss of desire (Graziottin 2000, Graziottin & Brotto 2004, Graziottin et al, 2001a, 2001b, 2004a).

What the clinician should look for

If a possible biological aetiology is suggested by the clinical history, the clinician should assess (Plaut et al 2004) the following.

Box 9.5: Sexual history for hypoactive sexual desire disorders and associated sexual comorbidities. Modified from Graziottin 2004a, with permission

GENERAL WELL-BEING

- How do you feel (physically and mentally)?
- Are you currently sexually active?
- If not, is that a concern for you? If yes, how's your sex life?

SEXUAL FUNCTION

- Have you always suffered from low sexual desire (lifelong) or has it faded recently (acquired)?
- Do you suffer from other sexual symptoms?
- For example, do you experience vaginal dryness?
- Do you have difficulty in getting aroused or lubricated?
- Do you have difficulty reaching orgasm?
- Do you feel pain during or after intercourse?
- Do you suffer from cystitis 24–72 hours after intercourse and/or other urinary symptoms?
- Is there any lifestyle-related factor that may affect your sexual desire (e.g. body weight, alcohol or drug abuse, little sleep, fatigue, professional distress)?
- What, in your opinion, is causing or worsening your sexual disorder? Is it a psychological problem, a past or current negative event (e.g. sexual harassment or abuse), something related to your physical health or your relationship, or something else?

SEXUAL RELATIONSHIP

- Do you have a stable relationship?
- How's your relationship? Are you satisfied with it?
- How is your partner's health (general and sexual)?
- Do you feel that your current sexual problem is more dependent on a physical or couple (loving/intimacy) problem?
- Is your sexual problem present in every context and/or with different partners (generalized), or do you complain of it in specific situations or with a specific partner (situational)?
- What made you aware of it and willing to look for help (e.g. intolerable personal frustration, fear of losing the partner, partner's complaints, new hope for effective treatment, more self-confidence in reporting)?
- Are you personally interested in improving your sex life?

- **Hormonal profile:**
  - total and free testosterone, dihydroepiandrosterone sulfate (DHEAS), prolactin, 17β-estradiol, sex hormone binding globulin (SHBG), with a plasma sample on the fifth or sixth day from the beginning of the menses in fertile women;
  - follicle stimulating hormone (FSH) and all of the above, in perimenopausal women;
  - thyroid stimulating hormone (TSH) when individually indicated.
- **The pelvic floor:** in all its components, with an accurate gynaecological, sexological and/or physiatric examination, particularly when comorbidity with arousal, orgasm and/or sexual pain disorders is reported.
- **Psychosexual factors and affective state:** depression first, with referral to a psychiatrist, sex therapist or couples therapist for a comprehensive diagnosis if indicated (Leiblum & Rosen 2000).

AROUSAL DISORDERS

Central arousal disorders ('I do not feel mentally excited') are comorbid with loss of sexual desire and can only be separated from it with difficulty. Genital arousal disorders with their key subjective symptom, vaginal dryness, are increasingly reported with age. In epidemiological surveys 19–20% of women complain of arousal disorders (Lauman et al 1999). This figure may increase to 39–45% in postmenopausal sexually active patients (Dennerstein et al 2003, 2005).

Mental arousal may be triggered through different pathways: biologically by androgens and oestrogens, psychologically by motivational forces such as intimacy needs, (i.e. the affective needs of love, tenderness, attention, bonding and commitment) (Laan & Everaerd 1995). With successful genital arousal, most women produce increased quantities of vaginal transudate. The neurotransmitter vasoactive intestinal peptide (VIP) stimulates this neurogenic transudate production. Oestrogens are believed to be powerful 'permitting factors' for VIP (Levin 2002). The neurotransmitter nitric oxide (NO) stimulates the neurogenic congestion of the clitoral and vestibular bulb corpora cavernosa. (Levin 2002). Reduction in vaginal lubrication is one of the most common complaints of postmenopausal women. When the plasma oestradiol concentration is below 50 pg/mL (the normal range in fertile women being 100–200 pg/mL) vaginal dryness is increasingly reported (Sarrel 1998). Physiological studies indicate that after menopause the vaginal pH increases from 3.5–4.5 to 6.0–7.39 owing to decreased glycogen production and metabolism to lactic acid, with dramatic modification of the vaginal ecosystem, and an average reduction of vaginal secretions of 50%.

Leading biological aetiologies of arousal disorders include loss of sexual hormones, primarily oestrogen, and PFDs.

- Hyperactivity of the pelvic floor may reduce the introital opening causing dyspareunia. (Unwanted) pain is indeed the strongest reflex inhibitor of genital arousal: genital arousal disorders, and the consequent vaginal dryness, are often comorbid with dyspareunia (Graziottin 2001a, 2004a, 2005b). Psychosexual and relational factors may also concur in this disorder (Box 9.6);
- A hypoactive or damaged pelvic floor (after traumatic deliveries, with macrosomic children or vacuum extraction) (Baessler & Schuessler 2004) may contribute to genital arousal disorder because it reduces the pleasurable sensations the woman (and partner) feel during intercourse (Graziottin 2004a).

What the clinician should look for

When a patient complains of an arousal disorder, the clinician should check (Plaut et al 2004):

- hormonal profile (see above), more so in hypoestrogenic conditions such as longlasting secondary amenorrhoea, puerperium, menopause (especially iatrogenic);
- general and pelvic health, focusing on pelvic floor trophism: vaginal, clitoral, vulvar, connective and muscular (looking for both hypertonic and hypotonic pelvic floor dysfunctions) (Graziottin 2001a, 2004a);
- vaginal pH with a simple stick because vaginal acidity correlates well with oestrogen tissue levels (Graziottin 2004a);
- biological factors, such as vulvar vestibulitis or poor outcome of perineal/genital surgery causing introital and/or pelvic pain (see dyspareunia);
- vascular factors that may impair the genital arousal response (smoking, hypercholesterolaemia, atherosclerosis, hypertension, diabetes mellitus) (Goldstein & Berman 1998);
- relational issues, inhibition and/or erotic illiteracy if a poor quality of mental arousal, poor or absent foreplay are reported; if this is so refer the willing couple to the sexual or couple therapist (Leiblum & Rosen 2000)

Box 9.6: Aetiology of dyspareunia. Modified from Graziottin 2004a, with permission

Many causes may overlap or be associated with coital pain with complex pathophysiological interplay. The relative weight of each cause in the individual woman may change with chronicity of pain and progressive involvement of other pelvic organs.

BIOLOGICAL

Superficial/introital and/or mid-vaginal dyspareunia

- Infectious: vulvitis, vulvar vestibulitis, vaginitis, cystitis
- Inflammatory, with mast cell upregulation
- Hormonal: vulvovaginal atrophy
- Anatomical: fibrous hymen, vaginal agenesis
- Muscular: primary or secondary hyperactivity of levator ani muscle
- Iatrogenic: poor outcome of genital surgery, pelvic radiotherapy
- Neurological: inclusive of neuropathic pain
- Connective and immunological: Sjögren's syndrome
- Vascular

Deep dyspareunia

- Endometriosis
- Pelvic inflammatory disease (PID)
- Pelvic varicocoele
- Chronic pelvic pain and referred pain
- Outcome of pelvic or endovaginal radiotherapy
- Abdominal nerve entrapment syndrome

PSYCHOSEXUAL

- Comorbidity with desire and /or arousal disorders, or vaginismus
- Past sexual harassment and/or abuse
- Affective disorders: depression and anxiety
- Catastrophism as leading psychological coping modality

CONTEXT OR COUPLE RELATED

- Lack of emotional intimacy
- Inadequate foreplay
- Conflicts: verbally, physically or sexually abusive partner
- Poor anatomical compatibility (penis size and/or infantile female genitalia)
- Sexual dissatisfaction and consequent inadequate arousal

ORGASMIC DISORDERS

Orgasmic disorder has been reported in an average of 24% of women during their fertile years in the epidemiological study of Lauman et al (1999). After the menopause, 39% of women complain of orgasmic difficulties, with 20% complaining that their clitoris 'is dead', according to Sarrel & Whitehead (1985).

Orgasm is a sensorimotor reflex that may be triggered by a number of physical and mental stimuli (Mah & Binik 2004).

Genital orgasm requires:

- integrity of the pudendal sensory nerve fibres (S2, S3, S4) and corticomedullary fibres;
- cavernosal structures that engorged and adequately stimulated convey pleasant sensory stimuli to the medullary centre and the brain;
- adequate motor response of the PFMs.

A short medullary reflex may trigger a muscular response characterized by involuntary contraction (three to eight times, in single or repetitive sequences) of the levator ani. The medullary reflex may be eased or blocked, respectively, by corticomedullary fibres that convey both excitatory stimuli when central arousal is maximal and inhibitory ones when arousal is poor. Performance anxiety may activate adrenergic input, which disrupts the arousal response. Inhibitory fibres are mostly serotonergic: this explains the inhibitory effects of selective serotonin reuptake inhibitors (SSRIs) on orgasm in both men and women (Seagraves & Balon 2003). Fear of leaking during intercourse may inhibit coital intimacy and/or orgasm (Barlow et al 1997, Cardozo et al 1998): leakage during coital thrusting is usually associated with stress incontinence, while leakage at orgasm is associated with urge incontinence.

Significant age-associated changes in the content of smooth muscle and connective tissue in the clitoral cavernosa contributing to age-associated clitoral sexual dysfunction causing hypo-anorgasmia, have been demonstrated from the first to the sixth decade of life and beyond by computer-assisted histomorphometric image analysis (Tarcan et al 1999).

What the clinician should look for

Using the information emerging from the clinical history as a starting point, the physician should assess:

- hormonal balance;
- signs and symptoms of vulvar dystrophy and, specifically, of clitoral and vaginal involution (Graziottin 2004a);

- traumatic consequences of female genital mutilation (infibulation);
- signs and symptoms of urge, stress or mixed incontinence, with either a hypotonic or hypertonic pelvic floor (Barlow et al 1997, Cardozo et al 1998);
- iatrogenic influences when potentially orgasm-inhibiting drugs are prescribed.

SEXUAL PAIN DISORDERS

Various degrees of dyspareunia are reported by 15% of coitally active women, and 22.5–33% of postmenopausal women. Vaginismus occurs in 0.5–1% of premenopausal women. However, mild hyperactivity of the pelvic floor, that could coincide with grade I or II of vaginismus according to Lamont (1978) may permit intercourse, causing coital pain (Graziottin 2003b, 2005a).

Vaginal receptiveness is a prerequisite for intercourse, and requires anatomical and functional tissue integrity, both in resting and aroused states. Normal trophism, both mucosal and cutaneous, adequate hormonal impregnation, lack of inflammation, particularly at the introitus, normal tonicity of the perivaginal muscles, vascular, connective and neurological integrity, and normal immune response are all considered necessary to guarantee vaginal 'habitability'. Vaginal receptiveness may be modulated by psychosexual, mental and interpersonal factors, all of which may result in poor arousal with vaginal dryness (Plaut et al 2004).

Fear of penetration, and a general muscular arousal secondary to anxiety may cause a defensive contraction of the perivaginal muscles leading to vaginismus (Reissing et al 2003, 2004, van der Velde et al 2001). This disorder may be the clinical correlate of a primary neurodystonia of the pelvic floor, as recently proven with needle electromyography (Graziottin et al 2004a). It may be so severe as to prevent penetration completely. Vaginismus is the leading cause of unconsummated marriages in women. The defensive pelvic floor contraction may also be secondary to genital pain of whatever cause (Travell & Simons 1983; Wesselmann et al 1997).

Dyspareunia is the common symptom of a variety of coital pain-causing disorders (see Box 9.6). Vulvar vestibulitis is its leading cause in premenopausal women (Abramov et al 1994, Friedrich 1987, Glazer et al 1995, Graziottin 2001a, Graziottin & Brotto 2004, Graziottin et al 2004b). The diagnostic triad is

1. severe pain upon vestibular touch or attempted vaginal entry;
2. exquisite tenderness to cotton-swab palpation of the introital area (mostly at 5 and 7, when looking at the introitus as a clock face);
3. dyspareunia (Friederich 1987).

From the pathophysiological point of view, vulvar vestibulitis involves the upregulation of:

- the immunological system (i.e. of introital mast cells with hyperproduction of both inflammatory molecules and nerve growth factors [NGF]) (Bohm-Starke et al 1999, 2001a, 2001b, Bornstein et al 2002, 2004);
- the pain system, with proliferation of local pain fibres induced by the NGF (Bornstein et al 2002, 2004, Westrom & Willen 1998), which may contribute to neuropathic pain (Graziottin & Brotto 2004, Woolf 1993);
- hyperactivity of the levator ani, which can be antecedent to vulvar vestibulitis (Abramov et al 1994, Graziottin 2005a, Graziottin et al 2004a), or secondary to the introital pain.

In either case, addressing the muscle component is a key part of treatment (Bergeron et al 2001, Glazer et al 1995, McKay et al 2001). Hyperactivity of the pelvic floor may be triggered by non-genital, non-sexual causes, such as urological factors (urge incontinence, when tightening the pelvic floor may be secondary to the aim of reinforcing the ability to control the bladder), or anorectal problems (anismus, haemorrhoids, rhagades). Comorbidity with other sexual dysfunctions – loss of libido, arousal disorders, orgasmic difficulties, and/or sexual pain-related disorders – is frequently reported with persisting/chronic dyspareunia (Graziottin et al 2001b).

What the clinician should look for

The diagnostic work-up should focus on:

- physical examination to define the 'pain map' (Graziottin 2001a, Graziottin & Basson 2004, Graziottin et al 2001c), (any site in the vulva, mid-vagina and deep vagina where pain can be elicited) because location of the pain and its characteristics are the strongest predictors of type of organicity (Meana et al 1997), and including pelvic floor trophism (vaginal pH), muscular tone, strength and performance (Alvarez & Rockwell 2002, Bourcier et al 2004), signs of inflammation (primarily vulvar vestibulitis) (Friedrich 1987, Graziottin & Brotto 2004), poor outcomes of pelvic (Graziottin 2001b) or perineal surgery (primarily episiotomy/episiorraphy) (Glazener 1997), associated urogenital and rectal pain syndromes (Wesselmann et al 1997), myogenic or neurogenic pain (Bohm-Starke 2001a, 2001b, Bornstein et al 2002, 2004) and vascular problems (Goldstein & Berman 1998);
- psychosexual factors, poor arousal and coexisting vaginismus (Leiblum 2000, Pukall et al 2005);

- relationship issues (Reissing et al 2003);
- hormonal profile, if clinically indicated, when dyspareunia is associated with vaginal dryness.

Pain is rarely purely psychogenic, and dyspareunia is no exception. Like all pain syndromes, it usually has one or more biological aetiological factors. Hyperactive PFDs are a constant feature. However, psychosexual and relationship factors, generally lifelong or acquired low libido because of the persisting pain, and lifelong or acquired arousal disorders due to the inhibitory effect of pain, should be addressed in parallel to provide comprehensive, integrated and effective treatment.

ETHICAL, LEGAL AND COUNSELLING RELATED CONSIDERATIONS

The topic of sexuality requires special attention being given to confidentiality and informed consent depending on the profession of the clinician and any local laws that place limits on confidentiality, such as in the reporting of sexual abuse. Although the discussion of sexual matters is often an appropriate part of medical evaluation and treatment, it is also important not to sexualize the clinical setting when it is not necessary. Patients may be confused or embarrassed by comments about their attractiveness, disclosure of intimate personal information by the clinician, or by sex-related questions that are neither clinically relevant nor justifiable. The modesty of the patient should be respected in touching, disrobing and draping procedures (Plaut et al 2004). Key aspects of appropriate counselling attitudes are summarized in Box 9.7.

Box 9.7: Talking with patients about sexual issues. From Plaut et al 2004, with permission

- Ask pointed questions and request clarification that will result in sufficiently specific data about the patient's symptoms
- Be sensitive to the optimal time to ask the most emotionally charged questions
- Look for and respond to non-verbal cues that may signal discomfort or concern
- Be sensitive to the impact of emotionally charged words (e.g. rape, abortion)
- If you are not sure of the patient's sexual orientation, use gender-neutral language in referring to his or her partner
- Explain and justify your questions and procedures
- Teach and reassure as you examine
- Intervene to the extent that you are qualified and comfortable; refer to qualified medical or mental health specialists as necessary

CONCLUSION

To address the complexity of FSD requires a balanced clinical perspective between biological and psychosexual/relational factors. Apart from counselling the FSD complaint in a competent way when the issue is openly raised by the patient, physicians and physical therapists can contribute to improving the quality of (sexual) life of their patients, by routinely asking them during the clinical history taking: 'How's your sex life'? so offering an opening for current or future disclosure. The wish is that the new attention to women's right for a better sexual life will significantly help increase the physician's confidence in asking and listening to complaints of FSD and his or her 'clinical impact factor' (i.e his or her ability to appropriately diagnose and effectively treat FSD).

In the tailoring of treatment, the physical therapist has a crucial role, especially in sexual pain disorders, either lifelong or acquired, and in acquired desire, arousal or orgasmic disorders secondary to coital pain. The enthusiasm that many physical therapists have when they can effectively treat or co-treat FSD for which a woman has been doctor-shopping for years are mirrored by the woman's satisfaction in finally feeling listened to, respected in the truth of her coital pain or other sexual complaints, and re-empowered in her body confidence, when she is taught how to command and appropriately relax her key muscles for sex and love.

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TREATMENT

Alessandra Graziottin

INTRODUCTION

There is no effective therapy without accurate and comprehensive diagnosis. This is even more true for female sexual dysfunction (FSD), which usually has a multifactorial aetiology. Biological, psychosexual and context-related factors (Basson et al 2000, 2004), further characterized as predisposing, precipitating and maintaining (Graziottin 2005a, Graziottin & Brotto 2004) may interact to give the FSD that the woman is complaining about its specific individual characteristics.