Sexual Dysfunction Disorders

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Cover Illustration by Kathryn K. Johnson

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PSYCHIATRY BOARD REVIEW MANUAL
Sexual Dysfunction Disorders

Keith A. Montgomery, MD, and Bethany J. Stockholm, MD

INTRODUCTION

Sexual dysfunction disorders affect both men and women. Despite their prevalence, these disorders are often not addressed by health care providers or patients due to their private and awkward nature. Physicians must move beyond their unease in order to adequately address patients’ sexual problems and implement appropriate treatment.

SEXUALITY DEFINED

Sexuality is a complex interplay of multiple facets, including anatomic, physiologic, psychologic, developmental, cultural, and relational factors. All of these contribute to an individual’s sexuality in varying degrees at any point in time as well as developing and changing throughout the life cycle. Sexuality in adults consists of 7 components: gender identity, orientation, intention, desire, arousal, orgasm, and emotional satisfaction. Gender identity, orientation, and intention form sexual identity, whereas desire, arousal, and orgasm are components of sexual function. The interplay of the first 6 components results in the emotional satisfaction experienced. In addition to the multiple factors involved in sexuality, there is the added complexity of the corresponding sexuality of the partner, as the expression of a person’s sexuality is intimately related to their partner’s sexuality.

SEXUAL RESPONSE CYCLE

The sexual response cycle consists of 4 phases: desire, arousal, orgasm, and resolution. Phase 1 of the sexual response cycle, desire, consists of 3 components: sexual drive, sexual motivation, and sexual wish. These reflect the biologic, psychologic, and social aspects of desire, respectively. Sexual drive is produced through psychoneuroendocrine mechanisms. The limbic system and the preoptic area of the anterior-medial hypothalamus are believed to play a role in sexual drive. Drive is also highly influenced by hormones, medications (eg, decreased by antihypertensive drugs, increased by dopaminergic compounds), and legal and illegal substances (eg, alcohol, cocaine).

Phase 2, arousal, is brought on by psychologic and/or physiologic stimulation. Multiple physiologic changes occur in men and women that prepare them for orgasm, mainly perpetuated by vasocongestion. In men, increased blood flow causes erection, penile color changes, and testicular elevation. Vasocongestion in women leads to vaginal lubrication, clitoral tumescence, and labial color changes. In general, heart rate, blood pressure, and respiratory rate as well as myotonia of many muscle groups increase during this phase.

Phase 3, orgasm, may last between 3 and 25 seconds, with continued elevation of respiratory rate, heart rate, and blood pressure and the voluntary and involuntary contraction of many muscle groups. In men, ejaculation is perpetuated by the contraction of the urethra, vas, seminal vesicles, and prostate. Conversely, in women, the uterus and lower third of the vagina contract involuntarily.

The duration of the final phase, resolution, is highly dependent on whether orgasm was achieved. If orgasm is not achieved, irritability and discomfort can result, potentially lasting for several hours. If orgasm is achieved, resolution may last 10 to 15 minutes with a sense of calm and relaxation. Respiratory rate, heart rate, and blood pressure return to baseline and vasocongestion diminishes. Women can have multiple successive orgasms secondary to a lack of a refractory period. The vast majority of men have a refractory period following orgasm in which subsequent orgasm is not possible.

The DSM-IV-TR lists 12 sexual dysfunction disorders (Table 1). There are 2 disorders of desire: hypoactive sexual desire disorder (HSDD) and sexual aversion disorder (SAD). There are 2 disorders of arousal: female sexual arousal disorder (FSAD) and male erectile disorder. The orgasmic phase of the sexual response cycle has 3 corresponding disorders: female orgasmic disorder (FOD), male orgasmic disorder (MOD), and premature ejaculation (PE). Vaginismus and dyspareunia are sexual pain disorders and do not correspond to the sexual response cycle. The DSM-IV lists 6 subtypes
for the aforementioned sexual disorders: lifelong or acquired, generalized or situational, and due to psychological factors or combined factors. In order for a patient to be diagnosed with a sexual dysfunction disorder, a psychophysiological problem must exist, the problem must cause marked distress or interpersonal difficulty, and the problem cannot be better accounted for by another Axis I diagnosis, substance, or general medical condition. In addition to the 9 disorders mentioned above, 3 more are defined in the DSM-IV. There are 2 sexual disorders that first must be ruled out before one can diagnose one of the aforementioned disorders. These are substance-induced sexual dysfunction and a sexual disorder due to general medical condition. The last disorder, sexual disorder not otherwise specified, is given if the patient’s symptoms do not meet the full criteria of any of the aforementioned disorders (Table 1). The sexual desire disorders, arousal disorders, orgasmic disorders, and pain disorders are discussed in more detail below.

### Sexual Desire Disorders

#### Prevalence

The prevalence of sexual desire disorders is often underappreciated. The National Health and Social Life Survey (NHSLS) found that 33.4% of women and 15.8% of men lacked sexual interest for several months within the last year. It also found that the prevalence of HSDD was 5% in men and 22% in women. Both HSDD and SAD have a higher female to male prevalence ratio, although this discrepancy is greater in SAD. The desire disorders can be considered on a continuum of severity with HSDD being the less severe of the 2 disorders.

#### Etiology

The proposed etiology of HSDD influences how it is subtyped (ie, generalized or situational, lifelong or acquired). For example, lifelong HSDD can be due to sexual identity issues (gender identity, orientation, or paraphilia) or stagnation in sexual growth (overly conservative background, developmental abnormalities, or abuse). Conversely, difficulty in a new sexual relationship may lead to an acquired or situational subtype of HSDD. Although it is theoretically possible to have no etiology, all appropriate avenues should be explored, including whether the patient was truthful in responses to questions regarding sexuality and if the patient is consciously aware that he/she has a sexual disorder. Diagnosis of sexual desire disorders is often difficult due to confounding factors. There are often high rates of psychiatric, medical, and substance-induced comorbidities.

Two important mediators of sexual desire are dopamine and prolactin. Dopamine acting through the mesolimbic dopaminergic reward pathway is hypothesized to increase desire, whereas, prolactin is thought to decrease libido, although the mechanism is poorly understood. Dopamine directly inhibits prolactin release from the pituitary gland. Medications that increase the release of prolactin or inhibit dopamine release or

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**Table 1. Delineating Criteria of 12 Sexual Dysfunction Disorders**

<table>
<thead>
<tr>
<th>Sexual Desire Disorders</th>
<th>Sexual Aversion Disorder</th>
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<tbody>
<tr>
<td>Hypoactive sexual desire disorder: persistently or recurrently deficient (or absent) sexual fantasies and desire for sexual activity</td>
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<tr>
<td>Sexual aversion disorder: persistent or recurrent extreme aversion to, and avoidance of, all (or almost all) genital sexual contact with a sexual partner</td>
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<table>
<thead>
<tr>
<th>Sexual Arousal Disorders</th>
<th>Orgasmic Disorders</th>
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<tbody>
<tr>
<td>Female sexual arousal disorder: persistent or recurrent inability to attain, or to maintain until completion of the sexual activity, an adequate lubrication-swelling response or sexual excitement</td>
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<tr>
<td>Male erectile disorder: persistent or recurrent inability to attain, or to maintain until completion of the sexual activity, an adequate erection</td>
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<table>
<thead>
<tr>
<th>Sexual Pain Disorders</th>
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<tbody>
<tr>
<td>Dyspareunia: recurrent or persistent genital pain associated with sexual intercourse in either a male or a female</td>
</tr>
<tr>
<td>Vaginismus: recurrent or persistent involuntary spasm of the musculature of the outer third of the vagina that interferes with sexual intercourse</td>
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<table>
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<tr>
<th>Sexual Dysfunction Due to a General Medical Condition</th>
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<tbody>
<tr>
<td>Any of the above-mentioned diagnoses must be judged to be exclusively due to the direct physiologic effects of a medical condition</td>
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<table>
<thead>
<tr>
<th>Substance-Induced Sexual Dysfunction</th>
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<tbody>
<tr>
<td>A sexual dysfunction that is fully explained by substance use in that it develops within a month of substance intoxication</td>
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</table>

<table>
<thead>
<tr>
<th>Sexual Dysfunction Not Otherwise Specified</th>
</tr>
</thead>
<tbody>
<tr>
<td>For problems that do not meet the categories described above</td>
</tr>
</tbody>
</table>

Adapted from Levine SB, Risen CB, Althof SE, editors. Handbook of clinical sexuality for mental health professionals. New York: Bruner/Routledge; 2003:1474. Copyright © 2003, with permission from Routledge/Taylor & Francis Group, LLC.
reuptake can decrease sexual desire along with other sexual side effects.\textsuperscript{10} If a patient has no history of sexual desire problems and has started a new sexual relationship, other possibilities for low sexual desire must be excluded. It is possible that neither individual has a sexual desire disorder but rather each individual’s level of desire is different, creating a discrepancy. Separate interviews with each partner are important to obtain a more accurate picture of the relationship.\textsuperscript{9}

**TREATMENT**

**Psychotherapy**

Although there are many proposed treatments for sexual desire disorders, there are virtually no controlled studies evaluating them.\textsuperscript{11} Psychotherapy is a common treatment for sexual desire disorders. From a psychodynamic perspective, sexual dysfunction is caused by unresolved unconscious conflicts of early development. Treatment focuses on bringing awareness to these unresolved conflicts and how they impact the patient’s life. While improvement may occur, the sexual dysfunction often becomes autonomous and persists requiring additional techniques to be employed.

A very successful approach to sexual desire disorders as well as other sexual dysfunctions, pioneered by Masters and Johnson,\textsuperscript{4} is dual sex therapy. In this therapy, the couple and a male and female therapist (gay and lesbian couples may opt for same-sex therapists) meet together. The relationship is treated as a whole, with sexual dysfunction being one aspect of the relationship. Another important underlying premise of dual sex therapy is that only 1 partner in the relationship is suffering from sexual dysfunction and absence of other major psychopathology. The aim is to reestablish open communication in the relationship. “Homework” assignments are given to the couple, the results of which are discussed at the following session. The couple is not allowed to engage in any sexual behavior together other than what is assigned by the therapists. Assignments start with foreplay, which encourages the couple to pay closer attention to the entire process of the sexual response cycle as well as the emotions involved and not solely on achieving orgasm. Eventually the couple progresses to intercourse with encouragement to try various positions without completing the act.\textsuperscript{1}

Many other sexual disorder therapies have been successful. The cognitive behavioral approach has shown efficacy in improving sexual and marital functioning in women.\textsuperscript{13} Specific exercises may be used. For example, men with sexual desire disorder or male erectile disorder may be instructed to masturbate to address performance anxiety related to achieving a full erection and ejaculation. Finally, analytically oriented sex therapy combines sex therapy with psychodynamic and psychoanalytic therapy and has shown good results.\textsuperscript{1} Specifically, for sexual desire disorders due to developmental and identity issues, long-term psychodynamic psychotherapy could be helpful.\textsuperscript{8} In general, lifelong and generalized desire disorders are more difficult to treat.\textsuperscript{9}

SAD is often progressive and rarely reverses spontaneously. It is also treatment-resistant.\textsuperscript{12} Poor prognostic indicators are global, lifelong, comorbid depression, or associated with anorgasmia.\textsuperscript{13} Despite difficulty in treatment, behavioral therapy has been shown to be effective for managing SAD.\textsuperscript{14,15}

**Pharmacotherapy**

Multiple hormones have been studied for treatment of sexual desire disorders. For example, androgen replacement has been studied as a possible treatment for HSDD. Androgen replacement has been found to increase libido in hypogonadal men; however, there was no observed benefit in eugonadal men.\textsuperscript{16} Side effects in men taking androgens include hypertension and prostatic enlargement.\textsuperscript{11} Unfortunately, the use of androgen therapy in women is not as clear.\textsuperscript{17} Although studies using supraphysiologic levels of androgens have shown increased libido, there is the risk of masculinization from chronic use.\textsuperscript{18}

Dehydroepiandrosterone-sulfate (DHEAS), a testosterone precursor, has also been studied for the treatment of sexual desire disorders. Low physiologic levels of DHEAS have been found in women presenting with HSDD.\textsuperscript{19} Increased libido was observed in women with adrenal insufficiency who were given DHEAS.\textsuperscript{20} Women with breast cancer reported increased libido while receiving tamoxifen, which increases gonadotropin-releasing hormone levels and therefore testosterone concentrations.\textsuperscript{1}

Some medications can be used to increase desire because they target certain receptors. For example, amphetamine and methylphenidate can increase sexual desire by increasing dopamine release. Bupropion, a norepinephrine and dopamine reuptake inhibitor, has been shown to increase libido.\textsuperscript{10} Multiple herbal remedies, such as yohimbine and ginseng root, are purported to increase desire, but this has not been confirmed in studies.\textsuperscript{1}

**SEXUAL AROUSAL DISORDERS**

**FEMALE SEXUAL AROUSAL DISORDER**

FSAD is defined as the inability (complete or partial)
to attain, or maintain until completion of the sexual activity, an adequate lubrication-swelling response of sexual excitement. In the NHSLS, 18% to 27% of women aged 18 to 59 years reported having trouble with lubrication.

Etiology

The etiology of FSAD may be vasculogenic, neurogenic, endocrinologic, or psychogenic. High blood pressure, hypercholesterolemia, diabetes, smoking, and heart disease cause atherosclerotic disease, which can lead to diminished pelvic blood flow. This arterial insufficiency disrupts the normal physiologic process of clitoral and vaginal engorgement and can lead to vaginal wall and clitoral smooth muscle fibrosis with resultant symptoms of vaginal dryness and dyspareunia. Spinal cord injury and diseases of the central and peripheral nervous system may be associated with FSAD. Menopause, premature ovarian failure, surgical or medical castration, dysregulation of the hypothalamic-pituitary axis, and long-term birth control use are common causes of primary endocrine abnormalities associated with sexual dysfunction. Despite the presence or absence of any organic cause, emotional and relational issues significantly affect sexual arousal. Common issues include low self-esteem, poor body image, relationship discord, and decreased ability to communicate sexual needs (caused by fear, anxiety, and guilt).

Clinical Presentation

The typical premenopausal female with FSAD complains of decreased drive, motivation, lubrication, and arousal. At times, it is difficult to determine if the complaint is primarily one of desire or arousal. In fact, many have challenged the accepted belief that desire and arousal are separate sequential processes. Basson and colleagues postulated that desire and arousal coexist and reinforce one another. Premenopausal women diagnosed with FSAD most likely have normal desire and wish for sexual arousal but have difficulty sustaining an arousal response. It is felt that a mental factor enters conscious awareness and distracts the patient from the process of lovemaking. Therapy in these cases would focus on this psychologic factor, which is likely to be influenced by the dynamics of current and past relationships.

Peri- and postmenopausal women with FSAD are more often focused on the body as a whole and present with multiple complaints, including decreased pleasure in response to oral and manual stimulation of the nipple, breast, and vulva or skin insensitivity. Although initial treatment with estrogen may improve vaginal dryness, sexual arousal may still be subjectively different. Therapy in this population may focus on the consequences of menopause in terms of body image, vitality, and attractiveness.

Treatment

Treatment must start with a complete physical examination, internal and external gynecologic examination, and appropriate laboratory testing (ie, follicle-stimulating hormone, luteinizing hormone, total and free testosterone, and prolactin levels). Although pharmacologic management of FSAD is limited, estrogen and testosterone have been shown to be effective. Estrogen replacement in postmenopausal women can improve clitoral and vaginal sensitivity, increase libido, and decrease vaginal dryness and pain during intercourse. Estrogen is available in several forms including oral tablets, dermal patch, vaginal ring, and cream. Testosterone supplementation has demonstrated improved libido, increased vaginal and clitoral sensitivity, increased vaginal lubrication, and heightened sexual arousal. Testosterone is available in oral tablet and sublingual forms, dermal patch, and cream and may be used alone or in combination with estrogen. Side effects of testosterone supplementation include weight gain, clitoral enlargement, facial hair, and hypercholesterolemia.

Multiple medications known to be effective for erectile dysfunction (ED) are being studied for their potential efficacy in women with FSAD, including sildenafil, vardenafil, tadalafil, alprostadil (prostaglandin E), and apomorphine. Sildenafil works by decreasing the catabolism of cyclic guanosine monophosphate (cGMP) thereby promoting smooth muscle relaxation and vascular engorgement. In women, smooth muscle relaxation should theoretically enhance lubrication and vaginal/clitoral engorgement. To date, studies regarding the efficacy of sildenafil in women with FSAD are inconclusive. Alprostadil cream has been shown to increase overall satisfaction with arousal response, although the response was not significant.

Mechanical vibrators and Eros Therapy (Nugyn, Inc., Spring Lake Park, MN), a clitoral vacuum engorgement device, are available for the treatment of both FSAD and FOD. Normal sexual arousal results in clitoral engorgement, and a dysfunction in this physiologic process may give rise to arousal and orgasmic difficulties. Mechanical vibrators use vibratory stimulation to cause clitoral engorgement and have demonstrated efficacy in the treatment of lifelong and acquired FSAD and FOD. Eros Therapy is a U.S. Food and Drug Administration (FDA)–approved vacuum pump device that is placed over the clitoris to increase blood flow and enable the attainment and maintenance of clitoral engorgement. A recent study by Billups et al of
5% to 15% between age 40 and 70 years. The prevalence of complete impotence tripled from ED (including minimal, moderate, and complete). Men aged 40 to 70 years experienced some degree of the sexual activity, an adequate erection. The Massachusetts Male Aging Study found that 52% of men with psychologic treatments for FSAD. Currently, there are no well-controlled studies evaluating efficacy of psychologic treatments for FSAD.20

Table 2. Psychogenic Dilemmas Likely to be Encountered in Lifelong Erectile Dysfunction

<table>
<thead>
<tr>
<th>Unconventional sexual identity</th>
<th>Uncommon in men without organic etiology of ED.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender identity problem</td>
<td>Male erectile disorder is defined as the persistent or recurrent inability to attain, or maintain until completion of the sexual activity, an adequate erection. The Massachusetts Male Aging Study found that 52% of men aged 40 to 70 years experienced some degree of ED (including minimal, moderate, and complete). The prevalence of complete impotence tripled from 5% to 15% between age 40 and 70 years.20</td>
</tr>
<tr>
<td>Wish to be a woman</td>
<td></td>
</tr>
<tr>
<td>A history of cross-dressing in women's clothing in private and/or public</td>
<td></td>
</tr>
<tr>
<td>Suspected by a psychiatrist but information initially withheld</td>
<td></td>
</tr>
<tr>
<td>Homosexualism</td>
<td>Without sexual behavior with men</td>
</tr>
<tr>
<td>With sexual behavior with men but not known to the female partner</td>
<td></td>
</tr>
<tr>
<td>With sexual behavior with men and known to the female partner</td>
<td></td>
</tr>
<tr>
<td>Paraphilia</td>
<td></td>
</tr>
<tr>
<td>One or more of a wide range of paraphilic patterns</td>
<td></td>
</tr>
<tr>
<td>Preference for prepubertal or young adolescents often initially denied unless thoroughly, systematically, and nonjudgmentally questioned</td>
<td></td>
</tr>
<tr>
<td>Compulsivity with or without obvious paraphilic imagery confined to masturbation with the help of pornographic images for stimulation</td>
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</table>

Serious character disorders (men have strong fear of closeness to women)

| Obsessive-compulsive            | Psychoanalytic treatment in individual or couple's therapy depending on the nature of the precipitating factors. Emotions associated with ED include anger, guilt, disdain, sadness, shame, longing, worthlessness, and anxiety. Common precipitants of these emotions may include deterioration of the marital relationship, divorce, deterioration of personal or spousal health, death of a spouse, threat of or actual unemployment, financial reversal, a secret extramarital affair, or a reunited marriage after an extramarital affair. |
| Schizotypal                     | Regardless of the etiology, all men with ED experience performance anxiety. The loss of reliable erectile function causes the man to question his potency and fear erectile failure in future sexual activities. This fear leads to vigilant monitoring of erectile status during lovemaking; this focus interferes with maintenance of arousal and ultimately leads to repeated erectile failure, resulting in further decreased sexual self-confidence. |
| Schizoid                        | Etiology

There are both medical and psychologic causes of ED. Advancing age is the factor most strongly associated with ED development. Other factors shown to increase the risk for ED (after adjusting for age) include smoking, diabetes, cardiovascular disease, anger, and depression. Psychologic factors that may cause ED include punitive superego, inability to trust, feelings of inadequacy, and feeling undesirable as a sexual partner. For some men, expression of sexual impulses can be stunted by fear, anxiety, anger, or moral prohibition. In an ongoing relationship, ED may reflect relational difficulties particularly in the male patient who has difficulty communicating his needs or anger in a constructive manner.

Lifelong ED is usually psychogenic and involves fear of being sexually close to a partner for a variety of reasons (Table 2). The prognosis for lifelong ED is poor, even with long-term therapy and use of erectogenic medications. Although regular sexual intercourse may not be attained, therapy can often allow these men to be more emotionally intimate with their partner. In contrast, the man with a previous history of potency who develops ED with his partner (acquired psychogenic erectile disorder) has a far better prognosis. These individuals may be treated in individual or couple’s therapy, depending on the nature of the precipitating factors. Emotions associated with ED include anger, guilt, disdain, sadness, shame, longing, worthlessness, and anxiety. Common precipitants of these emotions may include deterioration of the marital relationship, divorce, deterioration of personal or spousal health, death of a spouse, threat of or actual unemployment, financial reversal, a secret extramarital affair, or a reunited marriage after an extramarital affair.

Regardless of the etiology, all men with ED experience performance anxiety. The loss of reliable erectile function causes the man to question his potency and fear erectile failure in future sexual activities. This fear leads to vigilant monitoring of erectile status during lovemaking; this focus interferes with maintenance of arousal and ultimately leads to repeated erectile failure, resulting in further decreased sexual self-confidence.

**Treatment**

Prior to initiating treatment for ED, the firmness and duration of erection under various circumstances must be evaluated, including masturbation, sex with other male or female partners, in the middle of the night, upon awakening, upon stimulation with explicit stimuli, and sex other than intercourse. Organic causes are to be considered negligible if normal erection can
be accomplished in some circumstances. In these cases, ED is felt to be psychogenic even in men aged older than 50 years.

There are 3 important elements of psychotherapy for ED: psychoeducation, understanding the emotional context of lovemaking for the man, and addressing cognitive distortions and myths about the role of men in sexual intimacy. Current literature indicates that multiple interventions (eg, systematic desensitization, sensate focus, interpersonal therapy, behavioral assignments, sex education, communications and sexual skills training, masturbation exercises) have produced short- and long-term benefits in men with lifelong and acquired ED. For example, performance anxiety is addressed therapeutically by identifying the problem with the patient and using techniques such as sensate focus to enable the man to experience sexual intimacy without the risk of failure. Further investigation into these treatment modalities is necessary, as randomized controlled trials are lacking.

Pharmacologic management of ED was revolutionized by the FDA approval of sildenafil in 1998. During sexual arousal, nitric oxide activates guanylate cyclase, which produces cGMP, a second messenger of nitric oxide. cGMP then signals smooth muscle relaxation, resulting in erection. Phosphodiesterase type 5 (PDE-5) causes breakdown of cGMP resulting in loss of erection. Sildenafil, a PDE-5 inhibitor, prevents the degradation of cGMP in the nitric oxide–cGMP pathway, thus allowing erection to be maintained. To date, there are 3 PDE-5 inhibitors currently available for the treatment of ED: sildenafil, vardenafl, and tadalafil. Although head-to-head comparative trials are not available, all 3 appear to be equally efficacious, although tadalafil has a longer half-life, allowing more spontaneity. Treatment options for patients with contraindications to PDE-5 inhibitors or in whom these agents are ineffective include intrarectal alprostadil and penile injection therapy with a combination of alprostadil, papaverin, and phentolamine. Nonmedical treatment of ED includes vacuum pump devices and penile implants, the later being available to those who have failed all medical interventions.

ORGASMIC DISORDERS

FEMALE ORGASMIC DISORDER

FOD is defined as persistent or recurrent delay in or absence of orgasm in the setting of a normal sexual excitement phase. In a study of 1749 women aged 18 to 59 years, 24% reported being unable to attain orgasm for several months over the past year. The physician must determine if orgasm has been possible under a variety of circumstances, such as during solitary or partner masturbation, oral-genital stimulation, with a partner other than the significant other, during sexual fantasizing, or during sleep. Patients should be classified as having lifelong generalized FOD if an orgasm has never been experienced by any stimuli, whereas the situation-al subtype denotes a female who is able to attain orgasm under certain circumstances (eg, only during solitary masturbation). Acquired FOD is more common and can consist of complete lack of orgasm, too infrequent orgasms, or too difficult to attain orgasms.

Etiology

The attainment of regular orgasms with a partner is important in adult sexual development and involves the interplay of physiologic, cultural, and individual psychologic factors. FOD is diagnosed when the individual psychology of the patient persistently interferes with the body’s physiologic progression through arousal. Multiple psychologic factors can contribute to inhibition of orgasm such as fear of rejection, embarrassment/guilt of sexual impulses, or fear of losing control. Despite modern society’s efforts toward equality of women’s sexuality, many are still bound by the old belief that “good girls” are devoid of sexual knowledge, behavior, and pleasure. The most common cause of FOD is treatment with a serotonergic agent. In patients being treated with serotonergic antidepressants, the prevalence of FOD approaches 70%. In cases in which the cause of FOD is not medication-related, the clinician must carefully explore the changes in the patient’s life prior to or coinciding with the onset of symptoms to determine their contribution.

Treatment

When considering treatment for FOD, the clinician should realize that many women gradually overcome this difficulty as they age and increasingly grow to trust their partners. Providing education and encouragement to these patients can often foster improved self-confidence and decrease anxiety. Individual therapy (most common), group therapy, couple’s therapy, and bibliotherapy (most cost-effective) are effective treatment options for FOD. Directed masturbation, when recommended in group, individual, and couple’s therapy, has been shown to be efficacious, particularly in the treatment of lifelong, generalized anorgasmia. A woman who is able to reach orgasm by solitary masturbation but not with her partner is better addressed in couple’s therapy. Many articles and books are available on this topic that educate
and encourage women to actively learn more about their sexuality. It is believed that these publications help women to become increasingly confident and competent in their pursuit and acquisition of sexual pleasure.²

Data on the use of pharmacologic agents in the treatment of FOD are lacking. A study recently examined the effect of bupropion sustained-release (150–300 mg) on orgasmic dysfunction in nondepressed females (n = 20).³⁵ In bupropion-treated patients, there were significant improvements in overall sexual satisfaction and satisfaction with orgasm intensity independent of the dose.³⁵ As previously discussed, mechanical devices have also been used to treat orgasmic dysfunction with some success.²⁷,²⁸

MALE ORGASMIC DISORDER

MOD is defined as the persistent or recurrent delay in, or absence of, orgasm following a normal sexual excitement phase during sexual activity that, taking into account the person’s age, judges to be adequate in focus, intensity, and duration.⁶ Many have suggested that a differentiation be made between orgasm and ejaculation to include entities such as anesthetic ejaculation (normal ejaculation with absence of orgasmic sensation) and partial ejaculatory incompetence.³⁶ Laumann et al³ reported that 8.3% of men aged 18 to 59 years described lack of orgasm over the past year.⁷

Etiology

MOD may be caused by psychogenic, congenital, neurogenic, infectious, or endocrinologic factors or may be medication-induced.³⁵ MOD is usually lifelong and not partner-specific, with severity existing on a continuum. Complaints range from men who are able to attain orgasm by means other than vaginal intercourse (most common), to men unable to ejaculate in their partners’ presence, and finally rarely the inability to ejaculate while awake.² MOD is classically attributed to fear, anxiety, hostility, and relationship difficulties. Some hypothesized manifestations of these difficulties include fear of castration by female genitalia, fear of impregnating the female, unwillingness to be “swept away,” fear of loss of self with loss of semen, or guilt caused by a rigid religious upbringing.³⁶ Unfortunately, there are no well-controlled studies that support generalization of any one of these factors to the development of MOD.

Treatment

Some patients with MOD improve with psychotherapy, while others improve spontaneously over time. MOD may lead to abandonment of the pursuit of sex with a partner.² Currently, there are no FDA-approved medications for the treatment of MOD. A study evaluating the efficacy of bupropion sustained-release (150 mg or 300 mg) in the treatment of orgasmic dysfunction in nondepressed men (n = 10) demonstrated significant improvement over placebo in overall sexual satisfaction, ability to achieve erection, and delay in orgasm/ejaculation.³⁵ In this study, no significant difference was noted in any area of sexual functioning between the 2 doses of bupropion.

PREMATURE EJACULATION

PE has been cited as the most common sexual disorder in men.³⁸ PE is defined as persistent or recurrent ejaculation with minimal sexual stimulation before, on, or shortly after penetration and before the person wishes it.⁶ In the NHLS, 28% to 32% of men aged 18 to 59 years reported that they ejaculate too rapidly; further symptom-cluster analysis calculated an overall PE prevalence of 21%.⁷ The definition/diagnostic criteria for PE varies from study to study, as do the ways of evaluating symptoms, thereby making exact measurements of prevalence difficult.³⁸ Common complaints of self-diagnosed patients can range from ejaculation immediately upon or prior to vaginal penetration, within minutes (usual), or sooner than desired by the patient and his partner.²

Etiology

Both psychogenic and biologic processes have been implicated as causes of PE, including penile hypersensitivity, hyperexcitable ejaculatory reflex, endocrinopathy, genetic predisposition, 5-HT receptor dysfunction, early sexual experience, frequency of sexual intercourse, ejaculatory control techniques, and psychodynamic factors.³⁷ The impact of PE on patient behavior and quality of life is significant. Symonds et al³⁹ interviewed 28 men aged 25 to 70 years with PE to assess whether patients had concerns about PE and what the concerns were. The overriding concern was a decrease in their sexual self-confidence (68%). Half of the patients were concerned about the impact of PE on their relationship; some had reluctance to start a new relationship and those in relationships feared disappointing their partner. Thirty-six percent of patients reported that anxiety caused PE or resulted from it. Sixty-seven percent did not speak with a physician about their PE because of embarrassment, and 47% believed there was no treatment.³⁹

Treatment

Current management of PE involves both pharmacologic and psychologic interventions. Pharmacologic treatments may include serotonergic antidepressants, topical anesthetics, and PDE-5 inhibitors; however,
none of these agents are approved by the FDA for this indication. It has been suggested that PE may be related to diminished serotonergic neurotransmission, as delayed ejaculation is often a side effect of antidepressant therapy with selective serotonin reuptake inhibitors (SSRIs). Antidepressants typically used to treat PE include fluoxetine, sertraline, fluvoxamine, and paroxetine dosed either daily or on-demand. McMahon and Touma demonstrated that the effect of paroxetine on intravaginal ejaculatory latency time (IELT) prolongation was significantly better in patients who were treated initially with regularly scheduled dosing (paroxetine 20 mg/day for 2 wks) followed by dosing before intercourse (paroxetine 20 mg) compared with patients treated only before intercourse. Others have demonstrated that the tricyclic antidepressant, clomipramine, was more effective than paroxetine for prolongation of IELT.

Collectively, studies demonstrate some benefit with SSRIs; however, limitations exist due to dosing schedules needed for the desired effect, time to onset of action (agent must be taken 3–8 hrs prior to intercourse), stigma of chronic antidepressant treatment, and incidence and nature of other side effects associated with SSRIs. Topical anesthetics such as lidocaine/prilocaine cream (25 mg/25 mg) applied to the penile shaft 10 to 20 minutes prior to intercourse significantly increased IELT. Side effects, such as retarded ejaculation of more than 30 minutes, decreased penile sensitivity, decreased vaginal sensitivity, and penile irritation may limit widespread use of topical anesthetics. Medications indicated for ED, such as sildenafil, have shown some efficacy in PE when combined with paroxetine and psychologic and behavioral therapy, but these results are limited because they were not placebo-controlled.

Psychologic interventions for PE include behaviorally oriented sex therapy in which men are trained to monitor penile sensations and employ various techniques to halt the progression of arousal to orgasm. The “squeeze technique” involves the man or his partner squeezing the glans or shaft of the penis long enough to prevent escalation of arousal. Other behavioral techniques include “the start stop method” and sensate focus.

**SEXUAL PAIN DISORDERS**

Sexual pain disorders include vaginismus and dyspareunia. Both disorders involve difficulty with vaginal penetration. Although vaginismus and dyspareunia are 2 separate diagnoses, they often coexist.

**VAGINISMUS**

Vaginismus is defined in the DSM-IV as the persistent or recurrent involuntary spasm of the outer third of the vagina that interferes with penile insertion and intercourse. Although vaginismus is thought to be common, the exact prevalence in the general population is unknown. Recent studies have suggested that using vaginal spasm as a diagnostic indicator is unreliable. Others have suggested vaginismus be defined as “the persistent or recurrent difficulties of the woman to allow vaginal entry of a penis, a finger, and or any object, despite the woman’s expressed wish to do so.” There is often (phobic) avoidance, involuntary pelvic muscle contraction, and anticipation/fear/experience of pain.

**Etiology**

Physical causes of pain that may contribute to vaginismus include genital tract infections, vestibulitis, postmenopausal estrogen deficiency, surgical trauma (eg, episiotomy), and radiotherapy. Historically, psychologic associations are early traumatic sexual experiences, sexual assault, and strict religious upbringing. A recent study investigated the potential correlation of sexual and physical abuse, sexual knowledge, sexual self-schema, and relationship adjustment to vaginismus. This study concluded that women with vaginismus were twice as likely to report a history of childhood sexual abuse, less positive attitudes about their sexuality, and lower levels of sexual functioning.

**Treatment**

Treatment of vaginismus involves education, relaxation techniques, cognitive behavioral therapy, psychosexual therapy, vaginal dilators, and botulinum toxin. Although several treatment approaches exist, their efficacy has not been well-established. Vaginal dilators have been shown to be successful (measured as vaginal intercourse) in 75% to 100% of patients with vaginismus. Psychotherapy usually focuses on the elucidation of the personal and interpersonal meanings of the symptom development. A recent study revealed that local injection of botulinum toxin in women with moderate to severe vaginismus enabled vaginal examination in 94.8% of patients and vaginal intercourse in 75% of patients. At 12.3-month follow-up, no recurrence of symptoms was reported.

**DYSPAREUNIA**

Dyspareunia is defined as recurrent or persistent genital pain associated with sexual intercourse in either a male or a female. The NHSLS found that 14.4% of
women and 3% of men aged 18 to 59 years reported experiencing pain during sex over the past year.2

### Etiology

For some women with dyspareunia, the initial sexual encounter creates intense anxiety and this may result in involuntary vaginal muscle contraction with resultant physical pain. In these cases, future sexual activity is approached with fear, which causes anxiety, vaginal muscle contraction, and pain, thereby creating a cycle where vaginismus and dyspareunia coexist. Recurring coital pain can also cause diminished arousal and sexual avoidance for both men and women.2 Latthe et al31 concluded that dyspareunia in women is associated with anxiety, depression, and sexual assault. In both men and women, multiple medical conditions are known to cause painful coitus (Table 3). Because the symptom of dyspareunia is associated with multiple organic causes, a thorough physical examination must be conducted.

### Treatment

Postmenopausal coital pain is often the result of a low estrogen state, which results in thinning of the vaginal mucosa, decreased lubrication, and loss of vaginal and labial elasticity.2 Use of local estrogen replacement therapy for postmenopausal changes and active dilation of the introitus with vaginal dilators or fingers has been effective in restoring intercourse in women who desire coitus.22 The exploration of psychodynamic issues is necessary when present, whether related to the individual or to the couple.2

**Table 3. Medical Etiologies of Dyspareunia in Women and Men**

<table>
<thead>
<tr>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irritated or infected hymenal remnants</td>
<td>Peyronie’s disease</td>
</tr>
<tr>
<td>Episiotomy scar</td>
<td>Prostatitis</td>
</tr>
<tr>
<td>Bartholin’s gland infection</td>
<td>Urinary tract infections</td>
</tr>
<tr>
<td>Vaginitis</td>
<td>Obstructed ejaculatory duct</td>
</tr>
<tr>
<td>Cervicitis</td>
<td>Postmenopausal vaginal mucosa thinning</td>
</tr>
<tr>
<td>Pelvic inflammatory disease</td>
<td>Postmenopausal reduced lubrication</td>
</tr>
<tr>
<td>Fibroids (age ≥ 49 years)</td>
<td>Pudendal nerve entrapment</td>
</tr>
<tr>
<td>Ovarian cysts (age ≥ 49 years)</td>
<td></td>
</tr>
<tr>
<td>Endometriosis (age ≥ 49 years)</td>
<td></td>
</tr>
<tr>
<td>Postmenopausal vaginal mucosa thinning</td>
<td></td>
</tr>
<tr>
<td>Postmenopausal reduced lubrication</td>
<td></td>
</tr>
</tbody>
</table>

### REFERENCES

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