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## TREATMENT

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## 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.

The accurate diagnosis of FSD is currently a challenge for researchers and clinicians. The temptation of searching for the aetiology, preliminary to finding the optimal treatment, is usually inappropriate, and continuously frustrated by the complexity of female sexuality.

The delay in the medical approach to FSD and the persistent psychological perspective make it difficult to have evidence-based medical treatments of FSD except in the domain of sexual hormones. As a result of diagnostic delays, inadequacies, and gender biases, **no treatment for FSD is currently approved with this specific indication** with the exception of a clitoral device indicated for female arousal disorders (Wilson et al 2001). From the clinical point of view, an integrated diagnostic and treatment approach is therefore necessary to tailor treatment according to the individual and couple's needs at the best of our current scientific and clinical knowledge (Basson et al 2000, 2004, Graziottin 2001a, 2004a, 2004b, Plaut et al 2004).

The available evidence for treatment of FSD will be reviewed. Special focus will be given to the role of the physical therapist in addressing the muscle and pelvic floor-related contributors to FSD.

## DIAGNOSTIC KEY POINTS

Key points in the FSD diagnosis, preliminary to a well tailored treatment, should be:

- accurate listening to the complaint's wording, to verbal and non-verbal messages, with:
  - definition of the nature of the disorders;
  - is it lifelong or acquired?
  - is it generalized or situational?
  - is it organic, psychogenic, contextual or, as in most cases, mixed? with definition of key predisposing, precipitating and maintaining factors;
  - how severe is the distress it causes?
  - are there sexual and/or medical (e.g. urogenital, proctological) associated comorbidity – comorbidities may be other types of FSD, but also other medical conditions, such as urological, gynaecological, proctological, metabolic, cardiovascular and neurological diseases – for example, urinary tract symptoms have a relative risk (RR) of:
    - 4.02 (2.75–5.89) of being associated with arousal disorders
    - 7.61 (4.06–14.26) of being associated with sexual pain disorders (Laumann et al 1999)
  - partner's related issues;
  - the personal motivation the woman has (or does not have) to treatment of FSD, which includes the meaning of the symptom for the woman;

- accurate examination of the woman, and particularly of the external genitalia, vagina and pelvic floor (Graziottin 2004a,b, Graziottin et al 2001a,b,c) – careful physical examination should be performed because the biological aetiology of FSD is better diagnosed when attention is paid to vulvovaginal trophism with pH recording; hypo- or hypertonic pelvic floor conditions, with tender and trigger point evaluation; diagnosis of inflammation and infection, with culture examinations when indicated; and the painmap accurate description (Graziottin et al 2001c) because location of pain and its onset characteristics are the strongest predictors of its biological aetiology (Meana et al 1997).

This is mandatory when genital arousal disorders, sexual pain disorders (vaginismus and dyspareunia) and orgasm disorders are complained of. It may be useful even when sexual desire disorders and/or subjective sexual arousal disorders ('I do not feel mentally excited') are the leading complaints to diagnose biologically rooted comorbidities with other FSD. Comorbidity should be accurately recorded with attention to which sexual disorder came first. On the positive side, the cascade of positive feedback when a treatment is effective may cause a significant improvement in all domains of sexual response as several studies have proven (Alexander et al 2004, Graziottin & Basson 2004, Laan et al 2001, Shifren et al 2000, Simunic et al 2003).

In stable couples, current feelings for the partner (i.e. quality of the relationship, and the quality of the partner's sexuality [inclusive of general and sexual health]) should be investigated as well (Dennerstein et al 1999, 2003, 2007, Klausmann 2002).

The woman's general health should be examined, with special focus on conditions that may directly or indirectly impair the woman's mental and/or genital response (Basson et al 2000, 2004, Graziottin 2000, 2003a, 2004a,b).

## PRINCIPLES OF FSD THERAPY

A growing body of evidence implicates hormonal factors in the genesis of FSD (Alexander et al 2004, Graziottin & Basson 2004, Laan et al 2001, Sarrel 1998, Shifren et al 2000, Simunic et al 2003). Indeed, during a woman's entire reproductive life span, sex hormones exert both organizational and activational effects on sexual behaviour. The action of hormones is mediated by non-genomic and genomic pathways. Current evidence indicates that there is a specific place in the treatment of FSD for pharmacological hormones, for the most part in postmenopausal women (Alexander et al 2004,

Graziottin & Basson 2004, Laan et al 2001, Sarrel 1998, Shifren et al 2000, Simunic et al 2003). Sexual hormones may be delivered by various routes: oral, transdermal, nasal, vaginal, through subcutaneous implants or intra-uterine devices. The most important difference between the oral route and those that bypass the first hepatic pass is that the oral treatment induces an increase of sex hormone-binding globulin (SHBG) by as much as 133%, thus significantly reducing free testosterone (Vehkavaara et al 2000). Levels of SHBG seem to be unaffected by hormones delivered via transdermal, nasal, and vaginal routes.

Depending on the aetiological diagnosis of the leading disorder, the therapy should consider one or more of the following leading options.

### Libido disorder

Libido and subjective sexual arousal disorder ('I do not feel mentally excited'), often diagnosed in comorbidity, either lifelong or, more frequently, acquired, may benefit from the following.

### Medical treatment

#### Hormones

**Androgen** The major androgens in women include testosterone (T) and dihydrotestosterone (DHT), dehydroepiandrosterone sulphate (DHEA-S), dehydroepiandrosterone (DHEA), and androstenedione (A) (Bachmann et al 2002). T is the most potent androgen. Plasma T levels range from 0.2 to 0.7 ng/mL (0.6–2.5 nmol/L), with significant fluctuations related to the phase of the menstrual cycle. T is converted to DHT, but can also be aromatized to estradiol ( $E_2$ ) in target tissues; DHT is the principal ligand to androgen receptors in women as well. Androgens peak in the early 20s, then decline steadily (Burger et al 2000).

T in premenopausal women: evidence concerning the role of hormones, particularly T, in premenopausal women is limited. Very few studies have been done in premenopausal subjects. Goldstat et al (2003) focused their controlled study on a small group of premenopausal women; subjects with lifelong hypoactive sexual desire disorder with T levels in the lower one-third or less of the normal range may significantly benefit from T cream when compared to placebo.

T in postmenopausal women: menopause can be natural or iatrogenic. Iatrogenic menopause may result from surgery, chemotherapy, or radiation therapy. The most common surgical cause of menopause is bilateral oophorectomy, which leads to a sudden 50% fall in circulating T levels (Bachmann et al 2002). Plasma T values

at or below the lowest quartile of the normal range for women in their reproductive years also suggest a diagnosis of androgen insufficiency syndrome. A recent, systematic review of all available data from randomized and placebo-controlled trials of treatment for FSD in postmenopausal women concluded that use of many frequently used treatments is not supported by adequate evidence (Madelska & Cummings 2003). In their review of randomized, controlled trials involving the use of T in oestrogen-replete women, Alexander et al (2004) found general support for the positive effect of T on different dimensions of women's sexuality. One limit of this analysis is that some of the reviewed studies involved supraphysiological doses. In a study by Shifren et al (2000), the total T was raised above the normal range, but the free and bioavailable T remained within the normal range. Sherwin (2002) and more recently Alexander et al (2004) in their reviews of randomized, controlled trials, found that adding androgens to the standard oestrogen replacement had added sexual benefit in different domains, sexual desire first.

**Oestrogens and progestogens** In naturally postmenopausal women, progesterone or progestogens protect the endometrium. The positive effect of oestrogens on the well-being and sexuality of postmenopausal women may be variably modulated according to the type of progestogens added in the hormonal replacement therapy (Graziottin & Leiblum 2005). Progesterone, the physiological hormone, may have a mildly inhibiting effect on sexual desire. Progestogens, synthetic molecules with progestinic action, have a wide spectrum of actions from strongly antiandrogenic to neutral to androgenic, according to:

- their structure (whether they are derived from 17-OH-progesterone, 19-nortestosterone or 17-alpha-spirolactone) and their consequent varying pattern of interaction with different hormonal receptors (Graziottin & Leiblum 2005, Schindler 1999, Stanczyk 2002) – progestogens may interact with progestinic, oestrogenic, androgenic, glucocorticoid, and mineralcorticoid receptors, so the consequent metabolic and sexual profile differs;
- their variable binding affinity to SHBG, which modulates the quantity of free T available for its biological action;
- the variable inhibition of the type 2,5-alpha-reductase, which activates T into DHT.

To assimilate progestogens in a unique category focusing on a generalized 'class effect' is wrong and may lead to inappropriate conclusions (Graziottin &

Leiblum 2005). The progestogen with the most favourable effect on sexual function in hormonal replacement therapy is norethisterone, with a positive impact on desire, arousal, orgasm, and satisfaction in natural postmenopausal women with an intact uterus. Controlled head-to-head studies are necessary to evaluate the correlation between the pharmacological profile and the clinical effect.

**Tibolone** Tibolone is a 19-nortestosterone derivative with mild oestrogenic, progestinic and androgenic activity. It lowers SHBG, thus increasing free E<sub>2</sub>, T, and DHEA-S levels. It is not available in the USA, but is widely used in Europe. In randomized studies comparing it with placebo, tibolone (2.5 mg/day) alleviated vaginal dryness and dyspareunia, increasing libido, arousal, and sexual satisfaction in postmenopausal women with natural or surgical menopause (Laan et al 2001, Madelska & Cummings 2002).

**DHEA-S** Studies conducted in elderly women have shown a positive effect of DHEA-S on mental well-being and on motivational aspects of sexuality with a mild relief of climacteric symptoms (Labrie et al 2001, Stomati et al 2000).

**Hypoprolactinaemic drugs** Prolactin is the most powerful inhibiting hormone when sexual desire is considered, with increasing inhibiting effect with increasing plasma levels. Hypoprolactinaemic drugs are useful to improve sexual desire when the prolactin level is supraphysiological.

**Antidepressants** Affective disorders, namely depression and anxiety, when associated with sexual desire disorders should be addressed with a mixed approach, both pharmacological and psychodynamic (Alexander & Kotz 2004). Among antidepressants, bupropion seems to have the most positive effect on sexual desire (Clayton et al 2004, Seagraves & Balon 2003). Comorbidity between low testosterone and depression should be considered and appropriately treated.

**Pelvic floor rehabilitation** A few physicians and medical sexologists recommend careful physical examination of the woman complaining of low desire on the wrong assumption that the disorder is either 'all psychogenic and/or couple dependent' or at best 'hormone-dependent'. Low desire can result from negative feedback from disappointing arousal, coital pain, coital anorgasmia, dissatisfaction (see Fig. 9.26, p. 267). Indeed, low desire may be concomitant to sexual aversion disorders associated with vaginismus (with a variable hyperactivity of the pelvic floor) (Graziottin et al 2004a)

or secondary to sexual pain disorders such as dyspareunia associated with vulvar vestibulitis (Graziottin et al 2001b), in which defensive contraction of levator ani is common (Bergeron et al 2001, Glazer et al 1995, Graziottin et al 2004b, McKay et al 2001).

**Antalgic treatment** When loss of desire is acquired and secondary to persistent chronic coital pain, antalgic treatment aimed at reducing or eliminating pain (especially if neuropathic) is preliminary to effective normalization of sexual desire (Vincenti & Graziottin 2004).

### *Psychosexual treatment*

**Individual psychosexual or behavioural therapy** Individual psychosexual or behavioural therapy is the approach of choice if the FSD aetiology includes sexual inhibitions, poor erotic skills, poor body image, low self-confidence or previous abuse (Leiblum & Rosen 2000, Rellini & Meston 2004).

**Couple therapy** Couple therapy is used when symbiotic dynamics with poor differentiation according to Schnarch (2000) or conflicts and/or destructive dynamics are reported.

### *Referral*

The multisystemic and multifactorial aetiology of FSD requires a professional multidisciplinary team. Appropriate referral is a key part of successful treatment (Box 9.8) (Plaut et al 2004). For example, referral of the partner to the uroandrogologist should be recommended when male disorders (premature ejaculation, erectile deficit, libido disorders) emerge as critical co-factors in the aetiology of FSD (i.e. if the partner appears to be the 'symptom inducer' and the woman is the 'symptom carrier' [Kaplan 1979, Plaut et al 2004]).

Acquired libido disorder should be treated on the basis of the leading aetiological factor, especially if it is comorbid with other lifelong or acquired FSD, such as pain disorder, arousal disorder or orgasm disorder (Graziottin et al 2001b), or biological factors such as iatrogenic menopause (Graziottin & Basson 2004).

### **Arousal disorders**

Subjective sexual arousal disorders, either lifelong or acquired, usually in comorbidity with sexual desire disorders, should be treated as mentioned above. Postmenopausal mixed genital and subjective arousal disorders may benefit from systemic hormonal replacement therapy, especially androgens (see above) (Alexander et al 2004, Traish et al 2002).

**Box 9.8:** Referral resources. Modified from Plaut et al 2004, with permission

- Medical sexologist or gynaecologist trained in sexual medicine: FSD requires appropriate medical diagnosis and treatment
- Urologist or andrologist: when the partner has erectile or ejaculatory dysfunction that requires medical intervention
- Family physician trained in sexual medicine: for sexual dysfunctions in either partner
- Oncologist: when hormonal treatment is considered for patients who have had cancer
- Psychiatrist: when depression and anxiety are associated with FSD
- Sex therapist: to carry out the psychosexual therapy
- Couple therapist: when relationship issues are a primary contributor to the sexual dysfunction
- Individual psychotherapist: when personal psychodynamic issues are inhibiting sexual function
- Physical therapist: when hyper- or hypotonicity of pelvic floor is contributory

Isolated acquired genital arousal disorders may benefit from the following.

### Medical treatment

**Topical oestrogens** A number of studies suggest that topical vaginal oestrogens may significantly reduce vaginal dryness, increase genital arousal, and reduce dyspareunia (Dessole et al 2004, Rioux et al 2000, Simunic et al 2003). A multicentre, double-blind, randomized, placebo-controlled study (n = 1612 postmenopausal women with urogenital and sexual complaints) indicates that 25 µg of estradiol applied vaginally twice a week for a year may significantly improve six vaginal symptoms and signs: vaginal dryness (p < 0.0001), itching/burning (p < 0.0001), recurrent vaginitis (p < 0.0001), petechiae (p < 0.0002), dyspareunia (p < 0.0001), and vaginal atrophy (p < 0.0001), and five bladder symptoms and signs: dysuria (p < 0.003), frequency/nocturia (p < 0.001), urinary tract infection (p < 0.034), urinary incontinence, urge mostly (p < 0.002), and urinary atrophy (p < 0.001) (Simunic et al 2003). Furthermore, cystometry performed at baseline and after 12 months indicates that the maximal cystometric capacity increases from 200 mL to 290 mL (p < 0.023); the bladder volume at first urgency increases from

140 mL to 180 mL (p < 0.048); and bladder volume at strong urgency increases from 130 mL to 170 mL (p < 0.045). The comorbidity between urogenital and sexual symptoms in postmenopausal women may therefore be effectively addressed with a topical vaginal treatment that is easy to use and safe both for the endometrium and the breast.

**Topical testosterone** Testosterone propionate powder 1% or 2% in vaseline jelly applied in minimal daily quantity to the clitoris and the vulvar region may improve genital arousal in the external genitalia (Notelovitz 2002). Controlled studies, however, are lacking.

**Vasoactive drugs** Evidence on the effectiveness of vasoactive drugs (sildenafil, vardenafil, tadalafil) in addressing genital arousal disorders in women is negative or at best controversial, with one exception (Berman et al 2003). The frequent comorbidity with desire disorders, the frequent couple issues, the difficulty in diagnosing a 'pure' genital arousal disorder and the lack of a personal motivation for a pharmacological treatment of genital arousal disorder may explain the substantial lack of efficacy in comparison to men's genital arousal disorders (i.e. erectile deficit of vascular aetiology).

**Clitoral vacuum device** Clitoral vacuum device is the only FDA-approved treatment for genital arousal disorders with a vascular and/or neurogenic aetiology (Wilson et al 2001). It may be useful in women treated for invasive carcinoma of the cervix who have undergone surgery and pelvic radiotherapy.

**Pelvic floor rehabilitation** Genital arousal disorders may be secondary to coital pain: unwanted pain is the strongest reflex inhibitor of vaginal congestion and lubrication. Diagnosing and treating the muscular component of coital pain (both in vaginismus and dyspareunia) is a key part of the medical treatment (Bergeron et al 2001, Glazer et al 1995, Graziottin 2004d, McKay et al 2001) and is preliminary to resuming a normal vasocongestive response (Graziottin & Brotto 2004).

### Psychosexual treatment

Indications for psychosexual treatment of subjective sexual arousal disorders overlap with those for desire disorders. Co-treatment may therefore effectively address comorbidity. However, treatment of the potential parallel biological aetiology of the genital arousal disorder is mandatory if cure for the reported FSD is to be achieved (Plaut et al 2004). Couple psychotherapy should be proposed when relational dynamics are con-

tributing to maintenance of the sexual problem (Clulow 2001, Leiblum & Rosen 2000).

### Orgasm disorders

Orgasm disorders have a prevalent psychogenic aetiology in young women (Mah & Binik 2004). Biological factors – age, menopause-related loss of sexual hormones, pelvic floor disorders, iatrogenic issues (such as antidepressant serotonergic drugs inhibiting orgasm), and comorbidities (mainly with stress and urge incontinence) – become increasingly important with increasing age (Graziottin 2004a). According to the aetiological diagnosis, the main therapeutic options include the following.

#### Medical treatment

**Systemic and/or topical hormonal replacement therapy** Systemic and/or topical hormonal replacement therapy is discussed above. Testosterone has a special role in the treatment of orgasmic disorders associated with loss of sexual hormones, especially after bilateral oophorectomy (Alexander et al 2004, Sherwin 2002, Shifren et al 2000). It behaves as ‘initiator’ in the brain and as ‘modulator’ in the cavernosal bodies, where it works as ‘permitting factor’ for nitric oxide (NO), in women as well as in men (Graziottin 2004d).

Change of pharmacological treatment inhibiting orgasm (e.g. antidepressants such as selective serotonin reuptake inhibitor [SSRI] or tricyclics) should be considered when feasible from the medical point of view if orgasm inhibition is reported as a side-effect. Bupropion seems to be a better choice (Clayton et al 2004, Seagraves & Balon 2003).

**Pelvic floor rehabilitation** Pelvic floor rehabilitation is of the highest importance for hypotonic conditions of the pelvic floor, as pioneered by Kegel (1952), after delivery (Baessler & Schuessler 2004, Glazener 1997); even more so when incontinence is a strong inhibiting orgasmic factor. Fear of leaking during thrusting in stress incontinence and at orgasm in urge incontinence is an often under-reported and yet powerful disruptor of orgasm potential. Orgasm inhibition may also be secondary to coital pain (Graziottin et al 2001b). Again, accurate diagnosis of comorbidity and appropriate co-treatment with relaxation of the pelvic floor in this latter case is key.

#### Psychosexual treatment

**Individual psychosexual or behavioural therapy** Lifelong ‘isolated’ orgasmic disorders may

benefit from a behavioural educational treatment, encouraging self-knowledge and eroticism with the experience of higher arousal sensations, use of vibrators or of a clitoral device up to orgasm (Meston et al 2004). More often, however, the orgasmic disorder is associated with poor arousal with or without performance anxiety. These conditions should therefore be treated together (Leiblum & Rosen 2000).

**Couple therapy** Lifelong orgasm difficulties may need a couple therapy when sexual inhibitions, poor erotic skills and/or low self-confidence are shared by the couple (Meston et al 2004).

Appropriate behavioural and pharmacological treatment of premature ejaculation should be proposed to the partner when it causes inadequate coital stimulation and increasing erotic dissatisfaction in the female partner.

If all of the sexual response is impaired, with significant comorbidity with desire and arousal disorders, accurate treatment of predisposing, precipitating and maintenance factors, biological, psychosexual and/or contextual, should be proposed (Plaut et al 2004).

### Sexual pain disorders

Dyspareunia and vaginismus because of coital pain directly inhibit genital arousal and vaginal receptivity. Indirectly, they may affect orgasm potential, the physical and emotional satisfaction, causing loss of desire up to avoidance of sexual intimacy. Dyspareunia may have many biological aetiologies: the leading cause of coital pain in premenopausal women is vulvar vestibulitis, whereas postmenopausally it is vaginal dryness.

Dyspareunia may benefit from the following (Box 9.9).

#### Medical treatment

**Multimodal therapy** Vulvar vestibulitis should be treated with a combined treatment aimed at reducing:

- upregulation of mast cells, both by reducing the agonist stimuli (such as candida infections, micro-abrasions of the introital mucosa because of intercourse with a dry vagina and/or a contracted pelvic floor, chemicals, allergens etc) that cause degranulation leading to chronic tissue inflammation, and/or with antagonist modulation of its hyper-reactivity, with amitriptyline or aliamides gel (Graziottin & Brotto 2004, Graziottin et al 2004b);
- upregulation of the pain system secondary to proliferation of introital pain fibres (Bohm-Starke et al 1999, 2001a,b, Bornstein et al 2002, 2004) induced by nerve

**Box 9.9:** Treatment of the medical causes of dyspareunia**INFLAMMATORY AETIOLOGY (UPREGULATION OF MAST CELLS)****Pharmacological modulation of mast cell hyper-reactivity**

- Antidepressants: amitriptyline
- Aliamides topical gel

**Reduction of agonist factors causing mast cell hyper-reactivity**

- Recurrent candida or *Gardnerella vaginitis*
- Microabrasions of the introital mucosa
  - from intercourse with a dry vagina
  - from inappropriate lifestyles
- Allergens/chemical irritants
- Physical agents
- Neurogenic stimuli

**MUSCULAR AETIOLOGY (UPREGULATION OF THE MUSCULAR SYSTEM)**

- Self-massage and levator ani stretching
- Physical therapy of the levator ani
- Electromyographic biofeed-back
- Type A botulinum toxin

\*Aliamides is a class of endogenous molecules with an anti-inflammatory activity. The most important is the palmitoiletanolamide, belonging to the class of fatty acid amides, chemically known as N-(2-idrossietil)-esadecanamide. They work through the down-regulation of the hyperactive mast cells. In Italy they are available in the form of vaginal gel and now of pills. They constitute an innovative approach to the vaginal and bladder chronic inflammation, secondary to mast cells' upregulation.

**NEUROLOGICAL AETIOLOGY (UPREGULATION OF THE PAIN SYSTEM)****Systemic analgesia**

- Amitriptyline
- Gabapentin
- Pregabalin

**Local analgesia**

- Electroanalgesia
- Ganglion impar block

**Surgical therapy**

- Vestibulectomy

**HORMONAL AETIOLOGY****Hormonal therapy**

- Local:
  - vaginal oestrogens
  - testosterone for the vulva
- Systemic:
  - hormonal replacement therapies

growth factor produced by the upregulated mast cells, and the lowered central pain threshold (Pukall et al 2006) – a thorough understanding of the pathophysiology of pain in its nociceptive and neuropathic component, is mandatory – antalgic treatment should be prescribed: locally, with electroanalgesia (Nappi et al 2003) or, in severe cases, with the ganglion impar block; systemically with tricyclic antidepressant or gabapentin in the most severe cases (Graziottin & Brotto 2004, Vincenti & Graziottin 2004).

- upregulation of the muscular response, with hyperactivity of the pelvic floor (Graziottin et al 2004a), which may precede vulvar vestibulitis when the predisposing factor is vaginismus (Abramov et al 1994, Graziottin et al 2001b) or be acquired in response to genital pain (Graziottin et al 2004a,b) – in controlled studies, electromyographic feedback (Bergeron et al 2001, Glazer et al 1995, McKay et al 2001) has proven to significantly reduce pain of vulvar vestibulitis; self

massage, pelvic floor stretching and physical therapy may also reduce the muscular component of coital pain (Graziottin 2004a, Graziottin & Brotto 2004), but high-quality randomized controlled trials are needed to determine the true effect of such interventions; for hyperactivity of the pelvic floor, treatment with type A botulinum toxin has been proposed (Bertolasi 2004, personal communication) – individually tailored combinations of this approach are useful for treating introital dyspareunia with different aetiologies from vulvar vestibulitis.

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

**Topical hormones** Vaginal oestrogen treatment is mandatory when vaginal dryness is causing postmenopausal dyspareunia, either spontaneous or iatrogenic

(Graziottin 2001a,b, 2004a, Simunic et al 2003). Vulvar treatment with testosterone may be considered when vulvar dystrophy and/or lichen sclerosus contribute to introital dyspareunia.

### *Psychosexual treatment*

**Psychosexual and/or behavioural therapy** Psychosexual and/or behavioural therapy is the leading treatment of lifelong vaginismus (Leiblum 2000). It should be offered in parallel with progressive rehabilitation of the pelvic floor and pharmacological treatment to modulate the intense systemic arousal in the subset of intensely phobic patients (Plaut et al 2004). In this latter group, comorbidity with sexual aversion disorder should be investigated and treated first.

Psychosexual and/or behavioural therapy contributes to the multimodal treatment of lifelong dyspareunia, which is reported in one-third of our patients (Graziottin et al 2001b). Anxiety, fear of pain and sexual avoidant behaviours should be addressed as well. The shift from pain to pleasure is key from the sexual point of view. Sensitive and committed psychosexual support to the woman and the couple is mandatory.

## **WHEN THE PHYSICAL THERAPIST COUNTS**

Pelvic floor muscles are critically involved in the physiology and pathophysiology of women's sexual response. The physical therapist should be part of the multidisciplinary team involved in the centre of sexual medicine. He or she should diagnose and address the following.

### **Hyperactivity/hypertonus of the pelvic floor**

The physical therapist should diagnose and address:

- primary pelvic floor hyperactivity in children and adolescents, thus preventing one of the most neglected predisposing factors to dyspareunia and vulvar vestibulitis (Chiozza & Graziottin 2004, Graziottin 2005a, Harlow et al 2001);
- acquired hyperactivity with levator ani myalgia by overexertion (i.e. 'Kegel dyspareunia'; DeLancey et al 1993);
- lifelong hyperactivity of the pelvic floor in vaginismus and lifelong or acquired hyperactivity in dyspareunia of any aetiology (Graziottin 2003a);

- levator ani tender and/or trigger points with referred pain (Alvarez & Rockwell 2002, Travell & Simons 1983);
- levator ani hyperactivity associated with recurrent cystitis, urge incontinence and dyspareunia (Graziottin 2004a);
- systemic postural problems in chronic pelvic pain, dyspareunia and vaginismus;
- chronic pelvic pain and chronic coital pain-associated myalgias and pertinent antalgic treatment (Bourcier et al 2004).

### **Hypoactivity/hypotonus of the pelvic floor**

The physical therapist should diagnose and address:

- pelvic floor damage after delivery;
- hypotonicity worsening after the menopause;
- pelvic floor hypotonus in comorbidity with urogenital and/or proctological disorders (Bourcier et al 2004, Wesselmann et al 1997);

The physical therapist may also help the patient to increase awareness of the levator ani role in sexual receptivity and vaginal sensitivity to increase the woman's and her partner's coital pleasure.

## **CONCLUSIONS**

The complexity of FSD requires a dedicated diagnostic and therapeutic team, sharing a common pathophysiological and psychodynamic cultural scenario with the aim of offering the most integrated understanding of the meaning of the symptoms and the most effective comprehensive treatment.

Pelvic floor muscles are critically involved in the physiology and pathophysiology of a women's sexual response. Physical therapists may therefore greatly contribute to improving women's sexual health. They deserve appreciation and an increasing role in the multimodal treatment of FSD. There is, however, an urgent need for high-quality randomized controlled trials to evaluate the effect of different physical therapy interventions for FSD. A collaboration between physical therapists and sexologists/gynaecologists in future research projects in this important field is highly recommended.



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# Male sexual dysfunction

Grace Dorey

## INTRODUCTION

Sexual function in normal men is dependent on satisfactory libido, erectile function, ejaculation and orgasm. Sexual dysfunction occurs when there is a problem in any of these events. Sexual dysfunction embraces low libido, erectile dysfunction, premature ejaculation, retrograde ejaculation, retarded ejaculation, anorgasmia, anejaculation, and sexual pain.

## LOW LIBIDO

### Definition

A low libido can be defined as 'a reduced sexual urge'. As men age, there is a partial androgen decline.