

David L. Rowland

Sexual Dysfunction in Men



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Sexual Dysfunction in Men

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David L. Rowland received his PhD from the University of Chicago in 1977, and since then has held numerous research fellowships in the US and abroad. His research focuses on understanding sexual response and disorders in men and women, particularly the interface between the physiological and psychological experience of sexual response. He has published over 130 articles, monographs, and chapters, including the 2008 *Handbook of Sexual and Gender Identity Disorders*. He served as editor of the *Annual Review of Sex Research*, 2005–2009, currently serves on the editorial boards of major journals in the field of sex research, and has provided expert consultation to pharmaceutical companies and professional societies, including service on the Standards Committee of the International Society of Sexual Medicine.

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Preface

The past decade has witnessed increased interest in and research on sexual problems in both men and women. At the same time, new pharmacological solutions to male sexual dysfunctions have become available, and a number of new agents are currently under investigation. These developments are, of course, not independent of one another, but in the course of these developments, two things have become apparent:

(1) We are still far from having a complete understanding of basic human sexual response, let alone sexual dysfunction. For example, we do not understand why some men seem unable to control or delay their ejaculation, or why other men are particularly vulnerable to developing “performance anxiety,” while others do not quickly lose their erections as a result of experiencing a sexual problem.

(2) Contrary to speculation that psychosexual counseling would become obsolete (given this pills-for-better-sex decade), its role has actually become better understood and defined. If nothing else, this decade has taught us that although pills can help fix the body and genitals, psychosexual counseling is needed to help heal both the person and the relationship.

This book is written for a broad audience that includes not only nonspecialist therapists, clinicians, and even physicians whose patients/clients raise concerns about their sexual well-being, but also for patients/clients themselves and their partners. Although some sexual problems invariably require the attention of a specialist, many others can be handled adequately by the nonspecialist assuming (1) he or she already has training as a therapist or clinician and (2) he or she has acquired a reasonably broad understanding of men’s sexual dysfunctions – their etiologies, diagnoses, and treatments. Meeting these assumptions ensures that the therapist’s general counseling skills are contextualized and tailored to meet the needs of clients who express a sexual concern or difficulty.

It is indeed the second assumption above that has provided the impetus for this book. Many therapists may not feel qualified or comfortable dealing with the sexual problems of clients. Together with its twin, *Sexual Dysfunction in Women*, authored by Marta Meana, these volumes not only give general frameworks for thinking about sexual response and sexual problems, but also set forth diagnostic and treatment strategies in a simplified and clear manner for the nonspecialist.

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And finally, to those who continue to enrich my life: my daughter, parents, special friends left unnamed, and the staff of the Graduate School and Office of Continuing Education at Valparaiso University.

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1

Description

The ability to have a fulfilling sexual relationship is important to almost all men's mental health and psychological well-being. Not only is this a biologically and socially defining characteristic for men in our society, but studies suggest that men in such relationships tend to have greater longevity and to report a higher quality of life and overall satisfaction (McCabe, 1997; Palmore, 1985). Men whose sexual relationships are disrupted because of their inability to respond adequately, typically experience a number of psychological symptoms, including lack of confidence, anxiety, and distress.

Although a select few therapists specialize in the treatment of sexual problems, most do not; therefore, the likelihood that a client or patient may approach a general therapist who counsels and treats patients with a variety of issues is quite high. Even the generalist can be helpful to men in need of sexual guidance and advice. Important to this process is an understanding of the components of sexual response, its etiology and diagnosis, and current treatment practices.

Even the nonspecialized therapist can be helpful by understanding the etiology, diagnosis, and treatment practices for various dysfunctions

1.1 Terminology

Sexual response is complex: It requires specific preconditions, involves multiple behavioral responses, and includes an array of psychosocial factors that have affective, cognitive, and relationship dimensions. Masters and Johnson (1966) succeeded in providing a rudimentary characterization of physiological sexual response, analyzing it into arousal, plateau, orgasmic, and resolution phases. Subsequent models introduced a role for *sexual desire* as a component of sexual response (Kaplan, 1979), with a more recent refinement that distinguishes between such constructs as *spontaneous desire* and *arousability*, the latter referring to sexual interest derived from a specific individual, object, or context as opposed to an “unprompted” desire. Further conceptualization has included separate pain-pleasure dimensions (Schover, Friedman, Weiler, Heiman, & LoPiccolo, 1982), as well as attention to other subjective factors such as the feelings, motivations, and attitudes that surround the sexual act (Byrne & Schulte, 1990). Recently, emphasis has also been given to the role of the dyadic relationship, an approach that seeks to understand and treat sexual dysfunction in its relational context (Schnarch, 1988, 1991).

Healthy sexual relationships, however, are not characterized merely by the absence of dysfunctional response. Key elements of healthy sexual relationships include passion, intimacy and caring, and commitment (Sternberg & Barnes, 1988).

Healthy sexual relationships involve more than just the absence of dysfunctional response; many problems include larger relationship factors beyond sexual response issues

- Passion typically involves such characteristics as sexual feelings, physical attraction, and romantic love.
- Intimacy and caring deal with dimensions of affection and expressiveness – the willingness to communicate and share beliefs, attitudes, and feelings.
- Commitment refers to the decision to be with one partner and to work hard to maintain the relationship.

Because many sexual problems are rooted in a couple's disparate expectations and emotional struggles, including the different ways in which these elements are often played out by each of the sexes, most sexual problems benefit not just from attention to specific sexual response issues but to larger relationship factors as well.

There are several different types of sexual disorders. In the field of sexuality, distinctions are made among the sexual dysfunctions, the gender identity disorders, and atypical and paraphilic behaviors.

- *Sexual dysfunction* refers to disruption or inadequacy of normal sexual responding and is the topic of this book.
- *Gender identity disorders* refer to cross-gender identity or the lack of assimilation of, or satisfaction with, the gender identity consistent with one's biological sex or assigned gender identity.
- *Paraphilias* refer to sexual arousal and behaviors that are directed toward inappropriate objects/partners or are carried out in inappropriate situations (e.g., fetishism, pedophilia, frotteurism, voyeurism, etc.).

1.2 Definition

The classification of sexual dysfunctions has evolved from the conceptual models discussed above and is related to the specific axes or dimensions important to functional sexual response (American Psychiatric Association, 2000). These include:

- lack of desire, also known as hypoactive sexual desire disorder;
- problems with either physiological sexual arousal (e.g., erection) or subjective sexual arousal (i.e., actually feeling aroused);
- disorders of ejaculation/orgasm, most commonly premature ejaculation and inhibited ejaculation.

Although not part of this review, problems with painful intercourse and sexual aversion are also included in the diagnostic classification system.

Typically, the scope of the sexual problem is characterized as either situation- (including person) specific or generalized, and as either lifelong or acquired. An acquired sexual dysfunction may result from either pathophysiological developments or sexual experiences. Several classification systems are currently in use to define and characterize sexual dysfunctions: The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision (DSM-IV-TR), the proposed 5th edition of the DSM (DSM-5), and the *International Statistical Classification of Diseases and Related Health Problems*, 10th Edition (ICD-10) classifications are included in Table 1. Characteristics of each dysfunction, along with alternate terminology and prevalence estimates are provided in Table 2.

Table 1
Comparison of Terminology Across Diagnostic Manuals for Sexual Dysfunctions in Men

| DSM-IV-TR (codes) | ICD-10 (codes) | DSM-5 proposed (as of May 15, 2011) |
|---------------------------------|---------------------------------------|---|
| Sexual Desire Disorders | | |
| HSDD (302.71) | Loss or lack of sexual desire (F52.0) | <i>Hypoactive sexual desire disorder in men</i> |
| Sexual Arousal Disorders | | |
| Male erectile disorder (302.72) | Failure of genital response (F52.2) | <i>Erectile disorder</i> |
| Orgasm Disorders | | |
| MOD (302.74) | Orgasmic dysfunction (F52.3) | <i>Delayed ejaculation</i> |
| Premature ejaculation (302.75) | Premature ejaculation (F52.4) | <i>Early ejaculation</i> |

Note. DSM-IV-TR = *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision (APA, 2000); HSDD = Hypoactive sexual desire disorder; ICD-10 = *International Statistical Classification of Diseases and Related Health Problems*, 10th Edition (World Health Organization, 1992); MOD = Male orgasmic disorder.

Although sexual dysfunctions in men and women generally parallel one another, the prevalence of the various dysfunctions differentiates the sexes; and, because of differences in physiology and evolution, they are often manifested in different ways (Lewis et al., 2004). For example, anorgasmia and lack of sexual desire are more common among women, whereas rapid ejaculation/orgasm and physiological arousal problems (e.g., erection in men versus lubrication in women) are more common among men.

In broad terms, no matter what the problem, men's sexual problems typically have three elements:

- (1) A *functional impairment* of some type is evident. For example, the man and his partner are unable to enjoy intercourse because he is unable to get or keep an erection, or because he ejaculates very quickly.
- (2) The man's sense of *self-efficacy is low*, as he is typically unable to correct or control the problem through psychobehavioral changes. For example, the man just cannot seem to get interested in sex, or he is unable to delay his ejaculation.
- (3) The man and/or his partner suffer *negative consequences* from the condition. For example, the man is bothered or even obsessed by the problem, perhaps to the point of avoiding intimacy; or the partner is distressed by the situation, not knowing what to do, perhaps feeling frustrated and unattractive, and so on. The challenge in the field of sexology, however, is that although there is only "one" unified sexual response in the patient's view – the man typically does not distinguish among the desire, erection, and ejaculation phases of the response – the physiology underlying the functional impairment associated with each

The functional impairment associated with each phase of the sexual response cycle has a distinct underlying physiology

Table 2
Definition and Prevalence of Major Male Sexual Dysfunctions

| Dysfunction/nomenclature | Defining characteristics | DSM-IV code | Prevalence | Age ^a | |
|---|--|---|------------|------------------|-----|
| Hypoactive sexual desire disorder (HSDD) | Loss of libido or sexual interest | – Diminished/absent interest of desire – Absent sexual thoughts or fantasies – Lack of responsive desire | 302.71 | 4–25% | ++ |
| Arousal disorder^b | | – No or diminished subjective erotic feelings despite normal erection | 302.70 | NA | |
| Erectile dysfunction (ED) | Impotence | – Inability to attain erection – Inability to maintain erection to completion of activity – Coital penetration impaired ≥ 50% | 302.72 | 10–45% | +++ |
| Premature ejaculation (PE) | Rapid ejaculation Early ejaculation | – Onset of orgasm and ejaculation before or shortly after penetration – Ejaculation within 1 min or less – Occurs before desired due to lack of control | 302.75 | 12–30% | ? |
| Male orgasmic disorder (MOD) | Ejaculatory incompetence Retarded or delayed ejaculation Inhibited ejaculation | – Delayed or absent orgasm following normal excitement (erection) phase | 302.74 | 5–10% | ++ |
| Dyspareunia | | – Pain associated with intercourse before, during, or after | 302.76 | 1–5% | |

Note. DSM-IV = *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition.

^aIndicates whether the dysfunction increases with age in a weak (+), moderate (++), or strong (+++) manner.

^bSubjective arousal disorder does not have a dedicated classification but is increasingly recognized as a potential problem for men with orgasmic disorders.

Adapted from "Sexual Health and Problems: Erectile Dysfunction, Premature Ejaculation, and Orgasmic Disorders," by D. L. Rowland, in J. Grant & M. Potenza (Eds.), *Textbook of Men's Mental Health* (pp. 171–204). Arlington, VA: American Psychiatric Association Press, 2007.

of these phases is quite distinct. Thus, dysfunction within each phase has its own prevalence, etiology, diagnosis, and treatment. To provide greater depth and understanding, this book takes a dual approach, dealing with common and comprehensive issues underlying all dysfunctions in Chapters 1–3, and then devoting individual sections to each phase/dysfunction in Chapter 4.

1.3 Epidemiology

For obvious reasons, determining the prevalence of any sexual dysfunction in men is complicated and challenging. To illustrate the point, defining a problem as a sexual *dysfunction* requires that the functional impairment meet specific criteria. In many instances, a man may view his response as problematic, but it may not meet the criteria necessary for a clinical diagnosis. For example, a man has occasional erectile failure, or ejaculates before he wants to even when ejaculation occurs 3 or 4 minutes after vaginal insertion. Furthermore, for a man to be classified as dysfunctional also requires that he recognize the symptoms and potentially defines himself in this category. Yet, issues of privacy, discretion, and sometimes stigmatization may inhibit men from disclosing what might be viewed as a “weakness” (what man wants to admit to being classified as *dysfunctional*?). Finally, prevalence is a moving target. For example, for some men, the problem may be transient, as when life stressors or particular sensitivity or vulnerability to specific relationship interactions contribute to or intensify a problem that is initially “subclinical.” Cultural attitudes, expectations, and acceptance regarding men’s sexual problems may influence the willingness to acknowledge and report such problems. And for those sexual dysfunctions that are partly age-related (e.g., problems with erection and/or sometimes sexual interest), as the age demographic of a population changes, so also does the prevalence (see Table 2 for prevalence estimates).

Many factors contribute to the difficulty of determining the prevalence of sexual dysfunctions in men

Men may be hesitant to identify themselves as dysfunctional, since classification as “dysfunctional” is stigmatizing and requires their recognition of the symptoms

1.3.1 Low Sexual Desire

Data from the National Health and Social Life Survey (Laumann, Paik, & Rosen, 1999) suggest the prevalence of low sexual desire is around 5%. This particular survey had several limitations, and the more recent Global Study of Sexual Attitudes and Behaviors (Laumann, Nicolosi, Glasser, Paik, & Gingell, 2005) suggests a higher prevalence, somewhere between 13% and 28%. Given the general superiority of this latter survey, these rates are probably more reflective of the true prevalence. Several other studies report rates ranging from 0 to about 25%, depending on the nature of the question, the population sampled, and whether the individuals were clinically- or self-diagnosed. Taken together, these and other studies (Lewis et al., 2010) suggest prevalence somewhere between 15% and 25% of men, with the potential of higher rates in men over 60. However, most such studies have not distinguished between

It is important to distinguish between sexual *desire* and sexual *interest*

sexual “desire” and “interest.” Whereas desire is presumed to be internally driven and physiologically based (at least for men), *interest* is a more inclusive term that may be affected by such factors as partner attraction, relationship issues, environmental stressors, and so on. The extent to which this more inclusive condition (low sexual “interest”) prevails in the population is not known despite the fact that low sexual interest undoubtedly affects the sexual relationship.

1.3.2 Erectile Dysfunction

Prevalence of erectile dysfunction (ED) among men varies significantly according to the way in which the dysfunction is defined, the population sampled, and information collected (Lewis et al., 2010). Overall, an estimated 18% of males 20 years and older, or about 18 million men in the United States, have erectile dysfunction (Selvin, Burnett, & Platz, 2007). However, the most important source of variation in ED prevalence is the age of the respondents. Among men below the age of 40, ED ranges from 1% to 10%; for those between 40 and 49, ED is higher, perhaps closer to 8–12%; for those 50 or older, the prevalence approaches 25% and can reach as high as 50%+ for men in their 70s and 80s. Clearly, the age-related increase in prevalence strongly suggests an ED of somatic/biogenic origin; reliable estimates regarding the prevalence of psychogenic ED are not available.

The prevalence of biogenic ED is easier to estimate than psychogenic ED, as the age-related increase offers evidence of a biogenic origin

Undoubtedly, the link between biogenic and psychogenic ED is important – as men experience increased difficulty getting and maintaining an erection due to biogenic factors, they are more likely to experience anxiety and performance demand, two psychological factors that interfere with erectile response. Nevertheless, such links between biogenic and psychogenic factors may not necessarily presume higher levels of psychogenic ED in older men. Just as readily, younger men having limited experience with sexual intimacy, entering new relationships, or having to deal with strongly defined social roles are also vulnerable to the antierecile effects of anxiety and performance demand within a sexual relationship.

1.3.3 Premature Ejaculation

PE is probably the most common sexual dysfunction in men

Premature ejaculation (PE) appears to be fairly common; in fact, it is probably the most common sexual dysfunction in the general population, including primary care patients, with studies reporting ranges from 11% to 66% (Ahn et al., 2007; Aschka, Himmel, Ittner, & Kochen, 2001). Most studies estimate 20–30% of men, regardless of nationality, will experience PE at some point in their lives (see Rowland, 2011). Variability in estimates likely arises from:

- (1) the use of different definitions of premature ejaculation, particularly definitions for the latency to ejaculation, which has ranged anywhere from one to several minutes after partner penetration;
- (2) whether the estimate is based on a clinical diagnosis versus self-report by the responder;

- (3) the extent to which the man is actually bothered by the condition. Several studies, for example, report rates as high as 30%, but only half those men indicate the problem is sufficiently serious to seek treatment.

As expected, prevalence estimates from clinical samples of men attending sex clinics or seeing primary care physicians suggest somewhat higher rates than in the general population (Aschka et al., 2001), with 34.8% of men in one sample reporting having experienced PE at some time during their lifetime (Riley & Riley, 2005). Overall, such data suggest that the rates of PE or PE-like complaints are quite high – probably around 15-25% within the general population. Some research indicates that age is not a factor with respect to the prevalence of PE; other research, however, suggests that PE may either increase or decrease with increasing age (e.g., Ahn et al., 2007). Given the inconsistent results, the most parsimonious position is to assume that there is no *large* change in PE prevalence related to age.

1.3.4 Delayed and Inhibited Ejaculation

The prevalence of delayed or inhibited ejaculation (IE; the latter being the complete inability to reach ejaculation) is unclear – normative data for defining the duration of “normal” ejaculatory latency, particularly regarding the right tail of the distribution (i.e., beyond the mean latency to ejaculation), is essentially nonexistent. Furthermore, larger epidemiological studies have not subdivided men into various types of diminished ejaculatory function. For example, the continuum (and/or overlap) from delayed to inhibited ejaculation has not been adequately explored.

In general, IE has been reported at fairly low rates in the literature, typically around 3% (Rowland et al., 2010), and thus it has been seen as a clinical rarity. Masters and Johnson (1966) initially reported only 17 cases; Apfelbaum (2000) reported 34 cases and Kaplan fewer than 50 cases in their respective practices (see Perelman & Rowland, 2008). However, based on clinical experiences, some urologists and sex therapists are reporting an increasing incidence of IE (Rowland et al., 2010) leading to newer estimates of anywhere between 3% and 10% (see Lewis et al., 2010). The prevalence of IE appears to be moderately and positively related to age – not surprising in view of the fact that ejaculatory function as a whole tends to diminish with age. However, no large-scale studies have systematically investigated the strength or reliability of this putative relationship.

1.3.5 Other Considerations

The various dysfunctions themselves do not represent mutually exclusive categories. In fact, the interrelatedness of the components of the sexual response cycle increases the likelihood that men with a problem related to one phase of the sexual response cycle may exhibit a problem in another phase. Important to any evaluation and treatment process is determining which problem is primary and which is secondary. Thus, a man who has significant problems with erection may eventually “lose” or suppress interest in sex altogether. Similarly,

Due to the interrelatedness of the phases of the sexual response cycle, men with one sexual problem will often manifest other sexual problems

When multiple problems are encountered, it is important to distinguish between which problem is primary and which is secondary

nearly one third of men reporting problems with premature ejaculation also experience difficulties with erection.

Generally, a common underlying theme for all sexual dysfunctions is that they are bothersome and cause significant worry or distress to the individual. In some instances, the distress may not just be caused by inadequate sexual performance, but may stem from the impact the dysfunction has on the couple (e.g., disruption of intimacy, lack of partner satisfaction, etc.). In contrast, some men may experience minimal distress due to their condition. For example, a man who ejaculates very rapidly may employ strategies other than coitus to ensure his partner's sexual enjoyment and therefore may have little distress and consequently little motivation to seek treatment. The question – one that has been debated vigorously in sexological circles – is whether such men, that is, those showing the symptoms but without stress or bother, manifest a true sexual dysfunction.

1.4 Course and Prognosis

1.4.1 Psychophysiology of Male Sexual Function: A Brief Overview

Libido or sexual desire is a psychological construct

To understand risk factors and treatment options, a basic familiarity with the psychophysiological processes of sexual desire, sexual arousal, and orgasm is helpful. *Libido* or *sexual desire* is a psychological construct intended to explain the likelihood or strength of a sexual response. Constructs do not have the same observable qualities as, say, erection or ejaculation, but they are nevertheless presumed to exist and are invoked to explain variations in response frequency and intensity. In men, libido or desire is usually assessed through self-reports of interest in sexual activity and a sexual partner, the presence of self-generated fantasies, and the frequency of sexual activity (coitus, masturbation). At the neural level, libido represents a state of “arousability” that most likely involves “motivation” centers in the diencephalon (e.g., medial preoptic area; paraventricular nucleus of the hypothalamus), operating in conjunction with cortical level sensory and cognitive centers responsible for processing sexually relevant information about the environment (e.g., appropriate partner, appropriate time, etc.) (Pfaus, Kippin, & Coria-Avila, 2003). The presence of androgen, particularly testosterone, appears to be an important modulator of sexual desire in men, “priming” (i.e., lowering the threshold for) neural responsivity under specific contexts/conditions and to sexually relevant stimuli.

Sexual arousal involves both brain and genital activation

Given the appropriate stimulus conditions, the man will respond with *sexual arousal*, a process that involves both central (brain) and genital activation. The precise brain mechanisms for arousal appear to be centered in the hypothalamic and limbic areas, but whole brain processing of contextual stimuli (sensory input), emotional state (positive or negative), and past experience/future consequences (probable frontal lobe contribution) is important to the process. Arousal most probably involves autonomic (sympathetic?) activation (giving rise to “erotic feelings”) integrated with the aforementioned “motivation” and

cognitive processing centers that then regulate the descending neural impulses responsible for penile response.

Penile erection is a vascular process involving increased arterial inflow to the penis, penile engorgement with blood, and decreased venous outflow from the organ, processes that result in sufficient rigidity for sexual intercourse (Lue, 1992). Whether the penis is erect or flaccid depends upon the physiology of corporal cavernosal smooth muscle tone, that is, the equilibrium between proerectile and antierectile mechanisms controlling, respectively, relaxant and contractile responses of the smooth muscle cells comprising the penile blood vessels and cavernous tissue. Specifically, the erect penis results from *relaxation* of smooth muscle cells – the vasculature (arteries, arterioles, and capillaries) in the penis opens to allow the increased flow necessary for engorgement. The flaccid penis is characterized by *contraction* of smooth muscle cells – constricted vasculature limits the blood flow to the penis (Burnett, 1999).

In response to the descending neural innervation (probably parasympathetic), a number of events occur at the target *cell membrane* and the *intracellular level*. Specifically, second messenger molecules (e.g., cyclic guanosine monophosphate [cGMP] or cyclic adenosine monophosphate [cAMP]) and ions carry out the neural signal via the action of receptor proteins at the cell membrane of the target cell (e.g., smooth muscle) or via enzyme pathways. Regarding the latter process, these enzymatic pathways (e.g., phosphodiesterase) within the muscle cell may inactivate various pathways and therefore inhibit erectile function. Indeed, medications for erectile dysfunction (e.g., sildenafil [Viagra]) operate through these pathways. Such agents inhibit phosphodiesterase-5 (PDE-5), the enzyme that deactivates cGMP, the energy-consuming process that stimulates relaxation of corporal cavernosal tissue (and thus erection). As a result, cGMP remains active in increasing amounts to exert corporal smooth muscle relaxant effects (Boolel et al., 1996). Stated simply, these prosexual drugs for ED act by inhibiting the system that inhibits erection at the level of penile tissue.

Prosexual drugs for ED act by inhibiting the system that inhibits erection at the level of penile tissue

Ejaculation represents the sequencing of two reflexes under cerebral control that typically coincide with the high point of sexual arousal (Motofei & Rowland, 2005a; Rowland & Slob, 1997). Unlike erection, which may occur in the absence of direct penile stimulation, the ejaculatory reflexes generally require penile stimulation. The first reflex – emission – is a sympathetic response that closes the bladder neck (preventing urination and retrograde ejaculation) and stimulates excretion of seminal fluid (which mixes with sperm) from the prostate into the urethral tract. This first stage of ejaculation is associated with the “ejaculatory inevitability” that men experience prior to actual expulsion of the seminal fluid, and serves as the trigger for the second reflex. The second reflex – putatively involving the parasympathetic system and/or somatic motor system – involves the expulsion of the seminal fluid from the urethra, achieved through the rhythmic contractions of the bulbocavernosal and ischiocavernosal muscles (associated with anal sphincter muscle contraction). The subjective (brain) perception of these contractions, mediated through sensory neurons in the region, gives rise to the experience of orgasm, which comprises a distinct and separate loop. Thus, ejaculation can and does (rarely) occur without concomitant orgasm.

Subjective (brain) perceptions of the contractions that lead to the expulsion of seminal fluid lead to the experience of orgasm

Serotonin has been implicated in the trigger for ejaculation. Various anti-depressant drugs that affect the serotonin have been used to treat premature ejaculation

The mechanism that actually triggers the entire ejaculatory process is not well understood, but the brain neurotransmitter serotonin has been implicated. Accordingly, various antidepressant drugs that affect the serotonergic system – for example, tricyclics (Anafranil) and selective serotonin reuptake inhibitors (SSRIs [Prozac, Zoloft, etc.]) have been used fairly effectively to prolong intercourse in men who usually ejaculate very rapidly. Not surprisingly, since ejaculation is also mediated in part by the sympathetic nervous system, prescription and over-the-counter drugs that attenuate sympathetic response (and there are dozens) may interfere with a normal ejaculatory process.

The above summary makes it clear that the basic rudiments of sexual response are complex and not fully understood. Furthermore, it underscores the many possible points at which the process can go awry. Given the high level of psychophysiological integration required for coordinated sexual response, it is not surprising that sexual response, important as it is to procreation, is sensitive to a myriad of physiological and psychological factors.

1.4.2 Etiology of Male Sexual Dysfunction

Causes of sexual problems can be attributed to one or more sources, which are not mutually exclusive and which often overlap

The causes of sexual problems in men vary, but generally they might be attributed to one or more of four sources: physiological, psychological, relational, and sociocultural. These sources are, of course, overlapping domains and therefore represent convenient rather than mutually exclusive classifications. That is, a distressful relationship between the man and his partner may affect his psychological well-being, which in turn may influence his physiological response. Conversely, a man with a clear medical etiology responsible for diminishing erectile function may lose self-confidence and begin to avoid sexual intimacy, a situation that typically impacts the dyadic relationship.

Factors that contribute to development of a sexual dysfunction may be different from those that contribute to its maintenance

The etiological factors identified herein represent *potential* causes for problems, or “risk factors,” in that while they increase the likelihood of a sexual dysfunction, they do not determine it. Furthermore, the factors responsible for precipitating or predisposing a sexual problem may be quite different from those that eventually end up maintaining it. For example, failure to respond with an adequate erection due to stress or medication may result in anxiety and diminished self-confidence surrounding future sexual encounters, factors that may eventually come to maintain or even intensify the problem. Finally, there is a great deal of variation in how each of these sources (physiological, psychological, relational, and sociocultural) might affect any given individual, with clear etiologies for some dysfunctional states simply not fully elaborated. In a broader sense, however, it is often useful to conceptualize each man as having his own biopsychosocial vulnerabilities, such that the combination of factors resulting in sexual dysfunction for one man may be quite different from those for another man. In the following sections, a number of common risk, predisposing, and maintaining factors for male sexual dysfunction are discussed (see Table 3).

Pathophysiological-Based Risk Factors

Given the priming role of testosterone in sexual arousability in men, disruption of the hypothalamic-pituitary-gonadal (HPG) axis is likely to lead to

Table 3
Examples of Common Risk, Predisposing, and Maintaining Factors for Male Sexual Dysfunction Using a Biopsychosocial Approach

| Dysfunction | Pathophysiological | Psychological/ behavioral | Relationship |
|--|--|--|--|
| Erectile dysfunction | Tobacco use Diabetes mellitus Cardiovascular disease/hypertension Urinary tract disease Pelvic/spinal injury or trauma Chronic neurological disease Endocrine axis disturbances Various medications | Stress/emotional Depression/anxiety Performance anxiety Psychiatric disturbances Body image Arousal disorders | Partner dysfunction Hostility/anger Partner attractiveness |
| Premature ejaculation | Chronic neurological disease Pelvic/spinal injury or trauma Various medications Urinary tract disease Thyroid disease | Anxiety (general or specific) Novel situations or partners | Partner dysfunction Hostility/anger Control/dominance |
| Retarded/inhibited ejaculation | Thyroid disease Urinary tract disease Neuropathy Various medications, alcohol | Idiosyncratic masturbation Low arousal Anxiety | Partner attractiveness Partner dysfunction |
| Hypoactive sexual desire disorder | Androgen deficiency Dopaminergic disturbances Prolactin imbalance | Depression Psychiatric disturbances History of sexual abuse General life stressors | |

Note. Adapted from "Sexual Health and Problems: Erectile Dysfunction, Premature Ejaculation, and Orgasmic Disorders," by D. L. Rowland, in J. Grant & M. Potenza (Eds.), *Textbook of Men's Mental Health* (pp. 171–204). Arlington, VA: American Psychiatric Association Press, 2007.

loss of libido and sexual interest. Such problems, however, tend to be fairly uncommon and are typically accompanied by a variety of other physical/physiological symptoms. Although men having low or absent gonadal function (i.e., hypogonadal) may show little interest in sex, they are not necessarily impotent and may obtain erections when presented with certain kinds of psychosexual stimuli (see Bancroft, 1989). In general, however, these men are less likely to seek out or attend to sexual stimulation and fulfillment because of their hormone deficiency. As might be expected, diseases that interfere with neural control over the HPG axis (e.g., dopamine deficiency in Parkinson's disease) or that result in high levels of prolactin may also interfere with sexual desire.

In some instances, a cascading effect may occur such that decreased sexual interest results in reduced arousal and inability to maintain an erection.

Any condition, disease, or drug that diminishes responsiveness of the nervous, vascular, and smooth muscle (autonomic) systems has the potential to disrupt the “mechanics” of erection (Feldman, Goldstein, Hatzichristou, Krane, & McKinlay, 1994). Common risk factors for ED directly or indirectly compromise the process of smooth muscle relaxation (and arteriole dilation) necessary for penile engorgement. In addition, disruption of neural control due to neuropathy or chronic neurological diseases also accounts for erectile impairment. In some instances, the association between a specific condition (e.g., low levels of dehydroepiandrosterone [DHEA]) and ED has been documented, but the mechanism of action has not yet been clarified. Risk factors that impact erectile function may in some instances be additive, in that the greater the number of conditions present that affect the response system (e.g., smoking plus diabetes), the greater the probable impact on erectile function.

Unlike risk factors for ED, pathophysiological risk factors for ejaculatory disorders – premature and retarded/inhibited ejaculation – do not have easily identifiable common underlying pathways. Factors most often correlated with ejaculatory disorders, however, are procedures, diseases, or conditions that interfere with the neural integrity in the genito-pelvic region (see Section 1.6 Comorbidities).

Age and Medications

Inasmuch as aging serves as a proxy for a general increase in pathophysiological conditions, it is not surprising that men over 50 years are much more likely to report problems with erection (Feldman et al., 1994; Lewis et al., 2004). Although age may also impact ejaculatory function, the relationship between the two is not always straightforward. An increased prevalence of premature ejaculation (PE) seen in men 50 and older probably represents the development of coexisting erectile problems in those age groups; that is, the PE may develop in response (or be secondary) to age-related ED. The long-time supposition that rapid ejaculation is attenuated with age, although logically sound, has not been adequately tested in longitudinal studies, and therefore no firm conclusions can be drawn. On the other hand, given that penile sensitivity decreases substantially with age (Rowland, 1998), a tendency toward increased ejaculatory latencies with aging is not implausible and may account for the slight increase in the prevalence of IE in older men. Finally, as might be expected, various medications and recreational drugs may interfere with erectile and ejaculatory function, including many medications commonly used for depression, hypertension, and gastrointestinal disturbances. Comprehensive reviews of the impact of psychotropic agents on sexual function, many of which are used increasingly with age, are available (e.g., see Clayton & Balon, 2009).

The relationship between age and ejaculatory function is not always straightforward

Psychological Risk Factors for Male Sexual Dysfunction

In contrast to the pathophysiological risk factors noted above, the relationships among psychological risk or predisposing factors and sexual dysfunction are substantially more tenuous (though no less important) and more likely to affect the entire sexual response cycle rather than just specific components. Also, because psychological factors are many and varied, they have the potential