Problems with Arousal and Orgasm in Women

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Learning Objectives

In this chapter, we:

- Provide definitions and epidemiological data.
- Describe biological and psychosocial etiological risk factors that contribute to arousal and orgasm disorders in women.
- Provide an extensive assessment that addresses the biopsychosocial nature of arousal and orgasm difficulties.
- Recommend pharmacological and psychological treatments.

Definitions and Epidemiology

Arousal and orgasm are intimately linked as important components in healthy female sexual function. Disorders of arousal and orgasm are quite common, with prevalence rates estimated at 25% of sexually active women reporting difficulties in each domain. The classification of female sexual dysfunction has undergone change over the past 50 years (for a review, see Leiblum, 2006). And while definitions have recently been revised (Sidebar 7.1), they will continue to evolve as new research and data emerge (Leiblum, 2006).
Defining Sexual Arousal Disorders (FSAD) in Women

The DSM-IV-TR definition of FSAD is limited to physiological sexual response only. The following three subtypes of FSAD have been recommended:

1. Subjective sexual arousal disorder.
2. Genital sexual arousal disorder.
3. Combined genital and subjective sexual arousal disorder.

A new arousal disorder: persistent sexual arousal disorder (PSAD), has been suggested.

Sexual Arousal Disorders

The most widely used definition for female sexual arousal disorder (FSAD) within psychological/psychiatric settings is that of the DSM-IV-TR (American Psychiatric Association, 2000), which states that FSAD is a persistent or recurrent inability to attain or to maintain until completion of sexual activity an adequate lubrication or swelling response of sexual excitement that causes marked distress or interpersonal difficulty. A panel of 13 experts in female sexual dysfunction selected from five countries convened to review the existing definitions of women’s sexual dysfunction. With regard to women’s arousal concerns, the committee criticized the DSM-IV-TR definition because it was based exclusively on a physiological response. An underlying assumption of this definition is that physiological and subjective experiences of sexual arousal are synchronous in women when, in fact, research indicates they are often desynchronous. In the publication that resulted from this conference, the committee suggested that the following three subtypes of FSAD better describe women’s sexual arousal concerns than did existing definitions (Basson et al., 2003):

1. **Subjective sexual arousal disorder**, which refers to the absence of or markedly diminished feelings of sexual arousal (sexual excitement and sexual pleasure) from any type of sexual stimulation. Vaginal lubrication and other signs of physical response still occur.

2. **Genital sexual arousal disorder**, which is often seen in women with autonomic nerve damage and in some estrogen deficient women and refers to absent or impaired genital sexual arousal (e.g., minimal vulval 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.

3. **Condensed genital and subjective arousal disorder** which is the most common clinical presentation and is usually comorbid with lack of sexual interest.

The committee also recommended a provisional definition of a new disorder, persistent sexual arousal disorder (PSAD), which includes a spontaneous, intrusive, and unwanted genital arousal (e.g., tingling, throbbing, pulsating) in the absence of sexual interest and desire. Awareness of subjective arousal is typically but not invariably unpleasant, and the arousal is unrelieved by orgasm and persists for hours or days (Basson et al., 2003; Leiblum, Brown, Wan, & Rawlinson, 2005). This disorder was previously considered extremely rare, but is now increasingly reported by clinicians (Basson, Leiblum, et al., 2004).

Epidemiological research indicates that between 8% to 15% of all women and 21% to 31% of sexually active women experience lubrication difficulties (for a review, see Lewis et al., 2004). Approximately 21% of these women report distress about their sexuality (Bancroft, Lotus, & Long, 2003) and 44% would like to receive help for their disorder (Dunn, Croft, & Hackett, 1998). To date, prevalence rates of PSAD have not been established.

**Female Orgasmic Disorder**

Female orgasmic disorder (FOD) is defined in the *DSM-IV-TR* as follows: "persistent or recurrent delay in, or absence of, orgasm following a normal sexual excitement phase" (American Psychiatric Association, 2000, p. 549). The diagnosis of FOD is made only when there is no dysfunction in the preceding phases of sexual response; so, women who experience difficulties in the excitement phase of sexual response would not be diagnosed with FOD (see Sidebar 7.2). "Women exhibit wide variability in the type or intensity of stimulation that triggers orgasm. The diagnosis of FOD should be based on the clinician’s judgment that the woman’s orgasmic capacity is less than would be reasonable for her age, sexual experience, and the adequacy of sexual stimulation she receives" (American Psychiatric Association, 2000, p. 549). According to the *DSM-IV-TR* criteria, in order to be diagnosed with FOD, the patient must have "marked distress or interpersonal difficulty" as a result of the orgasmic difficulties. FOD diagnoses should specify the nature of the onset (lifelong versus acquired), the context in which the problem occurs (generalized versus specific), and the associated etiological factors (psychological or combined). Lifelong FOD is often referred to
in the literature as primary anorgasmia and acquired FOD as secondary anorgasmia. A woman who can achieve orgasm through masturbation or through manual stimulation with a partner but not from intercourse alone would not meet American Psychiatric Association criteria for a diagnosis of FOD.

**SIDEBAR 7.2**

**Defining Female Orgasmic Disorder (FOD)**

FOD is only diagnosed when all preceding phases of sexual response are functional and when the woman is distressed by the lack of orgasm.

Primary anorgasmia is diagnosed in women who have never had an orgasm, while secondary anorgasmia is acquired after previously being orgasmic.

The definition of orgasm itself has proven difficult in part because the neural changes that underlie it are not well understood (Meston, Coll, Levin, & Sipski, 2004). An article cataloging definitions of orgasm included more than 25 comprehensive definitions written by different authors (Mah & Bini, 2001). The following definition of female orgasm was derived by the committee on female orgasm, presented at the International Consultation on Urological Diseases in Official Relationship with the World Health Organization (WHO), Paris, 2003:

> An orgasm in the human female is a variable, transient peak sensation of intense pleasure, creating an altered state of consciousness, usually accompanied by involuntary, rhythmic contractions of the pelvis, situated circumvaginal musculature often with concurrent uterine and anal contractions and myotonia that resolves the sexually induced vascongestion sometimes only partially, usually with an induction of well-being and contentment. (Meston et al., 2004, p. 785)

According to the National Social and Health Life Survey (NSHLS), orgasm difficulties are the second most frequently reported sexual problems for women (Laumann, Gagnon, Michael, & Michaels, 1994). The women who completed the NSHLS were a random sample of 1,745 American women, and 24% reported orgasm difficulties over the past year. Further studies of healthy women recruited in clinic settings reported similar percentages of women with orgasmic difficulties or anorgasmia (e.g., Read, King, & Watson, 1997).
Etiologic Factors

Biological factors such as an endocrine deficiency or other organic problems may be the sole cause of the development of FSAD. More often, biological factors contributing to FSAD arise in conjunction with or as a result of psychological factors, and therefore it is nearly impossible to separate conditions due to organic causes from those due to psychological causes (Leiblum, 2006). To date, etiological explanations for PSAD have not been well established, FOD is usually psychologically based.

For expediency, biological factors have been categorized into endocrine, autonomic nervous system, and medical/cardiovascular. Endocrine factors include sex steroids, peptide hormones, and general endocrine conditions. Autonomic factors refer to the relative contributions of the sympathetic and parasympathetic nervous systems. Medical and cardiovascular factors include those that are endocrine-related, cardiovascular, mobility/physical, and drug related. Table 7.1 lists organic factors that affect sexual arousal in women.

Endocrine Factors

Sex Steroids

It is difficult to separate the effects of the various sex steroids (estrogens, androgens, and progestins) on female sexual arousal and orgasm because they are all structurally related and derived from one another. However, researchers have been able to make edu-

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<th>Table 7.1</th>
<th>Organic Factors Related to Sexual Arousal in Women</th>
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<td>Estrogens are critical for vaginal functioning; postmenopausal women experience the most difficulties due to reduced estrogen.</td>
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<td>Androgens are believed to be related to arousal and orgasm, but the nature of the relationship is still unclear.</td>
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<td>PDE-5 inhibitors increase genital arousal in women, but not subjective arousal.</td>
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<td>An optimal level of sympathetic nervous system arousal appears necessary for sexual arousal in women.</td>
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<td>Medical factors that interfere with arousal and orgasm include endocrine and cardiovascular problems and use of antidepressants and anxiolytics.</td>
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<td>Aside from using antidepressants, women with Persistent Sexual Arousal Disorder (PSAD) report low levels of medical problems.</td>
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lated assumptions about which hormones are active for specific purposes. Estrogens in the peripheral nervous system are critical for the maintenance of vaginal tissue function and structure (Traish & Kim, 2004), and estrogen deficiency has been linked to various vaginal problems, including reduced or delayed lubrication, reduced vaginal blood flow, and increased dyspareunia (e.g., Sarett, 2006). Estrogens are not related to the ability to experience orgasm, as demonstrated through studies of estrogen treatment on postmenopausal and oophorectomized women (for a review, see Meston et al., 2004).

Most research on the effects of estrogen on sexual function has been done in women who are postmenopausal or who have undergone ovarian removal (oophorectomy). In postmenopausal women, the reduction in estrogen levels occurring when the menstrual cycle ends is associated with increased pH in the vagina, a reduction or delayed onset of lubrication in response to sexual stimulation, and structural changes in the vagina and vulva. These structural changes can include thinning and reduction of elasticity of the vaginal wall, changes to the vaginal epithelium, and loss of collagen in the vulva (Bachmann, Ebert, & Burd, 1999). In turn, these changes can lead to arousal difficulties, due to a reduction in tissue sensitivity and impaired vaginal lubrication. In a study of postmenopausal women, Sarett (1999) found that women treated with estradiol experienced fewer problems with vaginal dryness and pain than counterparts with less estradiol. Furthermore, oophorectomized and postmenopausal women who received estrogen replacement therapy showed increased lubrication, increased vulvar and vaginal blood flow, decreased pain with intercourse, and a reduction in vaginal pH (e.g., Patel, Brown, & Bachmann, 2006).

In premenopausal women, androgens are secreted from both the adrenal glands and the ovaries. Dehydroepiandrosterone (DHEA) and androstenedione are precursors to testosterone, and all three hormones are involved in desire and motivation for sexual activity. Some speculate that androgens are also involved in female sexual arousal and orgasm, although the mechanisms are not well understood (Traish, Kim, Min, Munarriz, & Goldstein, 2002). One study found a positive correlation between testosterone and genital arousal over the menstrual cycle in healthy, premenopausal women (Schreiner-Engel, Schnatt, Smith, & White, 1981), and more directly, administration of testosterone to premenopausal women increases genital arousal (Tuiten et al., 2002). However, androgens also affect sexual arousal indirectly because they moderate mood, energy, sexual desire, and overall well-being (Traish & Kim, 2006).

Postmenopausal women with low levels of testosterone and DHEA tend to have difficulties with orgasm (Meston & Davis, 2002).
Decreased testosterone has been anecdotally related to a reduction in intensity of orgasm, but this information is based on a population of patients taking antiepilepsy drugs (Duncan, Blacklaw, Bresnall, & Brodie, 1997) and on postmenopausal women (Guay & Sparks, 2006). As women age—even before menopause—levels of testosterone, DHEA, and DHEA-S decline significantly and reliably (e.g., Labrie, Belanger, Guse, Goyon, & Cauda, 1997; Zirmoll, Strain, Miller, & Rosner, 1995). To date, no studies have shown a definitive relationship between the decline in these hormones and changes in sexual arousal or orgasm (Guay et al., 2004). Of the few studies directly assessing the effect of DHEA on sexual arousal, administration of exogenous DHEA resulted in increased genital arousal in postmenopausal women (Hackbert & Heiman, 2002), but had no influence on genital or subjective arousal in premenopausal women (Meston & Heiman, 2002).

Clinically assessed androgen insufficiency in pre- and postmenopausal women is associated with general difficulties in sexual functioning, including sexual arousal. Sexual symptoms of androgen insufficiency include reductions in desire and motivation for sexual activity, sexual arousability, vaginal lubrication, and orgasmic capabilities (Guay & Davis, 2002). The etiology of androgen insufficiency is often rooted in a specific medical problem or its treatment, such as cancer treatments, oophorectomy, natural menopause, adrenal problems, hypopituitarism, Addison's disease, and use of estrogen, progesterone, antiandrogens, or corticosteroids (Bachmann et al., 2002).

Oxytocin

Research on animals has shown a strong relationship between oxytocin and muscle contractions similar to those in human orgasm. In humans, this link is less certain due to difficulties measuring oxytocin during sexual activity, or in the central nervous system given that central oxytocin does not cross the blood brain barrier. Measured in blood plasma, oxytocin has been positively correlated with the intensity of smooth muscle contractions during orgasm (Anderson-Hunt & Dennerstein, 1995) and, as it changes across the menstrual cycle, with vaginal lubrication (Salonia et al., 2005). To our knowledge, no studies have examined the effects of oxytocin deficiency on arousal or orgasmic ability in women.

General Endocrine Factors

Although the influence of steroid hormones on sexual arousal and orgasm is not fully understood in women, an appropriate balance of androgens, estrogens, and progestins appears critical (for a review, see Meston et al., 2004). Gonadal steroid hormones are believed to increase sensitivity and arousability to sexual stimuli, acting in the brain to increase attention to sexually related incen-
lives, emotions, and potential rewards (Guan & Davis, 2002); and on vaginal tissues to make genital arousal and orgasm possible (Stephenson, Georgeson, & Elans, 2000). Hormones control much of the female arousal response both directly and through control of various neurotransmitters, which influence arousal components (Basson, Weijman, Shulz et al., 2004). Although alteration in hormone balance may lead to problems in arousal and orgasm, they rarely are the cause of such problems in premenopausal women (Harish & Kim, 2006).

**Nitric Oxide**

Nitric oxide, released from the autonomic nervous system into the genital area, plays an important role in vaginal vasodilation. In animals, the administration of a phosphodiesterase type 5 (PDE-5) inhibitor (e.g., sildenafil), which indirectly increases nitric oxide in vaginal tissues, greatly enhances clitoral and vaginal blood flow resulting from stimulation of the pelvic nerve (e.g., Angulo, Cuevas, Cuesta, Bischoff, & Saez de Tejada, 2003). Several placebo-controlled studies of sildenafil and other nitric oxide agonists have been conducted on women with and without arousal problems. Self-report studies have shown improvement in measures of arousal and orgasm in premenopausal women with FSAD (Caruso, Intesano, Luppo, & Agnello, 2001) and increased arousal and genital sensation in postmenopausal women with FSAD (J. R. Berman, Berman, Tozer, Gill, & Haugbue, 2003). Studies using physiological measures of arousal have found increased vaginal pulse amplitude (VPA, a measure of genital arousal) in women with FSAD who received the nitric oxide precursor, L-arginine, combined with yohimbine (Meston & Worcel, 2002) and in healthy premenopausal women who received sildenafil (Laan et al., 2002). However, in one study of postmenopausal women with genital arousal disorder, only those women with the most severely reduced levels of vaginal vasodilation responded to treatment with a PDE-5 inhibitor (Basson & Brody, 2003). Although there appear to be improvements in physiological arousal in response to nitric oxide agonists, none of the studies has shown a significant improvement in women's subjective sexual arousal.

**Autonomic Nervous System**

Sympathetic and parasympathetic nervous systems (SNS and PNS) each play an important role in genital arousal and orgasm in women, but the relationship between the two systems is not well understood. Norepinephrine (NE) is the key neurotransmitter involved in SNS communication, and when measured after exposure to a sexually arousing film, plasma NE is higher than pre-film levels (Cox et al., 2000). Acetylcholine is the main neurotransmitter involved in PNS cholinergic communication, but...
because it is also involved in SNS communication, it is difficult to isolate the contribution of the PNS in genital arousal and orgasm. However, both adrenergic (NE) and cholinergic (ACH) systems are important contributors to normal arousal and orgasm.

The SNS contribution to genital arousal has been demonstrated in animal models, in women with spinal cord injuries (SCI), and in humans through drug manipulations and exercise-induced activation of the SNS. In animal models, stimulation of both PNS fibers and SNS fibers triggers contractions in uterine and cervical smooth muscle (Kim, Min, Huang, Goldstein, & Traish, 2002; Sato, Hotta, Nakayama, & Suzuki, 1996). However, stimulation of the pelvic nerve (part of the PNS) causes increased uterine blood flow, while stimulation of the hypogastric nerve (SNS) causes decreased uterine blood flow (Sato et al., 1996). Women with spinal cord injuries in areas that give rise to efferent (outgoing) SNS fibers provide excellent models for studying the contributions of the SNS to sexual arousal. Specifically, women with injuries between T10 and T12 levels in the spinal cord, an area of sympathetic nerves that project to the genital region, show a lack of lubrication in reflex and psychogenic arousal conditions (Berrard, 1989).

Evidence for the role of SNS involvement in genital arousal has also been demonstrated through laboratory manipulations designed to increase SNS activity. Anxiety-evoking films, thought to increase SNS activity, increase vaginal blood volume (VVBV) during subsequent erotic films in functional and dysfunctional women (e.g., Palace & Gorzalka, 1990). But more recent research has shown a curvilinear relationship between acute anxiety and VVBV response, with the optimal arousal response occurring at a moderate level of anxiety (Bradford & Meston, 2006).

Similarly, several studies on the effects of exercise (e.g., Meston & Gorzalka, 1995, 1996) and ephedrine (Meston & Heiman, 1998) on sexual arousal support the notion of an optimal level of SNS arousal necessary for adequate genital arousal. Interference with normal SNS activity, through stress for example, can influence a woman's ability to become aroused. In women with orgasm disorders, this optimal level of SNS arousal may be disrupted; for example, women with orgasm difficulties respond to increased SNS activity with a reduction in physiological sexual response (Meston & Gorzalka, 1996).

Medical and Cardiovascular Factors

Endocrine-Related
Many medical treatments and/or conditions that interfere with endocrine function can alter the female sexual arousal and orgasm. Hyperprolactinemia, a condition in which abnormally high amounts of prolactin are secreted, interferes with sexual arousal
Problems with Arousal and Orgasm in Women

and orgasm: subjective measures of sexual functioning as measured by the Female Sexual Functioning Index (FSFI). Rosen et al. (2000) report that women with hyperprolactinemia compared to control women, and FSFI arousal and orgasm domain scores are negatively correlated with prolactin levels (Kadioglu et al., 2005). High levels of prolactin inhibit gonadotropin releasing hormone (GRH; Sauier, Frager, Case, Kelch, & Marshall, 1984) thus, in turn, inhibits hormone release further down the hypothalamo-pituitary-gonadal (HPG) axis.

Other endocrine-related disorders may also affect women’s sexual response. Women with diabetes mellitus show various sexual problems, including sexual arousal dysfunction, reduced vaginal lubrication, and inability to reach orgasm (Salonia, Briganti, Briganti, & Montorsi, 2006). The mechanisms by which the insulin abnormalities associated with diabetes affect the rest of the endocrine system are not known. Bilateral oophorectomy, which results in a reduction of estrogen and testosterone secretion, also causes problems with arousal through mechanisms discussed in the previous section on endocrine factors. Finally, women who have undergone hysterectomy often have long-term difficulties attaining orgasm and vaginal lubrication (Jensen et al., 2004).

Cardiovascular Disease

A connection between coronary artery disease and erectile dysfunction has been well established in men (for a review, see Russell, Khandheria, & Nehra, 2004), but less is known about the effects of cardiovascular disease on women’s sexual functioning. One comprehensive study showed that women with coronary artery disease reported clinically significant problems with arousal, lubrication, and orgasm compared to healthy controls (Salonia, Briganti, & Montorsi, 2002 as cited in Salonia et al., 2006). Women with hypertension have a general impairment of physiological response and, because normal blood flow to the genitals is restricted, problems in attaining adequate genital arousal. Hypertensive women have been shown to have reduced lubrication, reduced frequency of orgasms, and greater difficulty attaining orgasm (L. E. Duncan et al., 2001). Regarding women with PSAD, an Internet survey reported that medical problems (diabetes, stroke, hypothyroidism, seizure disorders, myocardial infarctions, angina pectoris) occurred in less than 10% of women believing they had PSAD (Leiblum et al., 2005).

Physical/Mobility Related

An ailment that impinges on a patient’s ability to move or feel sensation can interfere with sexual arousal. Both multiple sclerosis (MS) and spinal cord injuries (SCI) have been linked to difficulties in attaining orgasm, although both arousal and orgasm are possible in these women (e.g., Salonia et al., 2006; Sipinski, Alexander, &
Rosen, 1995b). SCI and control women respond differently to erotic visual stimuli. Control women show both increased genital and subjective arousal to erotic visual stimuli whereas women with SCI show only increased subjective arousal. Only when tactile clitoral stimulation is applied do SCI women respond with an increase in genital arousal. Tactile stimulation alone (without erotic visual stimuli) in women with SCI does not increase subjective sexual arousal because the neurological pathway mediating this response via the brain is interrupted by the injury (Sipski, Alexander, & Rosen, 1995a). Women with SCI at T6 and below have been able to achieve orgasm 52% of the time in a laboratory study, compared to 100% of healthy controls (Sipski et al., 1995b). Women with SCI in the sacral region—thus interfering with the sacral reflex arc—show the most difficulty attaining orgasm (Sipski, Alexander, & Rosen, 2001). Data from human and animal studies have led to the suggestion that the vagus nerve connection between the cervix of the uterus and the brain is key in maintaining SCI patient's ability to experience orgasm (Whipple, Gerdes, & Komisaruk, 1996).

Drug-Related

Some antidepressants, especially ones that increase serotonin such as the SSRIs, have an inhibitory influence on libido and orgasmic functioning (Rosen, Lane, & Menza, 1999). Patients using the antidepressants bupropion, moclobemide, and nefazodone are less likely to report orgasm difficulties than patients using SSRIs. The latter increases levels of norepinephrine and serotonin, while the former two increase the levels of NE and dopamine (DA), in addition to serotonin. The increase in NE and DA seems to reduce the interference in arousal and orgasm caused by increased serotonin (Meston et al., 2004). Consistent with this, mirtazapine, an SSRI with noradrenergic effects, has also been shown to reduce the orgasmic difficulties experienced by depressed patients (Bovartsky, Haque, Rouleau, & Hirschfeld, 1999). Of the SSRIs, paroxetine has the strongest inhibiting effect on orgasm (Bobes et al., 2002; Montejo-Gonzalez et al., 1997), possibly due to its stronger effect on the serotonin transporter and lack of effect on the dopamine transporter (Rosen et al., 1999). In an Internet survey of women who met at least one criterion for PSAD, 48% of respondents answered the question about "use of antidepressants," and all of these respondents answered affirmatively regarding their use (Leiblum et al., 2005).

Several antipsychotic drugs have also been shown to inhibit or delay orgasm in women. Trihexyphenidyl, fluphenazine, and thioridazine interfere with orgasm (Gladstein, Chouinard, & Annable, 1982; Shen & Sata, 1990) whereas haloperidol and clozapine do not (Hummer et al., 1999). Antipsychotics may cause orgasmic difficulties either directly by blocking dopamine receptors or indirectly by increasing prolactin levels or causing se-
Psychological Factors, Sexual Arousal, and Orgasm

Psychological factors also play an important role in women's sexual arousal and orgasm. Among these broadly classified factors are relationship issues, cognitive and affective factors, and cultural and societal factors.

Relationship Issues

Higher orgasm frequency and level of arousal have been linked to marital satisfaction (Hurlbert, Apt, Hurlbert, & Pierce, 2000), while problems with arousal and orgasm have been linked to marital difficulties and relationship dissatisfaction (e.g., Laumann, Paik, & Rosen, 1999). Couples reporting sexual difficulties, compared to nonclinical control couples, have less satisfaction in their relationships (e.g., Chesney, Blakerney, Cole, & Chan, 1981), an increased number of disagreements (Chesney et al., 1981), more communication and conflict resolution problems (e.g., Chesney et al., 1981; Ernst, Foldényi, & Angst, 1993), and more sexual communication problems (e.g., Chesney et al., 1981), including discomfort discussing sexual activities related to their particular sexual difficulty (Kelly, Strassberg, & Kircher, 1990). Within their relationship interactions, they also display less playfulness and spontaneity (Metz & Lutz, 1990), less closeness, intimacy, and feelings of mutual love, and more aversive feelings and thoughts regarding their interactions (Birnbaum, Glaubman, & Mikulincer, 2001). Warmth, caring, and affection within the relationship have also been linked to increased sexual arousal (Persky et al., 1982), while conflict and hostility have been linked to lower orgasmic responses (Świerczkowski & Walker, 1978). However, overall sexual satisfaction for women is determined more by closeness with one's partner than by the number of orgasms or sexual arousal (Hurlbert, Apt, & Rabehl, 1994).

Cognitive and Affective Factors

Depression and Anxiety

There is a strong relationship between depression and impaired sexual arousal and orgasm (e.g., Kennedy, Dickens, Lischied, & Bagby, 1999; Leiblum et al., 2005). High levels of anxiety have
also been reported among sexually dysfunctional women (see Norton & Jehn, 1984 for a review), and high levels of sexual difficulties have been reported among women with anxiety disorders (e.g., Bodine et al., 2002). The specific type of anxiety disorder may play an important role; for instance, women with obsessive-compulsive disorder have more difficulties with orgasm than women with generalized anxiety disorder (Aksaray, Yelken, Kaptanjılu, Ollu, & Ozalpınar, 2001).

Findings on anxiety and sexual response in laboratory studies show mixed results. Anxiety-inducing stimuli prior to erotic visual stimulation have been shown to increase physiological sexual response in sexually functional women (e.g., Laan, Everaerd, van Aanholt, & Rebel, 1993) and dysfunctional women (e.g., Palace & Gorzalka, 1992), but such stimuli may increase, decrease, or not affect subjective sexual arousal (for a review, see Meston & Bradford, in press). The variable findings regarding the effects of anxiety-inducing stimuli on subjective arousal may be attributable to different definitions of anxiety across studies. State anxiety can acute emotional response that can be easily manipulated, but not trait anxiety (a relatively stable measure that reflects one’s dispositional tendency to experience state anxiety), is negatively linked to subjective sexual arousal in response to erotic stimuli (Bradford & Meston, 2006). In terms of genital arousal, as noted earlier, the relationship between state anxiety and physiological sexual arousal is curvilinear, such that moderate levels of state anxiety facilitated, and high levels of state anxiety impaired, vaginal arousal.

Based on an Internet survey of women who met at least one criterion for PSAD, stress and anxiety were the most common triggers of PSAD symptoms, reported by approximately 46% and 34% of women, respectively. Furthermore, approximately 62% of all women described themselves as worriers, 68% reported that they “carried a lot of stress in their body,” 31% reported having anxiety or panic attacks, and 22% reported having obsessive thoughts or behaviors (Leiblum et al., 2005).

**Cognitive Distraction and Self-Focused Attention**

Barlow’s model of sexual functioning implicates cognitive interference in the cause and maintenance of sexual difficulties through a shift of attention from erotic cues to internal negative self-evaluative cues (Barlow, 1986). Consistent with this model are findings showing that cognitive distraction can impair the sexual response in women (e.g., Koukounas & McCabe, 1997), as well as studies linking “trait private self-consciousness” (i.e., tendency to focus on internal bodily sensations) with enhanced sexual functioning (Meston, 2006) and “state self-focused attention” (i.e., instructed focus on oneself) to impaired physiological sexual arousal response in sexually functional (Meston, 2006)
women. However, in response to erotic stimuli among sexually dysfunctional women who were made to self-focus on their appearance, subjective sexual response was found to be unchanged (Meston, 2000) or enhanced (Sear & Meston, 2007), suggesting that the way a woman feels about her body and body image may be important factors in understanding sexual arousal (e.g., Wiederman, 2000).

Women experience a variety of concerns during sexual activity, including worries about pleasing their partner, their ability to reach orgasm, becoming pregnant, contracting sexually transmitted infections, and losing their partner because of sexual problems. Women who experience orgasmic disorder have particularly strong anxieties related to intercourse (Birmann, 2003) and tend to blame themselves rather than external factors for their difficulty (Loos, Bridges, & Critelli, 1987).

**Personality Characteristics**

Women with sexual dysfunctions have higher extraversion (Anderson & Cypriano, 1994) and neuroticism scores (e.g., Costa, Fegan, Piedmont, Portnàas, & Wise, 1992; Hartmann, Keiser, Rüfer, Hesse, & Kloth, 2002). Neuroticism has also been linked to lower sexual satisfaction among women (Costa et al., 1992) and to poorer sexual adjustment among college-aged women (Meston, Trapnell, & Gorzalka, 1993). Women diagnosed with mixed sexual difficulties (desire, arousal, and orgasm) are often characterized by lower extraversion and lower openness to experience than women without sexual difficulties (Hartman et al., 2002). Compared to women without a personality disorder, women diagnosed with histrionic personality disorder exhibit more orgasmic dysfunction, despite having higher sexual esteem (Apt & Hurlbert, 1994). In contrast, general openness and sensation seeking have been linked to increased sexual functioning and arousability, respectively (Apt & Hurlbert, 1992; Costa et al., 1992).

**Sexual Abuse**

Sexual abuse in childhood and/or adulthood can result in negative affect during physiological arousal (L. A. Berman, Berman, Bruck, Fawar, & Goldstein, 2001) and reduced feelings during sexual activity (Berman & Breslow, 1997). As noted earlier, in sexually healthy women, physiological sexual arousal is enhanced with SNS activation (via exercise; Meston & Gorzalka, 1995). In contrast, in women with a history of childhood sexual abuse, physiological arousal is not enhanced with SNS activation and, in fact, may be impaired (Rellini & Meston, 2006). This difference may reflect disruptions in endocrine function—in particular the hypothalamus-pituitary-adrenal axis—known to exist in women with a history of childhood sexual abuse and comorbid posttraumatic stress disorder (Rellini & Meston, 2006). Sexual arousal difficulties following...
sexual abuse may also be related to a misinterpretation of physical sensations, such as heart rate and lubrication, with women associating these sensations during sexual activity with similar negative traumatic responses experienced during the early sexual abuse experience (Rekin, 2006).

Cultural and Societal Factors

Research suggests cultural differences in sexual functioning (e.g., Brotto, Chik, Ryder, Gorzalka, & Seal, 2005). Sexual satisfaction (e.g., Fugl-Meyer & Fugl-Meyer, 1999), knowledge about and attitudes toward sexuality (e.g., Meston, Trumel, & Gorzalka, 1996), and sexual experiences such as age of first intercourse and rates of masturbation (e.g., Tang, Lai, & Chung, 1997). In a study of over 3,000 women from a variety of ethnic groups, Cain et al. (2003) found less frequent physical pleasure reported by Hispanic, Chinese, and Japanese women than Caucasian women, and less frequent sexual arousal reported among all ethnic groups, including African American women, than among Caucasian women.

Aging

An abundance of research shows a decline in normal female sexual functioning with age, including decreased frequency of orgasm (e.g., Adams & Turner, 1985) and impaired sexual arousal (e.g., Cain et al., 2003). While this decline may be partly related to menopause, pre- and peri-menopausal women aged 42 to 52 years do not differ in their experience of physical pleasure and arousal (Cain et al., 2003), suggesting that other factors are involved, such as an age-related decrease in sexual communication (Deeks & McCabe, 2001) and in the relative importance of sex (e.g., Bergstrom-Walan & Neilson, 1990).

In summary, the following points regarding the role of psychological factors in sexual arousal and orgasm in women can be made:

- Difficulties with orgasm and/or arousal have been linked to sexual and nonsexual difficulties within the relationship.
- A strong relationship exists between depression and impaired sexual arousal and/or orgasm.
- State, but not trait, anxiety may be linked to decreased subjective sexual arousal.
- Based on findings from an Internet survey, stress and anxiety are reported by a large portion of women experiencing PSAD symptoms.
- There is a curvilinear relationship between state anxiety and physiological sexual arousal, such that moderate levels of
state anxiety facilitate arousal the most, and high levels impair arousal.

- Factors that cause cognitive distraction during sexual activity, such as body image concerns, may play a role in the cause and maintenance of sexual difficulties.

- Personality factors may be linked to sexual response (neuroticism, lower extraversion, and lower openness are linked to decreased sexual functioning).

- Sexual abuse can result in orgasm and arousal difficulties and may be related to misinterpretation of the physical sensations during sexual activity.

- Research shows substantial cultural differences across sexuality measures.

- Age-related decline in sexual arousal and frequency of orgasm in women may be partially attributable to factors such as menopause and changes in sexual communication.

### Assessment

The presenting problem of the woman should first be ascertained in a clinic interview. She should be asked to describe her difficulty in her own words. To provide a more comprehensive structured assessment, the questions in Table 7.2, which expand on Basson, Weijmar Schultz, et al. (2004), Brandenburg and Schwenkhagen (2006), and Perelman (2006), may be used.

Self-reported information from the woman collected through interviews, questionnaires, and sexual behavior logs are suitable methods to assess and monitor changes in female sexual dysfunction (Rosen, 2002). Techniques may include logs of objective ratings of sexual response, such as orgasm and lubrication, and questionnaires assessing sexual functioning (e.g., The Female Sexual Function Index [FSFI]; Rosen et al., 2000; The Brief Index of Sexual Functioning for Women [BISF-W]; Taylor, Rosen, & Leiblum, 1994), sexual satisfaction (e.g., The Sexual Satisfaction Scale [SSS-W]; Meston & Trapnell, 2005), and sexual beliefs (e.g., The Derogatis Interview for Sexual Functioning, Sexual Beliefs Subscale: Derogatis, 1997). Some of these reliably differentiate women with FSAD, FOD, and no dysfunction (e.g., SSS-W; Meston & Trapnell, 2005; FSFI: Meston, 2003) and are sensitive to change over time (e.g., BISF-W: Shifren et al., 2000).

As outlined in Table 7.3, the practitioner should also assess relationship and sexual history, psychosocial history, and medical history. Questionnaires measuring relationship functioning include:
<table>
<thead>
<tr>
<th>Criteria</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degree of distress</td>
<td>What do these symptoms mean for her?</td>
</tr>
<tr>
<td></td>
<td>What has her reaction been?</td>
</tr>
<tr>
<td>Onset of problem</td>
<td>Has the problem been lifelong or was it acquired?</td>
</tr>
<tr>
<td></td>
<td>If acquired:</td>
</tr>
<tr>
<td></td>
<td>-What is her belief about the causes of the change? (e.g., major life stresses, change in medication use)</td>
</tr>
<tr>
<td></td>
<td>-What has the progression been since the onset?</td>
</tr>
<tr>
<td>Context of problem</td>
<td>Is the problem situational or generalized?</td>
</tr>
<tr>
<td></td>
<td>How has it been with other partners?</td>
</tr>
<tr>
<td></td>
<td>How is it when she is alone?</td>
</tr>
<tr>
<td></td>
<td>Does she experience this difficulty all the time or only under specific circumstances?</td>
</tr>
<tr>
<td>Frequency</td>
<td>Out of 10 times engaging in intercourse, how often does she experience this difficulty?</td>
</tr>
<tr>
<td></td>
<td>How has this changed over time?</td>
</tr>
<tr>
<td>Other sexual problems</td>
<td>Are there any other sexual problems present?</td>
</tr>
<tr>
<td></td>
<td>Does her partner have sexual dysfunction?</td>
</tr>
<tr>
<td>Nonsexual factors</td>
<td>What are the cognitive and emotional factors involved?</td>
</tr>
<tr>
<td></td>
<td>What are the woman's and her partner's responses to her sexual difficulty?</td>
</tr>
<tr>
<td></td>
<td>What does the partner think the cause of the problem is?</td>
</tr>
<tr>
<td>Prior treatment</td>
<td>Have any prior treatments been attempted?</td>
</tr>
<tr>
<td></td>
<td>If yes, what was the outcome?</td>
</tr>
<tr>
<td>Arousal questions</td>
<td>Mental excitement—How is it when she is alone (e.g., reading erotic) versus when she is stimulating her partner versus when her partner stimulates her?</td>
</tr>
<tr>
<td></td>
<td>Does she experience genital sensations (e.g., tingling, swelling, pulsing) and to what degree?</td>
</tr>
<tr>
<td></td>
<td>Is her genital lubrication completely absent, inadequate, or does it disappear?</td>
</tr>
<tr>
<td>Orgasm questions</td>
<td>Is orgasm absent, delayed, or of reduced intensity?</td>
</tr>
<tr>
<td></td>
<td>What is her frequency of masturbation?</td>
</tr>
<tr>
<td></td>
<td>How often does she experience sexual fantasy?</td>
</tr>
</tbody>
</table>
### Assessment of Patient History

**Sexual and Relationship History**

<table>
<thead>
<tr>
<th>Category</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexual history</td>
<td>Ask participant to describe her first sexual experiences.</td>
</tr>
<tr>
<td></td>
<td>What was her family's attitude toward sex?</td>
</tr>
<tr>
<td></td>
<td>Is there any history of sexual violence or trauma?</td>
</tr>
<tr>
<td>Relationships</td>
<td>What is the nature and duration of current and past relationships (sexual and in general)?</td>
</tr>
</tbody>
</table>

**Psychosocial History**

<table>
<thead>
<tr>
<th>Category</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children</td>
<td>Does the woman have children?</td>
</tr>
<tr>
<td></td>
<td>If yes, how many and what are their ages?</td>
</tr>
<tr>
<td>Work/Finance</td>
<td>How does the woman feel about her job if she has one?</td>
</tr>
<tr>
<td></td>
<td>Are there any stresses surrounding her finances?</td>
</tr>
<tr>
<td>Other stressors</td>
<td>Are there any other stressors or factors that would influence her sexual functioning?</td>
</tr>
<tr>
<td>Mental health</td>
<td>Measurements of mental health in the patient and her partner may be helpful (e.g., The Beck Depression Inventory: Beck, Ward, Mendelson, Mock, &amp; Erbaugh, 1961). What religious, societal, family of origin, cultural values, beliefs, or restrictions may be affecting sexual functioning?</td>
</tr>
</tbody>
</table>

**Medical History (Information Needed about Historical and Current Medical Factors)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Illness</td>
<td>Cervical or breast cancer</td>
</tr>
<tr>
<td></td>
<td>Diabetes</td>
</tr>
<tr>
<td></td>
<td>Prolactinemia</td>
</tr>
<tr>
<td></td>
<td>Multiple Sclerosis</td>
</tr>
<tr>
<td></td>
<td>Spinal cord injury</td>
</tr>
<tr>
<td></td>
<td>Brain injury</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Medications and treatments</td>
<td>Antidepressants</td>
</tr>
<tr>
<td></td>
<td>Psychotropic drugs</td>
</tr>
<tr>
<td></td>
<td>Anti-epileptics</td>
</tr>
<tr>
<td></td>
<td>Radiation</td>
</tr>
<tr>
<td></td>
<td>Chemotherapy</td>
</tr>
<tr>
<td></td>
<td>Antihypertensives</td>
</tr>
<tr>
<td>Emotional factors</td>
<td>Depression</td>
</tr>
<tr>
<td></td>
<td>Anxiety and stress</td>
</tr>
<tr>
<td></td>
<td>Childhood sexual abuse</td>
</tr>
</tbody>
</table>
The Dyadic Adjustment Scale (Spanier, 1976), the Relationship Beliefs Scale (Fletcher & Kimmons, 1992), and the Locke-Wallace Marital Adjustment Scale (Locke & Wallace, 1959).

A full physical exam is also recommended for all complaints of sexual function (for details, see Stewart, 2006). In addition to ruling out or identifying various medical factors, the exam serves to educate women about their anatomy, and what is normal or problematic.

**Pelvic Exam**

A pelvic exam is recommended for women who have been diagnosed with FSAD or FOD. However, unless the woman has specifically complained of pain during sexual arousal or intercourse or is post menopausal, usually no organic problems are associated with these sexual disorders. For women with a subjective arousal disorder or FOD, a pelvic exam can help educate them about their genitals, and information discounting the existence of organic problems may help alleviate concerns for many patients (Stewart, 2006). For women with genital arousal disorder, the exam may be able to reveal an estrogen deficiency, pH imbalance, or structural or organic problem such as vaginal atrophy (e.g., Patel et al., 2006).

**Genital Arousal**

Vaginal blood volume (VBB), genital engorgement, genital lubrication, and sexual sensations such as throbbing and tingling are important components of physical sexual arousal in women. Women with subjective sexual arousal disorder have VBB and vaginal pulse amplitude (VPA) responses similar to controls; so assessment of these measures would not be useful for diagnosing subjective FSAD as part of a routine exam. For women who have genital arousal disorder, where there is a possibility of a vascular genital problem, assessment of genital response could be informative. Genital arousal is most commonly assessed in research studies using vaginal photoplethysmography to determine VPA and, less commonly, VBB. This methodology for assessing genital response is not as effective for clinical assessment, however, because of the wide variance in VPA response among women—making it difficult to establish a “normal response”—and because vaginal photoplethysmography is not readily available outside of research settings. On the other hand, the use of VPA measurements can be useful for within-person assessment, for example, before and after treatment or to gauge changes in response to various components of sexual stimuli (Pransky & Janssen, 2006). Genital arousal can also be measured through IMRJ of the genital area. Although
MR) machines are now more readily available, the high cost remains an obstacle (Maravilla, 2006). Doppler ultrasound is a relatively new tool for assessing VVB. This technology is useful because it is not affected by movement, so participants can masturbate during the procedure, allowing examination of orgasmic response in addition to arousal (Garcia Nader, Matland, Munarritz, & Goldstein, 2006).

Hormone Assays

If a hormonal problem is suspected, practitioners can request assays for prolactin, total testosterone, free testosterone, sex hormone binding globulin (SHBG), DHEA, estrogens, and cortisol to ensure that all levels are within a reasonable range. Androgens should be measured during peak times in the morning on days 8 to 18 of the menstrual cycle. A limiting factor in diagnosis is a lack of norms for androgen levels in women, mainly due to a lack of sensitivity of most assays at the low end of the scale.

Treatment

General Psychological Treatments

Education, communication training, and cognitive behavioral techniques such as sensate focus and systematic desensitization have been successful in treating female sexual dysfunctions (see Sidebar 7.3). Sensate focus, introduced by Masters and Johnson in the 1970s, is a cognitive behavioral technique in which couples learn to focus on the pleasurable sensations brought about by touching, and to decrease the focus on goal directed sex (e.g., orgasm). Couples are first instructed to explore their partner’s non-sexual body regions without the potential for sexual activity, gradually moving to the next phase in which the female guides her partner in genital stimulation and sexual positions that enhance her arousal. Intercourse is incorporated only in the final stages when both partners feel comfortable. The effectiveness of sensate focus by itself on sexual dysfunction has not been examined in randomized, controlled research. Results from studies using sensate focus as well as treatment techniques such as increasing sexual skills, lowering sexual and performance anxiety, and addressing cognitions and behaviors have shown success among women with FOD and/or FSAD (McCabe, 2001), with the percentage of women who experienced FOD and FSAD dropping from 66.7% and 33.3% pretreatment to 11.1% and 14.8%, respectively, posttreatment.
SIDEBAR 7.3

Psychological Approaches to Sexual Arousal and Orgasm Disorders

Education and communication training are useful components of treatment for orgasm and/or arousal difficulties.

Sensate focus is a cognitive-behavioral technique used to treat both orgasm and arousal difficulties.

Systematic desensitization, involving relaxation and working through a hierarchy of anxiety-provoking stimuli, decreases anxiety surrounding sexual activity and may improve some aspects of sexual functioning.

Systematic desensitization involves deep relaxation exercises that enable the woman to replace fear responses with relaxation responses. A succession of anxiety-provoking stimuli is developed by the woman and the therapist to represent increasingly threatening sexual situations. For example, a hierarchy can be developed ranging from the least anxiety-provoking stimuli of lying naked next to one’s partner to the most anxiety-provoking stimuli of experiencing an orgasm following a partner’s request. The woman’s task is to approach each set of stimuli on her hierarchy and experience fearful to relaxed responses, resulting in a net decrease of anxiety. She moves up her hierarchy gradually, tackling items of increasing intensity over time. After the woman can successfully imagine each anxiety-provoking item from her hierarchy without anxiety, she engages in the actual activities of each item on her hierarchy until her anxiety is decreased.

Outcome studies for sensate focus and systematic desensitization show that these techniques can improve some aspects of sexual functioning, although the most reliable improvement is a decrease in sexual anxiety. No well-controlled studies have shown a clinically significant increase in orgasmic function after sensate focus or systematic desensitization training alone. Meston et al. (2004) have recommended that these techniques be used to treat FOD only when concurrent sexual anxiety problems exist.

Treatments Specific to FSAD/PSAD

Pharmacological Treatments

Currently no medications have been approved by the U.S. Food and Drug Administration for the treatment of FSAD. However, several placebo-controlled studies suggest that selective PDE-5 inhibitors (e.g., sildenafil or Viagra™) may be an effective treat-
ment for difficulties stemming from perceptions of physical sensations and physiological aspects of FSAD. Findings indicate a variety of positive effects. These include improved genital sensation, vaginal lubrication, satisfaction with intercourse, clitoral sensitivity, and overall sexual experience among postmenopausal women with FSAD (J. R. Bernian et al., 2003); increased subjective sexual arousal, perception of genital arousal, and reduced latency to orgasm among postmenopausal women with FSAD and FOD who had low physiological sexual arousal responses to erotic stimuli (Basson & Brotto, 2003); increased self-reported sexual arousal, orgasm, sexual fantasy, intercourse, and enjoyment of sexual activity among premenopausal women with FSAD and FOD (Caruso et al., 2001); and improved vaginal engorge ment among sexually functional premenopausal women with FSAD (Laan et al., 2002). The combined administration of the nitric oxide-precursor L-arginine and the alpha 2-blocker yohimbine in postmenopausal women with FSAD also improves genital arousal compared to placebo, despite no specific effect on subjective arousal (Meston & Worcel, 2002).

In a study comparing sustained-release bupropion to placebo in premenopausal women with hypoactive sexual desire disorder (HSDD), bupropion had no effect on desire but did improve self-reported sexual arousal, ability to attain orgasm, and sexual satisfaction, suggesting that this drug may be helpful in some women suffering from arousal and/or orgasm difficulties (Segraves, Clayton, Crotli, Wolf, & Wannock, 2004).

Drugs that act as vasodilators also appear to improve sexual response in women. For example, increases in self-reported vaginal lubrication and pleasurable sensations in the vagina, along with trends toward increased VPA, have also been found with oral phentolamine in postmenopausal women with FSAD. Compared with placebo, vaginally applied phentolamine increases physiological and subjective sexual arousal among postmenopausal women using hormone replacement therapy (Rubio-Aurioles et al., 2002). In an uncontrolled study, topical alprostadil cream increased labial and clitoral engorgement among women with FSAD and FOD assessed with Doppler ultrasonography (Becher, Bechara, & Casabe, 2001), but later research found no effect beyond placebo in women with FSAD, assessed with vaginal photoplethysmography and self-report questionnaires (Islam et al., 2001; Padma-Nathan et al., 2003).

The dopaminergic agonist apomorphine increases subjective sexual arousal in premenopausal women with FSAD and hypoactive sexual desire disorder (HSDD; Caruso et al., 2004), and tibolone, a Selective Estrogen Receptor Modulator (SERM), improves genital response and frequency of arousability and sexual fantasies in postmenopausal women (Laan, van Lunsen, & Everard, 2001).
Testosterone may increase genital arousal in healthy premenopausal women (Flutten et al., 2002). Clinical trials have shown improvement in sexual interest, desire, activity, and satisfaction following testosterone administration. As noted earlier, DHEA has no significant impact on premenopausal women (Meston & Heiman, 2002), but appears to impart positive effects for postmenopausal and older over 70 women, including increased subjective sexual arousal (Hackett & Heiman, 2002), physical sexual excitement, sexual activity, and sexual satisfaction (Baulieu et al., 2000). Treatment of sexual dysfunction with hormone therapy is inappropriate for premenopausal women who have ovulatory cycles, as evidence of effectiveness and safety is currently lacking (Davis, 2006).

Other Treatment
The FDA approved the EROS clitoral therapy device (CTD: Urometrics, St. Paul, MN, United States) for use in women with FSAD following a noncontrolled study showing that the device, which increases vasocongestion through suction, increased vaginal lubrication, sensation, orgasm, and overall sexual satisfaction (Billups et al., 2001).

Based on an Internet survey of 103 women experiencing at least one symptom of FSAD, orgasms were reported to provide some relief in almost half of all women, eliminating symptoms in approximately 13% of women. However, a mean of 5.2 orgasms were necessary to quell the feelings of genital arousal, and many women viewed the process as either painful or physically distressing. Other activities reported to provide relief of symptoms included taking medication (52%), distraction (39%), intercourse (36%), physical exercise (25%), and using cold compresses (13%: Leiblum et al., 2005).

Treatments Specific to FOD
Pharmacological Treatments
So far, no pharmacological treatments for FOD have been found more effective than placebo (for a review, see Meston et al., 2004). If the patient is taking SSRIs and the orgasmic difficulties coincide with the onset of the drug treatment, practitioners may recommend a change in prescription to an antidepressant that also affects DA and NE. These include bupropion, nefazodone, and moclobemide. Mirtazapine also improves orgasmic abilities compared to other antidepressants, but one study reported a 50% drop out rate due to side effects such as drowsiness (Boyarshi et al., 1999).

Nonpharmacological Treatments
Directed masturbation (DM) uses cognitive-behavioral therapy to educate a woman about her body and the sensations she is
able to elicit while manually stimulating herself. First, a woman engages in a visual exploration of her body, using a mirror and educational material depicting female genital anatomy. Following visual and manual identification of the sensitive genital areas that elicit pleasure, the woman is instructed to apply targeted manual stimulation to these regions. Allowing a woman to explore her body on her own is beneficial because it eliminates several factors that may be barriers to orgasm, including anxiety that may be associated with the presence of a partner. Once a woman is able to attain orgasm with masturbation, her partner may become involved in the DM sessions. Women experiencing FOD have successfully been treated using DM in a variety of therapy settings with success rates as high as 100% in one study (for a review of studies demonstrating the efficacy of DM, see Meston et al., 2004; for a detailed guide to DM, refer to Heiman & LoPiccolo, 1988).

Kegel exercises have been included as part of the treatment regimen for FOD but studies that have looked solely at the effect of Kegel exercises on orgasmic ability have found no substantial improvement (Table 7.4; Chambless et al., 1984).

Summary and Conclusions

The most efficacious treatments for FSAD are the EROS clitoral device; behavioral and cognitive techniques, including sensate focus, systematic desensitization, and anxiety reduction; and communication training and education. While no pharmacologic medications have been approved by the FDA for the treatment of FSAD, several hormonal and nonhormonal agents have shown benefit over placebo. There is no specific therapy for PSAD. For FOD, the most effective treatment seems to be directed masturbation, which involves educating the woman and her partner about her body, and increasing the familiarity and comfort level of a woman with her body. No pharmacological agents have been found to directly increase orgasmic ability.

In summary:

- No pharmacological treatments for FOD are available, but a woman taking SSRIs may change to antidepressant medication that also targets dopamine and norepinephrine.
- Directed masturbation can help a woman learn about her body and is beneficial for increasing orgasmic ability.
- Kegel exercises help strengthen pelvic floor muscles, which can aid in orgasm, but research evidence is lacking.
Table 7.4  Major Diagnostic Points and Treatment Options

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Diagnostic Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSAD</td>
<td>Subjective, genital, or combined?</td>
</tr>
<tr>
<td>FOD</td>
<td>Lifetime or acquired?</td>
</tr>
<tr>
<td></td>
<td>Completely absent, diminished intensity, and/or marked delay?</td>
</tr>
<tr>
<td>Both</td>
<td>Assess degree of distress</td>
</tr>
<tr>
<td></td>
<td>Assess other factors possibly involved:</td>
</tr>
<tr>
<td></td>
<td>- Relationship factors</td>
</tr>
<tr>
<td></td>
<td>- Emotional factors (depression, sexual abuse)</td>
</tr>
<tr>
<td></td>
<td>- Health/medical factors</td>
</tr>
<tr>
<td></td>
<td>- Medication use (e.g., SSRI)</td>
</tr>
<tr>
<td></td>
<td>- Age</td>
</tr>
<tr>
<td></td>
<td>- Menopause</td>
</tr>
<tr>
<td></td>
<td>- Endocrine factors</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSAD</td>
<td>Pharmacological treatments (none currently approved by FDA):</td>
</tr>
<tr>
<td></td>
<td>- Selective phosphodiesterase type 5 inhibitors (post and premenopausal women; genital and subjective sensations, orgasm, and physiological aspects)</td>
</tr>
<tr>
<td></td>
<td>- Nitric oxide-precursor L-arginine (postmenopausal women; physiological arousal)</td>
</tr>
<tr>
<td></td>
<td>- Alpha blocker yohimbine (postmenopausal women; physiological arousal)</td>
</tr>
<tr>
<td></td>
<td>- Bupropion sustained release (premenopausal women; subjective arousal, ability to attain orgasm)</td>
</tr>
<tr>
<td></td>
<td>- Oral phenotolamine (postmenopausal women; genital and subjective sensations and physiological aspects)</td>
</tr>
<tr>
<td></td>
<td>- Dopaminergic agonist apomorphine (premenopausal women; subjective reports of arousal)</td>
</tr>
<tr>
<td></td>
<td>- Selective Estrogen Receptor Modulator Thiolone (postmenopausal women; subjective and physiological aspects)</td>
</tr>
<tr>
<td></td>
<td>- Testosterone</td>
</tr>
<tr>
<td></td>
<td>- DHEA (postmenopausal women; subjective arousal)</td>
</tr>
</tbody>
</table>

**Note:** It is suggested that treatment of hormone therapy is inappropriate for premenopausal women who have ovulatory cycles, as evidence of effectiveness and safety data are currently lacking (Davis, 2006).

<table>
<thead>
<tr>
<th>FOD</th>
<th>Directed masturbation (DM):</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Visual exploration of own body with mirror</td>
</tr>
<tr>
<td></td>
<td>- Manual identification of sensitive areas that elicit pleasure</td>
</tr>
</tbody>
</table>
Table 7.4 (Continued)

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual stimulation by patient</td>
<td></td>
</tr>
<tr>
<td>Once orgasm is attained, involve partner in DM sessions</td>
<td></td>
</tr>
<tr>
<td>Refer to <em>Becoming Orgasmic: A Sexual and Personal Growth Program for Women</em> by Heiman &amp; LoPiccolo, 1988</td>
<td></td>
</tr>
<tr>
<td>Kegel exercises</td>
<td></td>
</tr>
<tr>
<td>Both</td>
<td></td>
</tr>
<tr>
<td>Education:</td>
<td></td>
</tr>
<tr>
<td>- Existence of difficulties</td>
<td></td>
</tr>
<tr>
<td>- Information about genitals</td>
<td></td>
</tr>
<tr>
<td>Relationship counseling (e.g., to increase communication about sexuality)</td>
<td></td>
</tr>
<tr>
<td>Treatment of underlying problems (e.g., medical factors, emotional difficulties)</td>
<td></td>
</tr>
<tr>
<td>Hormone treatment if hormonal problem is suspected</td>
<td></td>
</tr>
<tr>
<td>Sensate focus:</td>
<td></td>
</tr>
<tr>
<td>- Decrease goal-directed sex (e.g., attainment of orgasm)</td>
<td></td>
</tr>
<tr>
<td>- Focus on pleasurable sensations</td>
<td></td>
</tr>
<tr>
<td>- Explore partner's nonsexual body</td>
<td></td>
</tr>
<tr>
<td>- Gradually move to sexual exploration and intercourse when both partners comfortable</td>
<td></td>
</tr>
<tr>
<td>Systematic desensitization (if sexual anxiety is present):</td>
<td></td>
</tr>
<tr>
<td>- Relaxation techniques</td>
<td></td>
</tr>
<tr>
<td>- Create and work through fear/anxiety hierarchy</td>
<td></td>
</tr>
<tr>
<td>EROS clitoral therapy device (FDA approved)</td>
<td></td>
</tr>
<tr>
<td>- Increases vaginal lubrication, sensation, orgasm, and overall sexual satisfaction</td>
<td></td>
</tr>
</tbody>
</table>

References


Catt, V. S., Johannes, C. B., &_subsequent_text_here...


Sexual Dysfunctions


Sexual Dysfunctions


