CHAPTER 11

ERECTION DISORDERS

Despite the current rhetoric. . . . about sex and intimacy's involving more than penile-vaginal intercourse, the quest for a rigid erection appears to dominate both popular and professional interest. Moreover, it seems likely that our diligence in finding new ways for overcoming erectile difficulties serves unwittingly to reinforce the male myth that rock-hard, ever-available phalluses are a necessary component of male identity. This is indeed a dilemma.

ROSEN AND LEIBLUM, 1992[1]

GENERAL CONSIDERATIONS

The Problem

A 49-year-old widower described erection difficulties for the past year. His 25-year marriage was loving and harmonious throughout but sexual activity stopped after his wife was diagnosed with ovarian cancer six years before her death. Their sexual relationship during the period of her illness had been meager as a result of her lack of sexual desire. Although he missed her greatly, he felt lonely since her death three years before and, somewhat reluctantly at first, began dating other women. A resumption of sexual activity soon resulted but much to his chagrin he found that in contrast to when he would awaken in the morning or masturbate, his erections with women partners were much less firm. He felt considerable tension, particularly because some months before, he had developed a strong attachment to one woman in particular and was fearful that the relationship would soon end because of his sexual troubles. As he discussed his grief over the loss of his wife and talked about his guilt over his intimacy with another woman, his erectile problems began to diminish.

A 67-year-old man, married for 39 years, and having a history of angina prior to a coronary by-pass operation three years before was referred to a "sex clinic" together with his wife because of his erectile difficulties. Sexual experiences had been enjoyable and uncomplicated for both until he developed angina at the age of 62. Orgasm provoked his chest pain. Nitroglycerin was prescribed but he used it only occasionally because it resulted in headache. His angina during sexual activity was frightening to his wife who, nevertheless, recognized the importance of sexual

experiences in his life and supported his desire to continue being sexually active. Cardiac surgery resulted in the disappearance of his chest pain. However, some months before his operation, he began to experience difficulty becoming fully erect at any time, and would frequently lose whatever fullness he had before vaginal entry occurred. His erectile difficulties with his wife had become persistent and when questioned, it was apparent that his morning erections were not different. Sildenafil (Viagra) was dismissed as a treatment possibility because of his occasional use of nitroglycerin. He was referred to a urologist for intracavernosal injections.

Terminology

The phrase "erectile dysfunction" has provided competition for the more popular word "impotence." The latter has a tenacity for usage that does not exist for the female equivalent and now rarely-seen word, "frigidity." Both words have similar deficiencies: they are so broad in usage they (1) incorporate disorders of desire and function and (2) imply something pejorative about the patient's personality quite apart from their sexual expression.[2]

The social confusion surrounding the word "impotence" is, perhaps, exemplified by the first recommendation of the National Institutes of Health Consensus Statement on Impotence, which was to change the term *impotence* to *erectile dysfunction* as a way of characterizing "the inability to attain and/or maintain penile erection sufficient for satisfactory sexual performance."[3] (Interestingly, no conference was necessary to change usage of the word "frigidity").

Mechanism of Erection

The fundamental element in the development of an erection is the trapping of blood in the penis. The mechanism by which this occurs was described by Lue and Tanagho (Figure 11-1). A human penis has three cylinders: Paired corpora cavernosa (CC) on the dorsal surface, and the completely separate corpus spongiosum (CS), which carries the urethra and is responsible for the ventral bulge.

The CS anatomically includes the glans of the penis. The CC are each surrounded by an inflexible envelope of fibrous tissue: the tunica albuginea (TA). The CS has a much thinner TA and is connected to the glans, which has almost none.

As an erection develops, the smooth muscle around the arterial tree and walls of the sinusoids relaxes, increasing the inflow of blood into the penis and allowing more blood to remain. While expansion occurs, the venules are compressed between the sinusoids and TA, thereby stopping the outflow and in effect trapping blood in the sinusoids of the penis.

Blood is carried to the penis by the two internal pudendal arteries and within the penis by paired cavernosal arteries. The latter subsequently divide into smaller vessels (arterioles), which are surrounded by smooth muscle. The same can be said of the helicine arteries (small spiral shaped arteries). In the CC and CS, blood is then carried to interconnecting sinusoids (microlakes, which have the appearance of a sponge when filled but are mostly collapsed when a penis is flaccid), which are also surrounded by smooth muscle. Small veins (venules) carry blood away to the emissary veins, which in turn pierce the TA.

As an erection develops, there is relaxation of the smooth muscle around the arterial tree and walls of the sinusoids, increasing the inflow

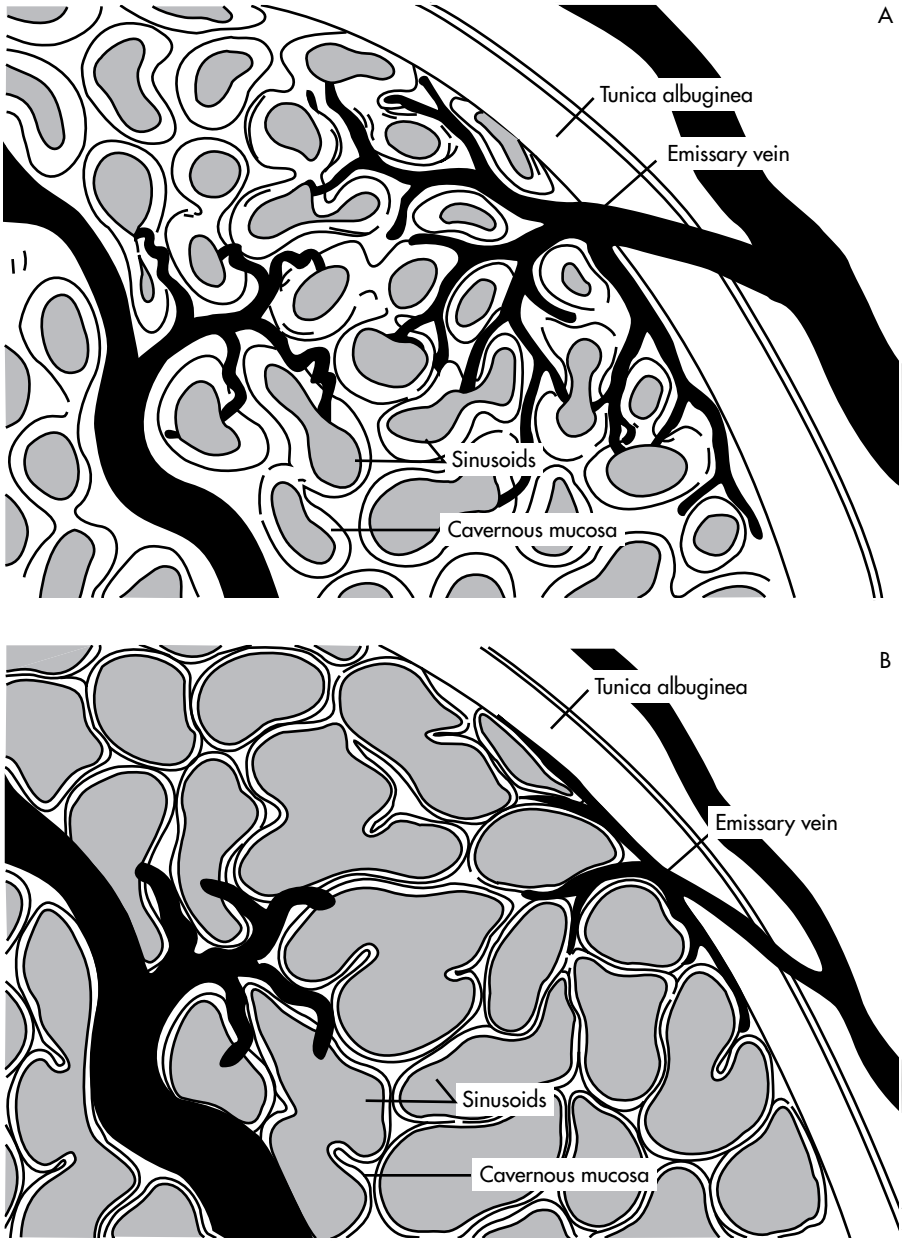

Figure 11-1 The mechanism of penile erection. In the flaccid state (**A**), the arteries, arterioles, and sinusoids are contracted. The intersinusoidal and subtunical venular plexuses are wide open, with free flow to the emissary veins. In the erect state (**B**), the muscles of the sinusoidal wall and the arterioles relax, allowing maximal flow to the compliant sinusoidal spaces. Most of the venules are compressed between the expanding sinusoids. Even the larger intermediary venules are sandwiched and flattened by distended sinusoids and the noncompliant tunica albuginea. This effectively reduces the venous capacity to a minimum. (From Lue TF: *Male sexual dysfunction*. In Tanagho EA, MaCninish JW: *Smith's general urology*, Stamford, 1992, Appleton & Lange, p. 669.)

of blood into the penis and allowing more blood to remain. While expansion occurs, the venules are compressed between the sinusoids and TA, thereby stopping the outflow and in effect trapping blood in the sinusoids of the penis. "The smooth muscles in the arteriolar wall and trabeculae surrounding the sinusoids are the controlling mechanism of penile erection."[4]

(Biochemical aspects of erection are discussed in the treatment section of " Generalized Erectile Dysfunction: Organic, Mixed, or Undetermined Origin" below in this chapter).

Definition

The main difficulty with the definition of erectile dysfunction is whether the diagnosis of erectile problems should refer only to the hardness or softness of a man's erection or if it should also include a behavioral component. For example, should a man who has erections that are persistently partial but whose penis is sufficiently enlarged to regularly engage in intercourse be designated as having an "erectile disorder?" If that same man designates himself as "impotent," what should be the diagnostic position of the health professional? Should there be a subjective component to an erectile disorder: does it make any difference what the man (or his partner) thinks? Is the fullness of a man's penis in intercourse all that matters? Is intercourse the only sexual activity on which the definition is based? What about erections with other sexual practices? These, and other questions, are not intellectual exercises but daily clinical quandaries.

Classification

Erectile dysfunction in all situations, including the lack of nocturnal erections, strongly suggests that a general medical condition or substance use is the cause.[5]

DSM-IV-PC summarized the criteria for the diagnosis of "Male Erectile Disorder" as follows: "persistent or recurrent inability to attain, or to maintain until completion of the sexual activity, an adequate erection, causing marked distress or interpersonal difficulty"[5](p. 116). The clinician is further instructed to "especially consider problems due to a general medical condition . . . such as diabetes or vascular disease, and problems due to substance use . . . such as alcohol and prescription medication. *Erectile dysfunction in all situations, as well as lack of nocturnal erections, strongly suggests that a general medical condition or substance use is the cause*"(italics added).

The subclassification of Erectile Disorders used in this chapter is summarized in Figure 11-2.

Epidemiology

The Massachusetts Male Aging Study (MMAS) provided revealing information about erectile function, dysfunction, and "potency" in middle-aged and older men.[6] The study was conducted in the late 80s, was concerned with health and aging in men, was community-based, and involved a random sample of noninstitutionalized men 40 to 70 years old. Individuals who completed a self-administered questionnaire on sexual function and activity included 1290 (75%) of the 1709 MMAS subjects. "Potency" was subjective in that it was defined by those who completed the questionnaires. Defined as "satisfactory functional capacity for erection," "potency" could "coexist with some

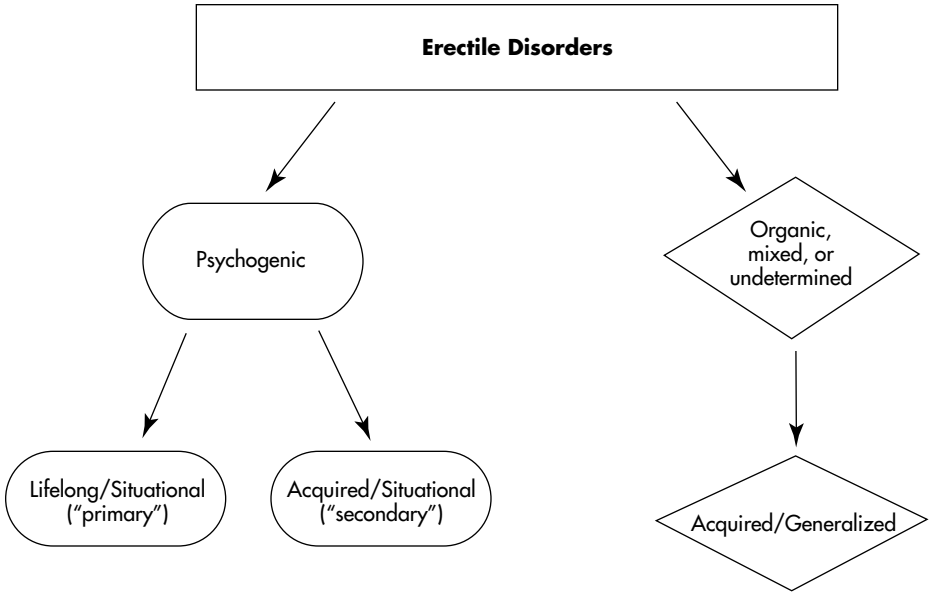

Figure 11-2 Classification of erectile disorders.

degree of erectile dysfunction in the sense of submaximal rigidity or submaximal capability to sustain the erection." Four degrees of "impotence" were described:

- None
- Minimal
- Moderate
- Complete

Sixty percent of men were not impotent at age 40 compared to 33% not impotent at age 70 years.

The overall prevalence of impotence in this study was found to be 52%, with 15% defined as minimal, 25% moderate, and 10% complete. Prevalence was highly related to age with the probability of moderate impotence doubling from 17% to 34% and complete impotence tripling from 5% to 15% between subject ages 40 to 70 years. Looking at this from the opposite perspective, 60% of men were *not* impotent at age 40 years, compared to 33% *not* impotent at age 70 years.

The frequency of erectile problems found in health care settings seems to depend somewhat on the clinical context. That is, different percentages can be found in various clinical settings: medical outpatients, urology, and sex therapy[7] (p. 11). In a review of the frequency of sexual problems presented to "sex clinics" between the mid-70s to 1990, 36% to 53% of men complained of "male erectile disorder."[8] Masters and Johnson subcategorized this diagnosis into "primary" and "secondary".[9] The former referred to a man who had never had intercourse (p. 137), and the latter referred to a man who had been able to have intercourse at least once in the past

(p.157). Of all the men with "impotence" who consulted Masters & Johnson, 13% had the primary form (p. 367).

Etiology

"Repeatedly bandied about is the hackneyed declaration that in the 1970s, mental health professionals pronounced 90% of impotence to be psychogenic; more recently urologists proclaim that 90% of impotence is organic. Both sides are wrong, not just for the disrespectful attitudes toward one another, but for failing to develop more sophisticated notions of etiology."[10]

LoPiccolo saw clinical limitations to the either/or approach and suggested an alternative way of thinking about the etiology of erectile dysfunctions: that organic and psychogenic factors be viewed as two "separate and independently varying dimensions" and that *both* should be examined in *each* instance.[11] To support this position, he reported on 63 men with erectile difficulties who were carefully and thoroughly investigated in both areas. Ten men were found to have a purely psychogenic etiology, and three men were found to have a purely organic etiology. The majority of men in this study (50/63) displayed a mixture of factors, indicating that a "two-category typology was . . . inappropriate." Furthermore, almost one third of the men (19/63) had "mild organic impairments" but "significant psychological problems." These men might have been considered "organic" in a two-part etiological scheme, however, they might also have been sufficiently responsive to psychological intervention such that physical treatment may not have been necessary.

In a diagnostic and a therapeutic sense, the implication of LoPiccolo's approach is quite serious. It means that even if a factor that is of *potential* etiological significance is found (biological or psychological), it is not necessarily *the* factor. Or, in other words, "the detection of some possible etiological factor . . . does not mean that the cause . . . has been fully explained. Such a factor may even be coincidental, of no (actual) etiological significance."[12]

The possible nature of the interrelationship between biological and psychological factors was suggested as the following: "When any one (organic factor) occurs in isolation, it may serve to make erections more vulnerable to emotional disturbances and sympathetic overactivity, facilitating the vicious circle of performance anxiety that maintains ED."[12]

Investigation

History

History-taking is an indispensable element in the investigation of erectile disorders and provides direction for further exploration and treatment. Issues to inquire about and questions to ask include:

1. Duration (see Chapter 4, "lifelong versus acquired")

Suggested Question: "**HAVE YOU ALWAYS HAD DIFFICULTIES WITH ERECTIONS OR IS THIS A RELATIVELY NEW PROBLEM?**"

2. Partner-related erections (see Chapter 4, "generalized" versus "situational")

Suggested Question: "**WHAT ARE YOUR ERECTIONS LIKE WHEN YOU ARE WITH YOUR WIFE (PARTNER)?**"

3. Sleep [including morning] erections (see Chapter 4, "generalized versus situational")

Suggested Question: "**WHAT ARE YOUR ERECTIONS LIKE WHEN YOU WAKE UP IN THE MORNING?**"

Additional Question: "**DO YOU WAKE UP AT NIGHT FOR ANY REASON?**"

Additional Question if the Answer is Yes: "**WHAT ARE YOUR ERECTIONS LIKE WHEN YOU WAKE UP AT NIGHT?**"

(Comment: the assessment value of asking about sleep-related erections is generally recognized but not universally accepted.[13] When full sleep-related erections exist, the information seems highly useful from a diagnostic viewpoint. However, partial or nonexistent sleep erections are not necessarily meaningful since this situation may coexist with daytime erections firm enough for intercourse.)

4. Masturbation erections (see Chapter 4, "generalized versus situational")

Suggested Question: "**WHAT ARE YOUR ERECTIONS LIKE WHEN YOU STIMULATE YOURSELF (OR MASTURBATE)?**"

5. Fullness of erections (see Chapter 4, "description")

Suggested Question: "**ON A SCALE OF ZERO TO TEN WHERE ZERO IS ENTIRELY SOFT AND TEN IS FULLY HARD AND STIFF, WHAT ARE YOUR ERECTIONS LIKE WHEN YOU ARE WITH YOUR WIFE (PARTNER)?**"

Additional Question: "**USING THE SAME SCALE, WHAT ARE YOUR ERECTIONS LIKE WHEN YOU WAKE UP IN THE MORNING?**"

Additional Question: "**IF YOU WAKE UP DURING THE NIGHT, USING THE SAME SCALE, WHAT ARE YOUR ERECTIONS LIKE AT THAT TIME?**"

Additional Question: "**USING THE SAME SCALE, WHAT ARE YOUR ERECTIONS LIKE WITH SELF-STIMULATION OR MASTURBATION?**"

Additional Question Under All Three Circumstances: "**ABOUT HOW LONG DO YOUR ERECTIONS LAST?**"

(Comment: Even though erections may be full under all three circumstances, the duration of erections may be important. Erections may consistently be short-lived—a matter of diagnostic significance, since that observation may indicate a "venous leak").[13]

6. Psychological accompaniment (see Chapter 4, "patient and partner's reaction to problem")

Suggested Question: "**WHEN YOU HAVE TROUBLE WITH YOUR ERECTION, WHAT'S GOING THROUGH YOUR MIND?**"

Additional Question: "**WHAT DOES YOUR WIFE (PARTNER) SAY AT THESE TIMES?**"

In men with erectile difficulties, physical examination is essential even if the "yield is low."[12] Many patients feel that they have not been properly assessed or taken seriously if there is no physical examination, and they may refuse a psychogenic diagnosis as a result.[12]

Physical Examination

In men with erectile difficulties, physical examination is essential even if the "yield is low."[12] "Without it many patients feel that they have not been properly assessed or taken seriously and they may refuse a psychogenic diagnosis as a result".[12] The physical examination concentrates particularly on the endocrine, vascular and neurologic systems, as well as local genital factors.

Signs of hormonal abnormalities include the following[14] (p. 85):

1. Testicular atrophy
2. Gynecomastia
3. Galactorrhea
4. Visual field abnormalities
5. Sparse body hair
6. Decreased beard growth
7. Skin hyperpigmentation
8. Signs of thyroid abnormalities
9. Low energy level and lack of "well-being"

Signs of vascular disease include the following[14] (p.91):

1. Weak pulses in legs or ankles
2. Hair loss on lower legs
3. Unusually cool temperature of penis or lower legs
4. High lipid levels
5. High cholesterol levels
6. Duputyren's contractures [Peyronie's disease only]
7. Fibrosis of outer ear cartilage [Peyronie's disease only]

Signs that indicate neurological factors include the following[14] (p. 93):

1. Weak or absent genital reflexes (bulbocavernosus, cremasteric, scrotal, internal anal, and superficial anal)

2. Neurological abnormalities in the S2 to S4 nerve root distribution
3. Reduced penile sensory thresholds to light touch electrical stimulation or vibration

An investigation conducted in a medical outpatient clinic found that the physical examination rarely helped to differentiate various etiological factors with two exceptions[15]:

- Small testes in patients with primary hypogonadism
- Peripheral neuropathy in patients with diabetes

Laboratory Investigation

The extent of a clinician's use of the laboratory in the investigation of erectile dysfunction depends on the results of the history and physical examination (see "Investigation" below in this Chapter in the sections on "Situational ['psychogenic'] Erectile Dysfunction" and "Generalized Erectile Dysfunction: Organic, Mixed, or Undetermined Origin."

Treatment

Failure to attend to psychological issues is indefensible. The potential effect of erectile difficulties on mood state, self-esteem, and self-efficacy (as well as the couple's relationship) cannot be overemphasized.[16]

As LoPiccolo has shown, psychological and physiological factors are present in the vast majority of men with an erectile disorder.[11] "Psychological" factors include social, cultural, religious, and interpersonal elements, and those within the person. Since *all* sexual behavior of men is influenced to a great degree by these issues, it is reasonable to assume that these factors are present in the context of erectile difficulties as well. The logical result of LoPiccolo's research is the concept that regardless of the etiology of a man's erectile difficulties, a health care clinician must always attend to universally concomitant psychological issues. That is: "Given the critical role of psychological factors, even in cases with clearcut organic etiology, *the failure to attend to psychological issues is indefensible* (italics added). The potential impact of erectile difficulties on mood state, self-esteem and self-efficacy, as well as on the couple's relationship cannot be overemphasized."[16]

SITUATIONAL ("PSYCHOGENIC") ERECTILE DYSFUNCTION

The assessment of situational erectile disorders is summarized in Figure 11-3.

Description

Lifelong ("Primary") Erectile Disorders

In this unusual syndrome, the man reveals that all, or most, attempts at intercourse result in diminution of his erection before attempts at vaginal entry. Levine reasonably suggested that the definition of the disorder be "liberalized" to include men who gain vaginal entry "occasionally"[17] (p. 208), Typically, the man has no difficulty obtaining full erections when alone, with masturbation, or when awakening. Ejaculation and orgasm have been similarly unimpaired. The sexual desire phase may have been problematic if thoughts associated with sexual arousal were atypical (as is

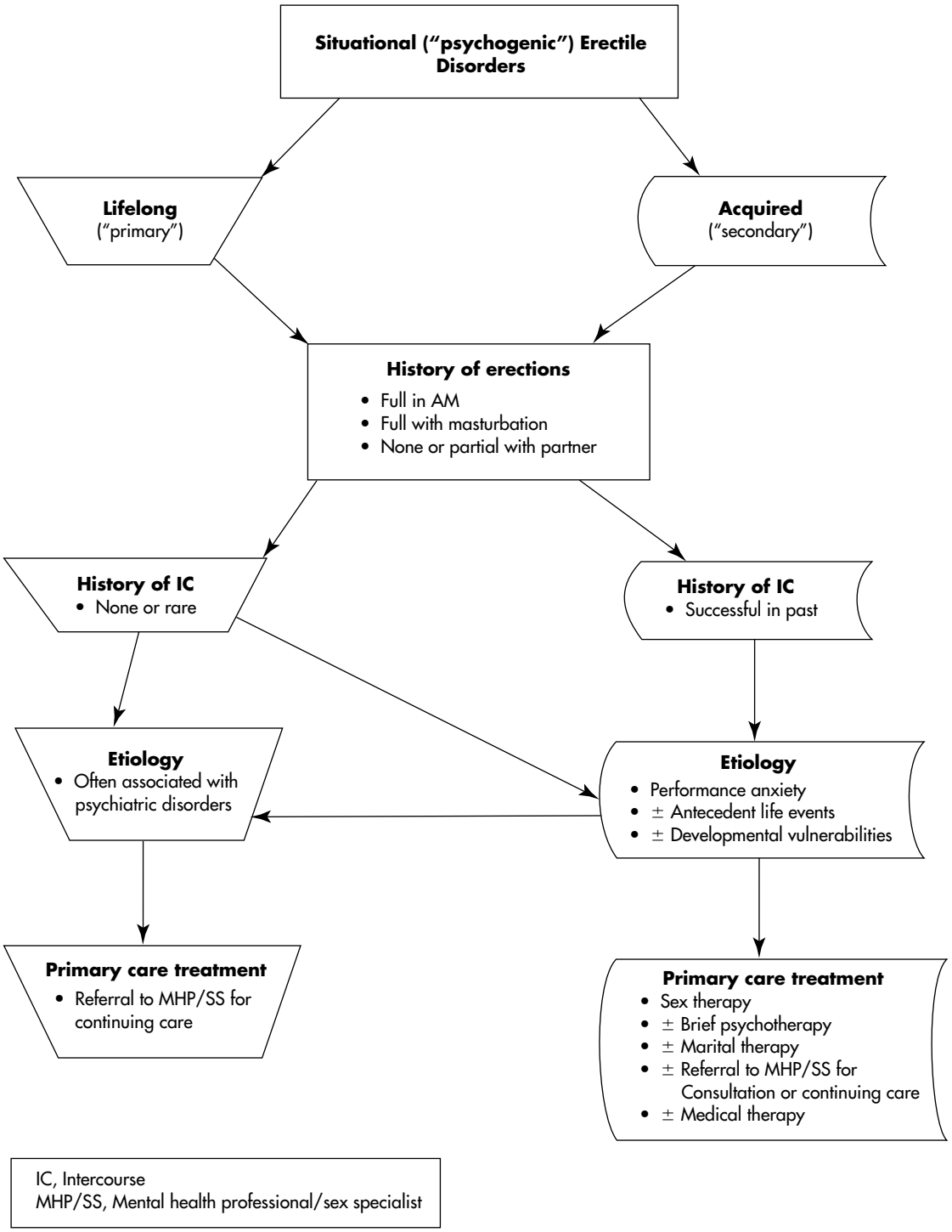

Figure 11-3 Assessment of situational erectile disorders.

often the case), such as fantasies related to paraphilias. Since behavior connected to such fantasies is often easier to carry out alone, such men tend to avoid intimate relationships and may depend on prostitutes (with whom they can be more candid) for partner-related sexual experiences. Even then, intercourse rarely, if ever, occurs. Pressure from a (non-prostitute) partner may be a major factor in seeking treatment. When this occurs, the patient may not be particularly forthcoming about his thoughts and feelings.

A 32-year-old man was seen with his 29-year-old wife. They were married for two years and despite being sexually active with one another several times each week, intercourse never occurred. (They previously agreed not to engage in intercourse before they married.) She was aware of the fact that he had never experienced intercourse in the past. His erection predictably diminished whenever he moved close to her vagina. She wanted to become pregnant and felt the "biological clock ticking."

Her desire for pregnancy and her love for her husband resulted in a single-minded pursuit of her attempts at solving their sexual difficulties. He was less enthusiastic. Attempts at psychotherapy with him alone and sex therapy as a couple proved unhelpful. Since he was a shy person and spontaneously revealed little about himself, he never previously told anyone about having been repeatedly sexually assaulted as a child by his mother. Nor had he ever discussed his current sexual fantasies (about which he was quite ashamed) that involved the insertion of a knife into a woman's vagina. He was again referred for individual psychotherapy and accepted the need for candor with his therapist concerning his sexual experiences as a child and his current sexual thoughts and feelings.

Acquired ("Secondary") Erectile Disorders

In contrast to the lifelong form of situational erectile dysfunction, the patient reports having had intercourse in the past, perhaps on many occasions for many years. However in the present, full erections might occur with his partner before clothes are removed but the fullness may diminish after he reaches the bed or after the commencement of sexual activity. Intercourse might occur sometimes but this seems unpredictable. Characteristically, he never had a problem obtaining full erections after a period of sleep and with masturbation and, as well, describes no difficulty with ejaculation and orgasm now or in the past. History reveals that when younger, he frequently had erection troubles with partners on the first few occasions when sexual intercourse was attempted. However, when in a long-term relationship, he functioned well sexually although erectile difficulties occasionally reappeared at times of "stress." After a relationship of many years, doubts about his sexual "performance" developed. There may have been a marked diminution of sexual activity in spite of his partner's attempts at reassurance. She believed him not to be sexually interested, and wondered about her own attractiveness to him.

Questioning revealed that his apparent sexual disinterest is actually avoidance. He remains privately interested but feels that he is not "a man" anymore with his wife.

A 51-year-old rather shy man was seen together with his 49-year-old outspoken wife. They were married for 23 years. Sexual activity had never been a problem until about five years ago when his erections sometimes became soft after vaginal entry, so much so that intercourse could not continue. This sequence of events, and erectile loss even before intromission, gradually occurred more often and culminated in the complete lack of intercourse in the previous six months. His sexual desire, while never as strong as that of his wife, had not changed and he would masturbate (without erectile problem) and ejaculate about once or twice each month. He thought that the origin of his erection troubles were mainly related to his age but he also wondered if his substantial use of alcohol for the previous 25 years was also a factor. His heavy drinking stopped completely about five years ago when he joined AA. This was about the same time that his erection problems began. After discussion of possible etiological factors, he understood that much of his erectile difficulty was connected to his feelings about his wife. They were referred to a treatment program that focused on both their marriage and their sexual relationship.

Etiology

Lifelong ("Primary") Erectile Dysfunction

This syndrome is "often, though not invariably, associated with a diagnosable major [psychiatric] condition"[18] (p. 133). Masters and Johnson described a group of 31 men with "Primary Impotence," eleven of whom were in unconsummated marriages[9] (pp. 137-156). Factors they considered to be of etiological significance were multiple and included the following:

1. Homoerotic desire
2. Mother/son incest
3. Strict religious orthodoxy
4. Psychologically damaging attempts at first intercourse with a prostitute (sometimes associated with drugs or alcohol)

Other investigators also reported associated paraphilias and gender identity disorders[18] (pp. 133-135);[19] (p. 245).

In almost all men with acquired erectile dysfunction, the etiology involves a combination of factors[17] in three areas:

1. Performance anxiety
2. Antecedent life events
3. Developmental vulnerabilities

Acquired ("Secondary") Erectile Dysfunction

Most men with a clearly situational erectile dysfunction also indicate that it is acquired.

In almost all such men, the etiology involves some combination of factors[17](p. 200) in the following three areas:

1. Performance anxiety

2. Antecedent life events
3. Developmental vulnerabilities

The "phase of time" for each of these three is, respectively, here-and-now lovemaking, months to years ("recent" history), and childhood/adolescence ("remote" history).

A major here-and-now issue is "performance anxiety," a concept introduced by Masters and Johnson to describe the worry that a patient may have about his or her sexual function and whether it will be similarly impaired on a current occasion as it was at a previous time.[9] Performance anxiety is partner related and probably universal in men with erectile difficulties. From a primary care perspective, performance anxiety is an important target of the treatment of "psychogenic impotence" in both solo men and couples. However, this component explains only part of the etiology of this syndrome, since eliminating performance anxiety does not always result in cure.

Antecedent life events and developmental vulnerabilities may be of therapeutic significance also, but they are difficult to consider in detail in a primary care setting.

The former "typically fall into one of five categories"[17] (p.202):

1. Deterioration in the nonsexual relationship with a spouse or partner
2. Divorce
3. Death of a spouse
4. Vocational failure
5. Loss of health

Developmental vulnerabilities include such issues as child sexual abuse and impairments in sexual identity.[17]

On a clinical level, one frequently has the impression of a link between psychogenic erection difficulties and difficulty with expression of anger. In the MMAS study, the suppression and expression of anger was assessed. "Men with maximum levels of anger suppression and anger expression showed an age-adjusted probability of 35% for moderate impotence and 16% to 19% for complete impotence, both well above the general level (9.6%)."[6] The MMAS study did not subcategorize men with "impotence" according to whether the origin was "psychogenic," "organic," mixed," or "undetermined." It may certainly be possible that problems with anger may also potentiate some of the etiological factors associated with erectile difficulties of organic, mixed or undetermined origin discussed below.

Performance anxiety is partner related and probably universal in men with erectile difficulties.

Eliminating performance anxiety does not always result in cure.

Antecedent life events typically fall into one of five categories[17]:

1. Deterioration in the nonsexual relationship
2. Divorce
3. Death of a spouse
4. Vocational failure
5. Loss of health

Developmental vulnerabilities include child sexual abuse and impairments in sexual identity.[17]

Laboratory Investigation

If in instances where the man reports being otherwise healthy, the history clearly indicates the situational nature of the man's erectile dysfunction, there is no sign of any contributory physical abnormality, and there are no other sexual symptoms (such as lack of sexual desire), little needs to be done to obtain additional specific laboratory data.

Treatment

"It is fortunate for many psychologically impotent men that a complete understanding of the causes is not necessary. Some men spontaneously get over their problem within a short period of time without any therapy"[17] (p. 202). For those whose problem is not solved, an approach is proposed that concentrates on five themes that have been identified in a review of the literature (Box 11-1).[16] Most importantly for generalist health professionals, some of these five themes listed in Box 11-1 can be easily integrated into primary care.

The first theme is accurate information and realistic ideas and expectations regarding sexual performance and satisfaction—all of which is a problem in many men with erectile difficulties (and their women partners).

Areas to be addressed include:

1. Genital anatomy and physiology
2. The sex response cycle
3. Masturbation
4. Male-female differences in sexual response
5. Effect of aging, illness, and medication on sexual desire, arousal, and orgasm

Areas to be addressed include:

1. Genital anatomy and physiology
2. The sex response cycle
3. Masturbation
4. Male-female differences in sexual response
5. Effects of aging, illness, and medication on sexual desire, arousal, and orgasm

Comprehensive, inexpensive, and up-to-date self-help books are easilly vailable and can be used as an adjunct in this education process.[20] The provision of information can correct unrealistic ideas and expectations—thoughts that could, themselves, significantly interfere with erectile function. For example, the "gold standard" of erectile function for many men is what occurred in their teenage years. The folly of "living in the past" becomes evident to a man in his (for example) 40s when he is asked to provide another example of a part of his body that functions in the present as it did when he was a teenager. In addition, it could be pointed out to him that he is, in effect, basing his sexual expectations for his 60 years of adult sexual function (approximate life expectancy minus 15 years of pre- and early

Easily-available, comprehensible and comprehensive, inexpensive, and up-to-date self-help books can be used as an adjunct in this educational process.[20]

The "gold standard" of erectile function for many men is what occurred in their teenage years. The folly of "living in the past" becomes evident to a man in his (for example) 40s when he is asked to provide another example of a part of his body that functions in the present as it did when he was a teenager.

Box 11-1 Themes in the Treatment of Situational ("Psychogenic") Erectile Dysfunction

- INFORMATION, including realistic ideas and expectations concerning sexual performance and satisfaction
- PERFORMANCE ANXIETY RELIEF through use of "sensate focus"
- "SCRIPT" MODIFICATION ('who does what to whom')
- ATTENTION TO RELATIONSHIP ISSUES (e.g., intimacy, control, conflict resolution, trust)
- RELAPSE PREVENTION

adolescence) on the five (or so) years of erectile experience as a teen! Other examples of ideas and expectations that might be discussed with a patient include his thoughts when he or his partner is initiating sexual activity and when his penis is becoming firmer or softer.

Yet another example of the therapeutic value of information is that of the sexual changes associated with aging. The educational effect on the treatment of erectile dysfunction was studied in a group of heterosexual couples between the ages of 55 and 75.[21] Investigators found that a four-hour workshop resulted in increased knowledge, especially about the normal changes that occur with age, thereby allowing participants to have more realistic expectations of themselves and their partners. Sexual satisfaction also increased despite the presence of associated organic factors.

The second theme is the relief of "performance anxiety." Diminishing or eliminating this frequently appearing factor involves inducing sexual response in the man (in this instance, erection) while he paradoxically avoids sexual invitations for intercourse. Masters and Johnson described this approach to the treatment of performance anxiety[9] (pp. 193-213). The method involves couple oriented touching "exercises" and concentrates on *sensate focus*, a term they coined (pp. 71-75) to denote a focus on immediate sensation rather than sexual goals of, for example, intercourse. Briefly, the exercises occur in stages and initially exclude intercourse and touching of breasts (in the woman) and genitalia, then include touching of the previously barred areas (while maintaining the exclusion of intercourse), and finally include unrestricted touching and intercourse. Couples do not move to the next level of the exercise until the previous one is mastered. While requiring repeated visits, this technique is not complex and might, therefore, be within the boundaries of primary care (depending on the clinician's time, comfort, and interest and the availability of specialists to whom one could refer).

Rather than the communication exercise it is, sensate focus is sometimes mistakenly thought of as a way of allowing one to discover previously unappreciated physical feelings in particular body areas.

One major (and often unappreciated) objective of "sensate focus" in the treatment of erectile dysfunction is change in the communication pattern between partners so they could, with "permission" (i.e., encouragement), and with a minimum of tension and embarrassment, tell one another what is, and is not, pleasurable. (Rather than the communication exercise it is, sensate focus is sometimes mistakenly thought of as a way of allowing one to discover previously unappreciated physical feelings in particular body areas.) A second objective of "sensate focus" is to remove the demand for intercourse. Since the man does not "need" an erection for any purpose other than intercourse and intercourse is not to take place, theoretically the "pressure" on the man to "perform" will be removed and the worry (which is thought to inhibit his erection) will disappear, thus allowing his erection to develop unhindered.

Two obstacles to sensate focus have been described[11] (p. 189). First, the passive process of sensate focus is contrary to the need of aging men for active and direct penile stimulation for an erection to develop. Second, the idea of performance anxiety is so popular that general knowledge of the concept has rendered its treatment less effective. Consequently, LoPiccolo coined the term *metaperformance anxiety* to explain

Functional men can become aroused on demand, whereas the same request in dysfunctional men results in interference with the arousal process.

why, on some occasions, "eliminating performance anxiety does not lead to erection during sensate focus body massage"[11] (p. 189).

Recently the role of "anxiety" in producing erectile troubles and the expected relief with its disappearance has been reexamined and reviewed from a research rather than clinical viewpoint.[22] Functional and dysfunctional men have been shown to respond differently to anxiety. The results of these studies are summarized as follows:

1. Functional men can become aroused on demand, whereas the same request in dysfunctional men results in interference with the arousal process (similar results were found in laboratory studies)
2. Functional men report their subjective arousal to be greater than dysfunctional men regardless of what occurs physically
3. (Particularly interesting from a therapeutic viewpoint) functional men report distraction to be an obstacle to sexual response, whereas this is neutral or actually helpful to dysfunctional men[16]

Functional men report distraction to be an obstacle to sexual response, whereas this is neutral or actually helpful to dysfunctional men.[16]

The third theme concentrates on sexual "script" modification (i.e., changes to what actually occurs sexually between two people). The *fourth theme concentrates on relationship issues* such as intimacy, control, conflict resolution, and trust. The *fifth theme concentrates on the prevention of relapse*. Since the third, fourth, and fifth areas are often more within the interests, practice pattern, and skills of the sex therapist, they are not discussed at length here.

Little published information exists on the treatment of situational erectile dysfunction by methods usually reserved for occasions when the etiology is "organic, mixed, or undetermined" (see below in the chapter). Few quarrel with the concept of considering such an approach when psychologically-oriented methods have been unsuccessful. However, when medical techniques are used early in the course of treatment, the concept is more problematic. The rationale sometimes given is one of providing the man an opportunity to have a erection in worry free circumstances as a way of overcoming an undefined obstacle. The rationale continues that after the man engages in successful sexual experiences that require an erection he will be able to do so without extra support.

A study of the use of intracavernosal injections in 15 men with "psychogenic impotence" did not convey a sense of optimism about the outcome of such an approach.[23] The authors concluded that performance anxiety was not alleviated, that dependence on injections for intercourse remained, and that the capacity for intimacy did not improve.

A study of the use of intracavernosal injections in 15 men with "psychogenic impotence" did not convey a sense of optimism about the outcome of such an approach.[23] The authors concluded that performance anxiety was not alleviated, that dependence on injections for intercourse remained, and that the capacity for intimacy did not improve. One can well imagine that the consequences (benefits and disappointments) of the use of such treatments for men with situational erectile difficulties become magnified when men who have these problems ask for, and are given, an oral medication such as sildenafil (see below in the chapter).

Few long-term follow-up studies have been conducted on the treatment of erectile dysfunction. Results for "primary" and "secondary" (i.e., acquired) erectile dysfunction were reported by Masters and Johnson as an "overall failure rate" (OFR) and were based on personal interviews conducted five years after the patients were

originally treated[9] (p. 367). The OFR for "primary impotence" was 41%. This modest improvement supports the clinical experience of greater complexity in the treatment of this form of the erectile dysfunction syndrome. Furthermore, it suggests that insofar as "primary" impotence is concerned, a focus on performance anxiety without considering other factors will likely result in quite limited gains.

The OFR reported by Masters and Johnson for the "secondary" form was 31%.[9] Another follow-up study in the United States, carefully conducted after three years, found that of the 18 men presenting with "difficulty achieving or maintaining erection," ten maintained the improvement, four were the same, and three were worse.[24] The authors found that there was "significant improvement maintained across time in erectile capability during intercourse . . . . improved satisfaction in the sexual relationship . . . [and] . . . longer duration of foreplay." Hawton and his colleagues conducted a rigorous one to six year follow-up study in the United Kingdom and found that the "gains made during therapy by couples who presented with erectile dysfunction were reasonably well sustained."[25] Of the 18 couples who undertook treatment, 14 reported the problem resolved or mostly so at the end of therapy, and 11 reported the same at follow-up.

Indications for Referral for Consultation or Continuing Care by a Specialist

1. Since the "primary" form of situational erectile disorders is so often associated with complex individual diagnosable psychiatric conditions rather than interpersonal conflicts, referral to a mental health professional for individual treatment is usually the most reasonable course of action[19] (p. 245). If the health professional is not also a sex-specialist, it may be useful to consult with one before proceeding with the referral.
2. Solo men with the "secondary" form of situational erectile dysfunction (i.e., those without a partner, with an uncommitted partner, in a relationship that is filled with so much discord that they are unable to cooperate with each other, or who have been raised in a culture in which men are clearly in control and women entirely submissive) often require an amalgam of traditional psychotherapy and sex therapy. Such men are candidates for individual care with a sex-specialist who is also a mental health professional.
3. Couples in which the man has the "secondary" form of situational erectile dysfunction and who would benefit by a here-and-now focus on information and performance anxiety (previously described in the treatment of situational problems in this chapter) could be cared for in primary care. Couples who do not respond to this approach may require an additional focus on two of the other elements: "script" modification and attention to relationship conflicts. Given the time and experience involved in providing these other components, referral would be reasonable in these circumstances. If referral does take place, the health professional should be a sex-specialist who also has clinical experience in the mental health area.
4. Consultation with a sex-specialist is warrented when consideration is given to providing a form of treatment usually reserved for men with erectile dysfunction of "organic, mixed, or undetermined origin." The purpose would be to examine

the possibility of integrating biological and psychologically oriented treatment methods.

GENERALIZED ERECTILE DYSFUNCTION: ORGANIC, MIXED, OR UNDETERMINED ORIGIN

The key differentiating feature of the acquired and generalized form of erectile dysfunction is that the difficulty experienced by the man exists in all major circumstances when he would be expected to have an erection: with a partner, masturbation, and with sleep (including the time when he awakens in the morning).

The assessment of generalized erectile disorders is summarized in Figure 11-4.

Description

The key differentiating feature of the acquired and generalized form of erectile dysfunction is that the difficulty experienced by the man exists in all major circumstances when he would be expected to have an erection: with a partner, masturbation, and with sleep (including the time when he awakens in the morning). In addition, he describes little or no difficulty with erectile function in the past. Typically in his 50s or older, his erection problems began in recent years. Sexual desire is usually present but, depending on the status of his health and the nature of any previous health troubles, there may have been problems with ejaculation or orgasm. Relationship conflict was not apparent except as a possible result of reluctance to seek help despite his partner's encouragement. Although unhappy, he is not clinically depressed.

A couple in their mid-60s were seen because of the man's erectile difficulties. They were married for seven years, both for the second time. Five years before, he held an executive position in a major computer software company but as a result of "downsizing" lost his job and subsequently retired. His wife had always been in good health but he had a "mild" heart attack about three years before. He felt well since then, stopped smoking, and was not taking any medications. On his last medical visit several months earlier, he talked with his physician about erection troubles, which had begun about one year before. Further history-taking revealed that the fullness of his erections during sexual activity with his wife (as well as in the morning and with masturbation—which occurred once every few months) had become consistently about 50% of what he had previously experienced. The last time he could recall having a full erection at any time was about one year earlier. He was referred to a urologist and after a thorough investigation he was told that the reason for his trouble was "organic." Intracavernosal injections were suggested. He was reluctant to pursue this option and wanted a second opinion from a sex therapist. This consultation primarily confirmed the opinion of the urologist and as a result he began injection treatment. He changed to sildenafil (Viagra) when this became available and after three months of use, he and his wife reported that they were pleased with the results.

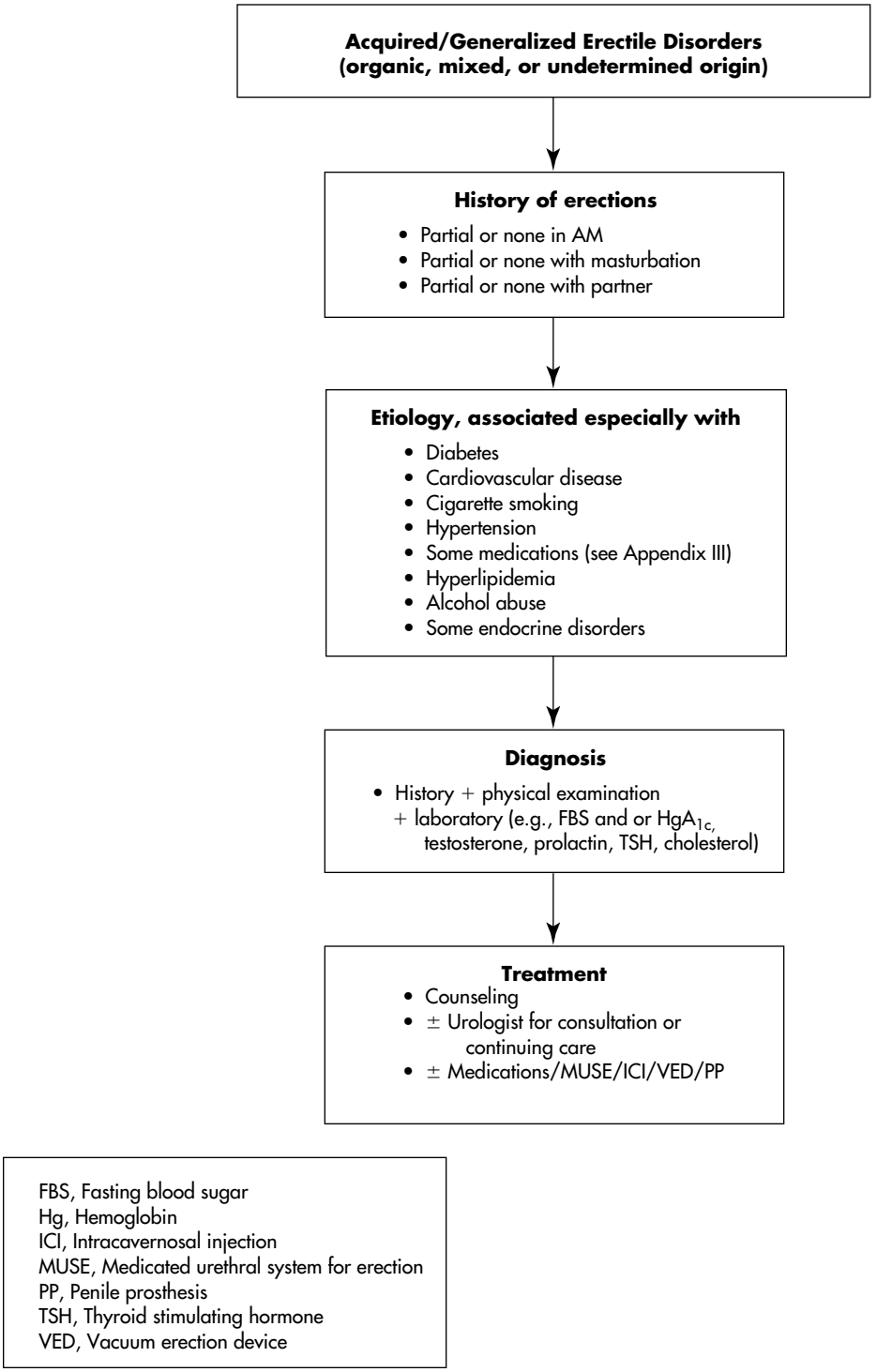

Figure 11-4 Assessment of acquired/generalized erectile dysfunction.

Box 11-2

Causes of Generalized Erectile Dysfunction

Organic Causes of Impotence*

Inflammatory
- Urethritis
- Prostatitis
- Seminal vesiculitis
- Cystitis
- Urethral stricture

Mechanical
- Congenital deformities
- Peyronie's disease
- Morbid obesity
- Hydrocele
- Phimosis
- Malignancy

Postsurgical
- Perineal prostatic biopsy
- Prostatectomy (simple, radical)
- Abdominal perineal resection
- Aortoiliac surgery
- External sphincterotomy

Occlusive Vascular
- Atherosclerosis
- Arteritis
- Priapism
- Thrombosis
- Embolism

Traumatic
- Penectomy
- Ruptured urethra

Endurance Factors
- Postmyocardial infarction
- Pulmonary insufficiency
- Anemias
- Systemic illnesses (infection, nutritional)
- Metabolic (renal and hepatic failure)

Sleep disorders

Neurogenic
- Parkinson's disease
- Temporal lobe lesions
- Head injuries
- Cord tumors/resection/injuries
- Multiple sclerosis
- Tabes dorsalis
- Neuropathies
- Spina bifida
- Subacute combined degeneration
- Amyotrophic lateral sclerosis

Chemicals
- Hypnotics
- Sedatives
- Phenothiazines
- Antidepressants
- Antihypertensives
- Antiparkinson agents

Endocrine
- Diabetes
- Pituitary disorders
- Thyroid disorders
- Adrenal disorders
- Klinefelter's syndrome
- Gonadal dysfunction

From Lakin MM: Diagnostic assessment of disorders of male sexual function. In Montague DK (ed): *Disorders of male sexual function,* Chicago/London/Boca Raton, 1988, Year Book Medical Publishers, Inc. Reprinted with permission.
*Modified from Smith AD: *Urologic Clinics of North America.* Philadelphia, WB Saunders, 1981, vol 8, no. 1, p. 83.

Etiology/Risk Factors

Many medical disorders are identified as "organic causes" of erection difficulties (see Box 11-2), however, only a few seem to account for a great many cases where the etiology is known. Major etiological factors are discussed below.

In the Massachusetts Male Aging Study, health status was ascertained by asking if diabetes, heart disease, and hypertension were present.[6] These three "were significantly associated with changes in the impotence probability pattern." After adjusting for age, 28% of men with treated diabetes, 39% of men with treated heart disease, and 15% of those with treated hypertension were described as having "complete impotence."

Forty men with diabetes with no other illnesses and taking no drugs other than antidiabetic medication were compared to the equivalent group of age-matched healthy controls.[26] The men with diabetes were found to have a variety of sex-related difficulties, including erectile function with attempts at intercourse, during sleep, and with masturbation; sexual desire; frequency of intercourse; premature ejaculation; and sexual satisfaction.

Diabetes

Estimates of the prevalence of erection problems in men with diabetes range from 27% to 71%.[26] As many as 50% of people with type 2 diabetes remain undiagnosed (about 8 million people in the United States), a serious situation since hyperglycemia in this condition causes microvascular disease and may contribute or cause macrovascular disease.[27] Erection problems may well be a manifestation of micro- and/or macrovascular disease. In the MMAS sample, "the age-adjusted probability of complete impotence was three times greater in subjects reporting treated diabetes than in those without diabetes."[6] In an attempt to clarify the connection between diabetes and erection problems and to eliminate the confounding effects of associated illness and medications, 40 men with diabetes (but free of other illness or drugs apart from antidiabetic medication) were compared to an equivalent group of age-matched healthy controls.[26] The men with diabetes were found to have a wide variety of sex-related difficulties, including:

1. Erectile dsyfunction with attempts at intercourse, during sleep, and with masturbation
2. Sexual desire disorders
3. Diminished frequency of intercourse
4. Premature ejaculation
5. Diminished sexual satisfaction

In another study, *sexually functional* men with diabetes were shown to have significantly diminished Nocturnal Penile Tumescence (NPT) profiles when compared to a similar control group.[28]

Cardiovascular Disorders

The association between erectile difficulties and cardiovascular disorders is well studied. "Vascular disorders" include two groups: arterial (i.e., obstruction to the penile inflow tract) and veno-occlusive (i.e., the inability to trap blood in the corpus cavernosum).[29] The former has attracted particular attention.

The presence of four *arterial* risk factors (ARF) (diabetes, smoking, hyperlipidemia, and hypertension) was assessed in 440 "impotent" men.[30] The frequency of "organic

impotence" occurred in 49% of men without any ARF, and 100% when there were three or four risk factors.

In the MMAS, "complete impotence" was higher in current smokers (versus current nonsmokers) who were also treated for heart disease, hypertension, or arthritis, and, as well, in those taking cardiac drugs, antihypertensives, or vasodilators.[6]

Cigarette Smoking

The link between cigarette smoking and arterial disease is well established. Smoking was found to be significantly more prevalent in men who were "impotent" compared to estimates of smoking among men in the general population.[31] In one study, two groups of men with and without penile arterial disease were compared, and the former was found to have smoked more pack-years.[32] In the MMAS, "complete impotence" was higher in current smokers (versus current nonsmokers) who were also treated for heart disease, hypertension, or arthritis, and, as well, in those taking cardiac drugs, antihypertensives, or vasodilators.[6] In subjects with treated heart disease the probability of "complete impotence" in the MMAS was 56% for current smokers compared to 21% for current nonsmokers. Likewise, treated hypertension together with smoking increased the probability of "complete impotence" to 20% of those who had both factors, as compared to 9% among hypertensive men who did not smoke. Apart from particular connections between smoking and other risk factors, "a general effect of current cigarette smoking was not noted."

Impotence was found in 7% of men with normal blood pressure. Impotence was found in 17% to 23% of untreated men with hypertension and in 25% to 41% of men treated for hypertension.[33]

Hypertension

The relationship between "impotence" and "erection dysfunction" and hypertension was examined in a review of studies that were conducted in the 1970s and 1980s[33](p. 204). Impotence was found in 7% of men with normal blood pressure. Impotence was found in 17% to 23% of untreated men with hypertension and in 25% to 41% of men who were treated for hypertension. From these studies, the association between "impotence" and hypertension (apart from drugs used in its treatment) is clearly evident.

Lip ids

In the MMAS, a negative correlation was found between "impotence" and high density lipoprotein cholesterol, although this was not so with total serum cholesterol. "In the older men (age 56 to 70 years), the age-adjusted probability of 'moderate impotence' increased from 6.7% to 25% as high density lipoprotein cholesterol decreased from 90 to 30 mg./dl."[6]

The increase in the number of new drugs that are currently being introduced for various human ailments and the increased speed with which they appear on the market make it likely that unanticipated side effects (including sexual side effects) of these agents will become apparent only after they have been used for a period of time. Physicians must therefore be constantly sensitive to the possibility of sexual side effects (including effects on the mechanism of erection) of newer drugs.

Medications

A variety of drugs used presently and in the recent past have been shown to interfere with erectile function in men (see Appendix III). The increase in the number of new drugs that are currently being introduced for various human ailments and the increased speed with which they appear on the market make it likely that unanticipated side effects (including sexual side effects) of these agents will become apparent only *after* they have been used for a period of time. Physicians must therefore be constantly sensitive to the possibility of sexual side effects (including effects

on the mechanism of erection) of newer drugs. The following comment was made about hypotensive agents but applies equally to other drugs as well: "Although . . . certain agents . . . . are likely to have sexual side effects [while] other agents . . . are unlikely to be associated with erectile dysfunction, it is important to stress that *any* agent may cause erectile difficulties in certain patients . . . there is [also] considerable individual variation in vulnerability of erectile function to different drugs. In other words, *the existing literature can only serve as a general guide to patient management*" (italics added)[34] (pp. 111-112).

In the MMAS study, "complete impotence" was found to be present significantly more often in men taking the following medications than in the sample as a whole (10%)[6]:

- Hypoglycemic agents (26%)
- Antihypertensives (14%)
- Vasodilators (36%)
- Cardiac drugs (28%)

This association was not found with lipid-lowering drugs.

Two groups of drugs (antihypertensives and medications used in psychiatry) have been particularly implicated in the development of erectile dysfunction[33] (pp. 197-337).

Issues learned from sexual side effect research on antihypertensive drugs (that apply to other substances as well) include the following items[32] (pp. 206-207):

- Possibility of late appearance (6 months or more)
- Lack of information about their effects on women
- Importance of assessing sexual symptoms of the underlying disease
- Need to include information from partners
- Usefulness of information about masturbation
- Need to assess alcohol use and abuse

Issues learned from sexual side effect research on antihypertensive drugs include the possibility of their late appearance (6 months or more), the lack of information about their effects on women, the importance of assessing sexual symptoms of the underlying disease, the need to include information from partners, the usefulness of information about masturbation, and the need to assess alcohol use and abuse[32] (pp. 206-207).

Alcohol

"With just a few drinks, most men experience transient boosts in sex drive and sociability. With continued drinking, however, erection and ejaculation abilities systematically decrease in a dose-related fashion to a point of total dysfunction."[32] Although there are acute and chronic effects of alcohol on sexual function, comments here focus on the latter. Studies relating to chronic alcoholism and epidemiology, Nocturnal Penile Tumescence (NPT), hormonal and neurologic effects have been reviewed.[35]

An examination of clinical experience in the care of approximately 17,000 male alcoholics over 36 years revealed that 8% spontaneously described continued erectile problems after detoxification.[36] Years later, despite abstinence, 50% had not fully recovered their erectile function. The continuation of sexual desire in these men indicates that their erectile problems were unlikely to be simply a result of insufficient testosterone but rather more fundamental structural and functional body changes, including:

An examination of clinical experience in the care of approximately 17,000 male alcoholics over 36 years revealed that 8% spontaneously described continued erectile problems *after* detoxification.[36] Years later, and despite abstinence, 50% had not fully recovered their erectile function.

- Effect on testosterone receptors

- Influence of estrogen
- Damage to organs in the body, including the central nervous system, testicles, and liver
- Existing diseases caused or made worse by alcohol abuse such as diabetes, heart disease, and peripheral neuropathy

In addition, two controlled studies on the effects of alcoholism on sexual function demonstrate that erectile dysfunction[37] and also desire problems[38] were more common in alcoholic men.

In an effort to assess the effect of alcohol use on NPT, 26 sober, healthy, sexually functional, and medication-free chronic alcoholics and controls were studied for two nights in a laboratory setting.[39] The subjects had fewer full penile tumescent episodes that were also shorter in duration. The authors speculated on the possible contribution of central processes to the effects of alcohol on erections.

The impact of chronic alcoholism on pituitary-gonadal function appears at both levels. Apart from liver disease, chronic alcoholic men show evidence of hypogonadism, abnormalities in spermatogenesis, and testicular atrophy.[40] Such men also demonstrate diminished androgens, elevated estrogens, and increased prolactin levels.

The neuropsychiatric effects of chronic alcoholism on sexual function may involve central and peripheral processes. Alcohol-induced peripheral neuropathy may result in both erectile and ejaculatory disorders. Schiavi outlined many of the psychological factors that may also be present such as preexisting personality problems, mood disorder, and feelings of inadequacy.[35] He concluded that "the reciprocal interaction between drug intake and psychological factors is so closely interwoven that it is impossible to identify the nature of this relation."

Erectile dysfunction in all situations, including the lack of nocturnal erections, strongly suggests that a general medical condition or substance use is the cause.[5]

Endocrine Abnormalities

"Erectile dysfunction of exclusively endocrine origin is uncommon. . . In most cases the primary effect of the endocrine abnormality is loss of sexual interest."[12] The authors of the MMAS study arrived at a similar conclusion.[6]" Of the 17 hormones measured in the MMAS, "only dehydroepiandrosterone showed a strong correlation with impotence." Specifically, no correlations were found between "impotence" and the following hormones:

- Testosterone (total or free)
- Sex hormone binding globulin (SHBG)
- Estrogens
- Prolactin
- Luteinizing hormone (LH)
- Follicle stimulating hormone (FSH)

When hormonal difficulties occur in the context of erectile dysfunction, the more common clinical abnormalities are those that involve the hypothalamus-pituitary-gonadal axis and include: hypo- and hypergonadotropic hypogonadism, hyperprolactinemia, and hypo- and hyperthyroidism.[41] (p. 85). The frequency of the association of endocrine and erectile problems depends on the age of the sample and the

clinical context of the investigation (e.g., outpatient medical, urology, endocrine, or sex therapy clinics), as well as the nature of the sample.

When the particular association of erection problems and hyperprolactinemia (HPRL) was reviewed, the observation was made that this was "often the first, and for a long time the only symptom of HPRL, an important point because in many cases the cause is a pituitary tumor."[12]

Buvat and Lemaire reviewed large published series of endocrine abnormalities in cases of erectile dysfunction and when combining their own results with others found an 8% prevalence for low testosterone and 0.7% of prolactin levels greater than 35 ng./ml.[42]

Aging

See "Sexual-developmenl History: The Older Years" in Chapter 5.

Laboratory Investigation

When the history and physical examination (see the "General Considerations" section above in this chapter) leave doubt about the origin of a patient's erectile dysfunction, the clinician must also consider the use of laboratory tests.

The availability of orally administered treatments for erectile dysfunction (for example, sildenafil—see "Treatment" below in this chapter) that appear to be easy to use, often effective, and have minimal side-effects may well have profound effects on the process of evaluating erectile dysfunction. The initial use of these medications by a patient may, itself, become a test, and in so doing may replace some other investigatory procedures. Nothing, however, should replace a *careful clinical assessment* and "when there is suspicion of an organic factor, one should rely (as well) on a combination of investigations."[13]

Laboratory investigation of generalized erectile dysfunction chiefly entails examining three body systems:

- Endocrine
- Vascular
- Neurological

Several diagnostic tests involving these three systems have been developed. The criteria used in considering which diagnostic tests are appropriate for investigating generalized erectile dysfunction on a primary care basis are:

1. Usefulness
2. Low cost
3. Noninvasiveness
4. Low complexity
5. Availability

Endocrine Blood Tests

Endocrine and blood tests for diabetes are probably the *only* procedures that fulfill the criteria outlined immediately above. Despite agreement on the endocrine disorders most commonly associated with erection difficulties,". . . . there is disagreement on the spe-

These authors also direct clinicians to repeat first results of abnormal prolactin and testosterone determinations because of their finding of normal second results in 40% of their cases.

cific tests to be employed or the interpretation thereof"[41] (p. 85). Many suggest the need to measure Testosterone (T) and Prolactin (PRL) in all men with erection difficulty (PRL can be abnormal when T is not). However, others promote a more specific policy. For example, Buvat and Lemaire suggest that *before* age 50, T should be determined only in cases of (accompanying) low sexual desire and abnormal physical examination but that *after* age 50 it should be measured in all men, and that PRL should be determined only in cases of (accompanying) low sexual desire, gynecomastia, and/or testosterone less than 4 ng/ml.[42] These authors also direct clinicians to repeat first results of abnormal prolactin and testosterone determinations because of their finding of normal second results in 40% of their cases.

Apart from measuring total testosterone, this hormone also can be determined in the bioavailable form (BAT) and as free testosterone (FT). BAT consists of FT and the fraction that is bound to albumin[41] (p. 76).

Conflicting opinions exist over the need to also immediately test some or all of the following other factors without waiting for an abnormal T or PRL result:

- Follicle Stimulating Hormone (FSH)
- Luteinizing Hormone (LH)
- Sex Hormone Binding Globulin (SHBG)
- Thyroid function tests

Cost of testing and the nature of the clinical context are two elements resulting in differences of opinion. In the literature, it seems assumed that these other factors would be measured in the event of an abnormal T or PRL level.

Tests for Diabetes

Fasting blood sugar (FBS) or fasting plasma glucose (FPG) and/or glycosylated hemoglobin (HgA_{1C}) is widely used as a screening test for diabetes. A positive test indicates that a confirming diagnostic test is warranted.[43]

Vascular Tests

Since the penis is, basically, a vascular organ, vascular tests are often important elements in the evaluation of erectile difficulties, particularly when there is suspicion that vascular elements may contribute to the etiology. However, tests of penile vascular function are mostly invasive, costly, complex, difficult to interpret, and have limited availability. As such they are generally conducted by urologists and *not* recommended in primary care unless a physician has special training. Although vascular testing procedures may not be recommended for use in primary care, clinicians should be aware of their potential diagnostic benefits and limitations to determine the need for urological consultation.

The most worrisome complication of ICI is that of prolonged erection. After six hours of continuous erection, there is insufficient blood supply to the erectile tissue. The corpora cavernosa must be drained to decrease the intracavernosal pressure and an adrenergic agonist administered, injected intracavernously.[29] In clinical practice, patients must be told to contact a physician long before six hours if their erection persists.

Assessment of the penile response to the intracavernous injection (ICI) of vasoactive agents has been found to be particularly useful in considering vascular function. While structural problems with cavernosal arteries may explain a negative response, anxiety can as well.[44] "A positive erectile response implies normal veno-occlusive function.

Nonresponders bear a high probability of a vascular origin with a predominance of veno-occlusive insufficiency."[29]

From a procedural viewpoint, the most common substances used for intracavernosal injections are papaverine, papaverine-phentolamine mixture, or prostaglandin E1 (PGE_1). The most worrisome complication of ICI is that of prolonged erection. A comparative study of these three medications demonstrates that PGE_1 had the highest erection rate (75%) and lowest prolonged erection rate (i.e., requiring "interruption" [0.1%]).[45] After six hours of continuous erection, there is insufficient blood supply to the erectile tissue. In this situation, the corpora cavernosa need to be drained to decrease the intracavernosal pressure and an adrenergic agonist (e.g., 10 mg of adrenaline) injected intracavernosly.[29] *In clinical practice, patients must be told to contact their physician long before six hours if their erection persists.*

Pharmacopenile Duplex Ultrasonography (PPDU) provides "an estimate of penile arterial inflow and venous outflow . . . [and] . . . has become a first-line test to define vascular [erectile dysfunction]"[29] It allows for accurate location of penile arteries and measurement of the diameter of each artery and provides evidence of the thickness and pulsatility of arterial walls.[12] In addition to assessment of the state of the cavernosal arteries, PPDU can locate well-defined pathological conditions such as Peyronie's disease. The procedure for PPDU involves the creation of an erection through ICI (needed because the procedure is unreliable when a penis is in the flaccid state) and then simultaneously combining ultrasound color imaging of the arteries to the cavernosal bodies of the penis with an analysis of blood flow patterns.

Nonspecific Tests: Nocturnal Penile Tumescence (NPT)

Any discussion of erectile dysfunction assessment is incomplete without including NPT testing, since it is so widely used and so frequently included in the literature on this subject. NPT is based on the discovery that a period of sleep involves different stages and that one of those stages (REM) is associated with many body changes, including the development of erection in men (three or four times each night and occupying about 20% of total sleep time). It was assumed that erections that occur at night and those which occur during the day involve the same body mechanisms and that by comparing sleep and daytime erections, it would be possible to distinguish the psychological or organic nature of the etiology of erection dysfunction. Sleep erections are considered to be unaffected by waking psychosocial factors.

When used in-home, NPT testing fulfills many of the primary care criteria previously described insofar as it is inexpensive, not extraordinarily complex to use, noninvasive, and available. However, when done in a sleep laboratory, NPT is the opposite in that it is expensive, cumbersome, and frequently unavailable in many geographic areas. The chief doubt about NPT is its usefulness.

When used in such a way as to provide clearest interpretation, the test is performed in a sleep laboratory with measurement of other sleep parameters such as electroencephalograph (EEG), respiration, and electromyograph (EMG), and recordings are made on at least two nights. The purpose in monitoring other sleep parameters is the detection of interference with sleep or REM such as might happen with illness or medication, which might result in mistaken conclusions.

Because of the complexity, expense, and difficulties with availability associated with formal testing of NPT in a hospital setting, three in-home procedures have been developed[46] (p. 153):

1. The "stamp" test (a ring of stamps placed around the base of a man's penis)
2. Snap Gauge Band (one ring of a thin plastic material containing three others that break at three different levels of tension as a penis enlarges)
3. Portable NPT monitoring

"Stamp" test results are difficult to interpret because of such problems as false-positive findings due to accidental tearing of the stamps for reasons other than an erection, false-negative results due to slippage, and lack of standardization. Snap Gauge has similarly been found to be of limited value. Both the stamp test and Snap Guage provide information about changes in circumference only, nothing about rigidity or stiffness (a vital issue in the assessment of erectile capacity for intercourse), and no data concerning the number or duration of tumescent episodes each night.

Portable monitors (e.g., Rigiscan) measure rigidity as well as the number and duration of NPT episodes. Measurement of rigidity has for some time been regularly included in NPT testing (now sometimes called NPTR with the "R" referring to rigidity). Reasons include the finding of substantial "interindividual differences in the increase of circumference associated with full erections and the recognition that maximal increases in shaft circumference did not indicate adequate rigidity."[47]

As inviting as these in-home methods of NPT testing might be, clinicians should be fully aware of their considerable limitations. It is suggested that this test "should be used only as screening tools in the context of a comprehensive medical and psychological evaluation"[46](p. 153).

Although the use of the Rigiscan at-home is certainly less troublesome and expensive than NPT in a sleep laboratory, opinions differ concerning its usefulness. "Apart from the lack of sleep data, it is impossible to know if such devices have been misused, manipulated, or otherwise mishandled by the patient. Deliberate faking of results cannot be excluded. . . ."[12]

Apart from issues of technology, patient reliability, and cost, the utility of NPT itself has been questioned. After many years, the importace of NPT in the process of evaluation of erectile dysfunction remains unclear. So, too, is the question about the equivalency of sleep and sexual erections. Confusion abounds in findings that show, for example, *abnormal* NPT results in *men who are not sexually dysfunctional* in circumstances such as aging,[47] depression,[48] and diabetes.[49] Conversely, *normal* NPT is reported in men with multiple sclerosis *who have daytime erectile difficulties*.[50]

Conclusions about the use of NPTR when used in the clinical evaluation of erectile dysfunction (in a sleep laboratory or at home) are summarized Box 11-3.

Treatment

"Whether psychological issues are co-determinates of the erectile problem or are reactive to it is immaterial. A man's emotional reaction to his erectile failure may be such that it serves to maintain the erectile problem even when the initial physiological causes are resolved . . . no patient, even those with a clear organic impairment of

Box 11-3

Nocturnal Penile Tumescence (NPT) Interpretation[45-47]

- Greatest benefit is to confirm a situational (psychogenic) erectile disorder
- Evidence of significant erectile activity during a single night may be sufficient to demonstrate the potential for normal functioning
- Repeated demonstration of insufficient rigidity in an otherwise normal male is not necessarily pathological
- Abnormal findings may coexist with normal daytime function in older men, men with diabetes, and nondepresssed men with a recent history of major depression
- May not be helpful in neurological disease in proving waking erectile capacity
- Should be conducted only in a sleep (versus home) laboratory when certain factors coexist (e.g., manual dexterity problems, dementia, malingering, medico-legal assessment, and sleep disorder)

erectile capacity, can be considered inappropriate for psychological as well as relevant surgical or drug therapies for his sexual problem".[54] For these reasons, everything that was written immediately above about the treatment of situational erectile difficulties is also applicable to the acquired and generalized form.

Specific Treatments

Specific disorders and their sexual symptoms are often (not always) therapeutically responsive to specific treatments. Therapy for hyper- and hypogonadotropic hypogonadism and hyperprolactinemia have been reviewed elsewhere[41] (pp. 86-91). Details concerning treatment will not be discussed here, since primary care health professionals likely seek consultation when encountering such a patient.

Other specific disorders and their sexual symptoms may not respond therapeutically to specific treatment methods. Diabetes is an example. Careful control of insulin-dependent and probably non-insulin-dependent diabetes mellitus will slow the onset and delay the progression of early vascular and neurologic complications, two body systems that have been particularly implicated in the etiology of erectile disorders in this disease.[55,56] Presumably, better control of diabetes would also delay the onset and slow the development of sexual dysfunctions, including erectile difficulties. When erectile dysfunction in specific disorders such as diabetes do not respond to specific treatment, nonspecific approaches can be used.

Nonspecific Treatments

Dramatic developments in the treatment of the acquired and generalized form of erectile dysfunction have been introduced in the past two decades in the form of intracavernosal injections, intraurethral medication, erections devices, and prostheses. As significant as these developments have been, they might well be overshadowed by the introduction of new oral therapies. The etiological and clinical heterogeneity of the acquired and generalized form of erection dysfunction will likely

As a result of factors such as the ease of administration, sildenafil (and other orally administered substances currently being tested) will likely result in a substantial shift in the treatment of men with erectile difficulties away from specialists and toward physicians in primary care.

Box 11-4 Sildenafil (Viagra) Highlights[62, product monograph]

- MECHANISM OF ACTION: Sexual stimulation results in release of nitric oxide (NO); NO stimulates the production of cyclic guanosine monophosphate (cGMP), which relaxes smooth muscle and promotes the inflow of blood into the corpus cavernosum; phosphodiesterase type 5 (PDE5) is an enzyme that inhibits cGMP; sildenafil inhibits PDE5 (and therefore causes increased levels of cGMP)
- PLASMA CONCENTRATION LEVELS: Maximal within one hour after oral administration
- HALF-LIFE of 3 to 5 hours
- ADMINISTRATION: Taken on an "as needed" basis about one hour (0.5 to 4 hours) before sexual activity
- FREQUENCY OF USE: Maximum recommendation is once/day
- STARTING DOSE: Recommendation is 50 mg but can vary from 25 to 100 mg, depending on efficacy
- EFFICACY: Eighty percent of men with erectile dysfunction (ED) taking 50 mg will have sufficiently firm erections for intercourse
- ASSOCIATED SEXUAL STIMULATION: Necessary
- ETIOLOGY OF ED: Similar benefit regardless of etiology
- OTHER SEXUAL BENEFITS: Orgasmic function, intercourse satisfaction, and overall satisfaction but not for sexual desire
- PARTNERS: Verify erectile improvement and report significant enhancement of their satisfaction
- SIDE EFFECTS: Increase with increasing dose and include (at 50 mg): headache (21%), flushing (27%), dyspepsia (11%), rhinitis (3%), and visual disturbance (6%; change in the perception of color hue or brightness). No priapism reported
- CONTRAINDICATION: Potentiates hypotensive effects of organic nitrates. Therefore, not to be taken with organic nitrates in any form, including nitroglycerin

necessitate the continued use of several treatment approaches. Not everyone will benefit from the new oral therapies. For this reason, the oral therapies and many of the other currently used treatment approaches are discussed below.

Oral Therapies

Sildenafil (*Viagra*), is a new oral treatment that may be a critical advancement in the care of men with erectile difficulties (Box 11-4).

As a result of factors such as the ease of administration, sildenafil (and other orally administered substances currently being tested) will likely result in a substantial shift in the treatment of men with erectile difficulties away from specialists and toward physicians in primary care. Sildenafil demonstrated in initial trials to be well tolerated and "effective in improving erectile activity in patients with male erectile dysfunction for which there is no established organic cause."[57]

As described above (see "Mechanism of Erection" in this chapter), relaxation of smooth muscle is an essential aspect of the development of an erection. This relaxation is mediated by nitric oxide via cyclic guanosine monophosphate (cGMP).[59] Cyclic nucleotide phosphodiesterase (PDE) isozymes hydrolyse cGMP. It was reasoned that an inhibitor of PDE would therefore enhance the action of nitric oxide/cGMP on penile erectile activity. Sildenafil is such an inhibiting agent.[59] It is described as the first representative of a new class of agents: an enzyme inhibitor (type 5 cyclic guanosine monophosphate-specific phosphodiesterase isozyme) that results in the relaxation of corpus cavernosum smooth muscle cells and thereby enhances penile erection in response to sexual stimuli.[60]

An initial report on sildenafil involved 12 subjects and was conducted in two phases: (1) a single dose in a laboratory and (2) once-daily doses at home for seven days, 1 to 2 hours before sexual activity was likely to occur.[57] Both phases were placebo-controlled, double-blind, and involved a cross-over design. The first included use of the drug at three different doses and measured erectile response to visual sexual stimulation using subject-chosen explicit videos and magazines. In the second phase, subjects kept a diary and graded their erections. Results from the first phase demonstrated a significant difference in penile rigidity between all three doses of sildenafil and placebo, with the difference being more substantial as the dose increased. The in-home phase showed that higher quality erections occurred more often when men were on the drug. Adverse events were described as "mild and transient."

Another report on sildenafil was conducted on 250 patients with erectile dysfunction of "predominantly no known organic cause."[61] Patients previously were involved in an open dose study and were randomized to receive their optimum dose of the medication or placebo. They were asked to compare their erections in the present study to those they experienced in the open trial. Of those given sildenafil, 59% reported no change, and of those receiving placebo, 72% reported their erections as "much worse." The authors concluded that sildenafil must be continued for the erectile improvement to be maintained.

The most complete report on oral sildenafil (as of Spring 1998) was published in the New England Journal of Medicine (NEJM). It involved a total of 861 men with erectile dysfunction described as organic, psychogenic or mixed.[62] Two studies were conducted: (1) a dose-response study on 532 men treated with 25, 50, 100 mg or placebo and (2) a dose-escalation study involving 329 different men treated initially with 50 mg or placebo and subsequently with one half or twice the amount of the original dosage, depending on efficacy and tolerance. These studies were performed in a natural environment and therefore relied on the subjects' reports of efficacy.

In a dose-response study, the frequency of erections sufficiently firm for intercourse to occur was 72%, 80%, and 85% for doses of 25 mg, 50 mg, and 100 mg, respectively (versus 50% for the placebo group; $p<0.001$). In the dose-escalation study, 69% of attempts at intercourse (versus 22% for the placebo group) were successful ($p<0.001$).

In the first study (dose-response) in the NEJM report, increasing doses of sildenafil were associated with significantly increased "frequency of penetration" and maintenance of erections after entry ($p<0.001$). Interestingly, the cause of the erection difficulty did not affect the outcome. In the second study (dose-escalation), improvement of the same two measures were significantly better for sildenafil compared to placebo ($p<0.001$), as were several other measures, including "overall satisfaction." (However, in this same study, sexual desire scores were not different in the two groups). In the dose-response

study, the frequency of erections sufficiently firm for intercourse to occur was 72%, 80%, and 85% for doses of 25 mg, 50 mg, and 100 mg, respectively (versus 50% for the placebo group; ($p<0.001$). In the dose-escalation study, 69% of attempts at intercourse (versus 22% for the placebo group) were successful ($p<0.001$).

Yohimbine is one of the more widely used and studied oral agents used recently in the treatment of erectile dysfunction. It is an alkaloid derivative that is found in the bark of the yohimbine tree and has a long-standing reputation as an aphrodisiac.[63] Part of the attractiveness of yohimbine is the "benign side effect profile"[33] (p. 123). Pharmacologically, yohimbine is a preferential presynaptic alpha 2 antagonist. The dose ranges from 2 to 6 mg three times per day, and has shown to have a positive effect on sexual behavior in animals).[64] However, a review of the outcome of several studies of men with erectile dysfunction indicates that while yohimbine may have the capacity to affect sexual desire and performance in some subjects, "results have been far from conclusive [since] more than half of all patients studied thus far have shown little or no benefit from the drug."[63] A meta-analysis of studies of the effect of yohimbine in men with erection difficulties concluded the opposite, namely, that it was consistently helpful compared to placebo.[65] It may be that the heterogeneity of men with erection difficulties that is evident in many studies has disguised a beneficial effect of this substance in a particular subpopulation. One hypothesis suggests that men with a "nonorganic etiology" might derive a greater benefit than other men.[64]

Androgens often have been administered to men with erection difficulties, frequently without establishing the presence of an endocrinopathy. Studies of androgen treatment in erectile dysfunction that included hormonal assessments strongly suggest that it "is of little value in eugonadal males"[40] (p. 91). The hazards of androgen therapy in this situation have not always been considered. Given the fact that erectile dysfunction and prostate cancer become more evident with increasing age, clinicians need to be especially concerned about the potentially negative effect of androgens (even on a trial basis) on the prostate gland (see Etiology, "Endocrine Abnormalities" above in this chapter).

Intracavernosal Injections (ICI)

Injection of medications directly into one of the corpora cavernosa of a man's penis as a treatment of erectile dysfunction became an accepted and widely used treatment method in the 1980s. It would not be surprising to see this approach greatly diminish in popularity with the advent of an efficacious and safe oral medication.

Three substances are currently used for ICI:

- Papaverine hydrochloride
- Phentolamine mesylate
- Prostaglandin E_1 (PGE_1)

Papaverine has been used alone or in combination with phentolamine; PGE_1 has been used alone or together with papaverine and phentolamine as Trimix. One formulation of PGE_1 is alprostadil. The subject of ICI treatment is thoroughly reviewed elsewhere.[66,67] Dosages are generally titrated to the response of the patient. PGE_1 alone is the substance most commonly used by urologists and the dose is typically in the range

of 1 to 40 μg.[67] Smaller doses of medications used in ICI are generally required in instances of neurogenic (and "psychogenic") erectile dysfunction.

ICI seems most efficacious in the context of a neurological deficit (e.g., spinal cord injury) and least helpful in men who have severe corporal veno-occlusive dysfunction and/or arterial insufficiency. Contraindications include poor manual dexterity, morbid obesity, and anticoagulant therapy.[67] Injection usually results in a partial erection within minutes and the addition of sexual stimulation usually increases the enlargement.[68] Patient's are taught the technique of injection (usually by a urologist or nurse-educator) and "observed while self-injecting so that the physician has an opportunity to advise and correct his technique." Patients then inject themselves at home.[67] One side effect, namely, prolonged erection (defined as more than four hours), requires immediate medical attention. The frequency of prolonged erection (priapism) and other side effects depends on the medication used. Side effects include the following:

- Fibrotic nodules (more with papaverine and/or phentolamine)
- Pain (about 10% to 34% with PGE_1 alone)
- Infection
- Bruising
- Liver function abnormalities
- Vasovagal episodes

Prolonged erections appear to more common with papaverine alone (10% of patients) than PGE_1 alone (2% of patients). The incidence of priapism seems to be less with the mixture of papaverine-phentolamine-PGE_1.[67] Priapism rates for home injections are considerably less (0.3%) than in-office trial injections. When priapism occurs, emergency intervention is required, and most cases resulting from papaverine and/or phentolamine respond to aspiration alone, or in combination with intracorporal installation of a diluted alpha-adrenergic receptor agonist such as epinephrine (limited to less than 15 μg at intervals of more than five minutes to avoid systemic side effects).[69]

Fibrotic plaques are reported to be less common with PGE_1 than with papaverine and/or phentolamine, and there have been no reports of liver disease with either of these substances or with PGE_1 despite abnormalities on liver function testing.[67] Pain during injection is commonly reported by men using PGE_1 (75% in one study) but pain is infrequent with papaverine and/or phentolamine.[67]

The impact of ICI on patients and their partners was studied and beneficial changes were described in each, particularly in the areas of self-esteem, sexual desire, frequency, and satisfaction.[66,67]

Although ICI is considered safe and reliable, many patients do not continue using it in the short-run for several reasons, including[67]:

1. The feeling that it was unnatural
2. Concerns about side effects
3. Lack of a regular partner
4. Fear of being belittled by the partner

In addition to immediate issues, there is a surprisingly high (50%) drop out rate at 12 month follow-up. Reasons given include loss of efficacy and loss of interest. The high

drop out rate suggests the need for a careful initial evaluation of the motivation of the patient and partner and willingness to accept ICI on the part of both.

Transurethral Alprostadil

Alprostadil is a synthetic compound identical to PGE_1. A transurethral method of delivering this medication was developed as an alternative to intracavernosal injections. With medicated urethral system for erection (MUSE), a proprietary drug delivery system, the medication is put into a tiny pellet and deposited into the end of the urethra with an applicator. A man urinates before insertion of the applicator to lubricate his urethra.

Route of administration of any medication may result in different side effects even though the substance might be the same. In the form of intracavernosal injections, alprostadil enters directly into the corpus cavernosum of the penis. When given transurethrally, the medication is absorbed from the urethral mucosa, enters the body's blood stream, and then is returned to the penis.

In a double-blind and placebo-controlled study, 1511 men aged 27 to 88 with "chronic erectile dysfunction from various organic causes" were treated with transurethral alprostadil.[70] To determine maximal penile response, subjects were given the opportunity to use up to four alternative doses of the drug: 125, 250,500, or 1000 μg. The 996 men who responded in a clinic setting were then randomly assigned to the selected dose or placebo. Eighty-eight percent of the men completed the three month course of treatment. Significantly more men in the alprostadil group (65%) reported having intercourse at least once and the medication was significantly more effective than placebo regardless of age or the cause of the erectile dysfunction.

The most common side effect of transurethral alprostadil was penile pain (reported by 33% of the men) but was considered mild and resulted in only 20% of the men leaving the study.[70] Other side effects include mild urethral trauma (5%), dizziness (2%), and urinary tract infections ("rare").

Transurethral alprostadil is particularly advantageous in primary care. For the physician the procedure is greatly simplified (compared to ICI) in that the medication is self-administered and does not require an in-office training procedure. From the patient's perspective, the process is less complex to learn and to use at home and free of the potentially serious side effects of priapism and fibrosis associated with ICI.

Vacuum Erection Devices

Vacuum erection devices (VEDs) are also called vacuum constriction, and external vacuum, devices (information available through Imagyn Technologies at 1-800-344-9688). Like intracavernosal injections, the use of VEDs may diminish considerably with the advent of a safe and effective oral treatment for erectile dysfunction. However, for the foreseeable future, VEDs are likely to remain in the armamentarium of health professionals who treat men with erectile dysfunction. The need is exemplified, for example, in men for whom a physical approach is recommended but who strongly prefer not to use any kind of drugs for an ailment that could be treated in a nonpharmacological manner. The subject of VEDs is reviewed in detail elsewhere.[66]

When first introduced in the early 1980s, "the concept [of VEDs] seemed difficult for physicians to accept. In an era of high technology, perhaps the low technology and simplicity of vacuum devices are disarming and provoke rejection"[66] (p. 297). Precisely

because they are "low tech," safe, and efficacious, VEDs are likely to remain of particular interest to primary care clinicians.

The mechanism of action is fundamentally the same for the various VEDs that exist. Procedures are as follows:

- A cylinder is placed over a man's flaccid penis and pressed firmly against his body to create an airtight seal
- Air is pumped out of the cylinder to create a vacuum
- Blood is, in the process, drawn into his penis
- After an erection exists, a tension band is transferred from the VED to the base of the man's penis
- A vacuum release valve is then opened and the cylinder is removed

VED-induced erections are passively created by suction and venous stasis that results from constriction, in contrast to erections produced naturally (and by ICI), which are actively created by neurotransmitters and relaxation of corpora smooth muscle. Several studies demonstrate that 90% of men who have "organic," "mixed," and "psychogenic" erection dysfunction and use this system are able to have sufficiently firm erections for the purpose of intercourse.[66]

The most common side effects reported with VEDs are hematoma and petechiae (8% to 50%).[66] These are generally not considered serious and resolve without medical intervention. Other side effects include the following:

- Pain
- Numbness of the penis
- Pulling of scrotal tissue into the cylinder
- Blocked and painful ejaculation

Patient acceptance is estimated at 80% to 95%. The reasons for discontinuing the use of a VED include the following:

- Mechanical difficulty
- Failure to produce an adequate erection
- Feeling that the device is cumbersome
- A sense that the erection is artificial

There are three contraindications: men with Peyronie's Disease, concurrent blood dyscrasia or use of anticoagulants, and poor manual dexterity (which can be overcome by the use of a battery operated device).

The irreversibility of prosthesis implantation limits its use given the rapid progress in the development of more benign approaches to the treatment of this disorder.

Table 11-1 summarizes comparisons between VEDs and ICI. Since they are both equally efficacious and have a positive effect on patients, "the critical discriminations need to be made on the basis of cost, potential side effects, patient acceptance, and aesthetic preferences of the man or couple"[66] (p. 304).

Penile Prostheses

The use of prostheses (or implants) in the treatment of men with erectile dysfunction is generally considered "a last resort," since surgery involves the destruction of structures which are otherwise normally involved in the erectile process. The irreversibility

Table 11-1 Comparison of Self-injection and External Vacuum Devices

FACTOR	SELF-INJECTION	EXTERNAL VACUUM DEVICES
Efficacy		
Neurogenic	Good response	Good response
Vasculogenic	Poor-good response	Good response
Idiopathic	Good response	Good response
Psychogenic	Adequate response	Adequate-good response
Psychological benefits	Positive effect	Positive effect
Patient acceptance	40%-50%	80%-95%
Cost	$75/monthly; $900/yearly	$200-$400 total outlay
Side effects	Prolonged erection	Hematoma, bruising
	Fibrotic nodules	Numbness
	Hepatotoxicity	Blocked/painful ejaculation
	Bruising	Pulling in scrotal tissue
	Pain	Fainting
	Vasovagal episodes	
	Infection	
Concealability	Easily concealed	Not easily concealed
Prolonged intercourse	Possible	Limited to 30 minutes
Frequency of Intercourse	Limited to twice weekly	No limitation
Conception	No limitation	Possible blocked ejaculation

From Althof SE, Turner LA: Self-injection therapy and external vacuum devices in the treatment of erectile dysfunction: methods and outcome. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment,* New York, 1992, The Guilford Press. Reprinted with permission.

of prosthesis implantation limits its use given the rapid progress in the development of more benign approaches to the treatment of this disorder. The subject of prostheses has been thoroughly reviewed elsewhere.[71]

Implants have been used since the early 1950s and now exist in a variety of forms: semirigid silicone only; semirigid, silicone interior; and inflatable.[71] "Most operating rooms stock one type of semirigid device and the inflatable prosthesis used most often by the implanting surgeons" (p. 270). Some factors that influence the choice of device include: cost, availability, esthetics, and manual dexterity (to use the inflatable type) (p. 271).

In a follow-up examination on the satisfaction of patients (n = 52) and their partners (n = 22), which involved interviewing the two people separately and had a response rate of 72%, the kind of device implanted made little difference to the men. However, the patient's partner preferred inflatable implants.[72] All except four had intercourse more than "infrequently." Almost 80% of the men said they would undergo the operation again but only 60% of the partners said that they had no hesitations.

The goal of treatment with penile prostheses can vary greatly and depends to a large extent on the perspective of the discipline of the person stating an opinion. Some

urologists focus specifically on the issue of erection, whereas mental health professionals and sex therapists concentrate more broadly on sexual satisfaction of the two partners. One follow-up study did not resolve the conflict (but leaned more toward the view of sex therapists), since it demonstrated that the greatest benefit is the sense of "restored manhood." "The feeling of being *capable* of coitus, was reported by many of the men in the study as a prime benefit of surgery" (italics added)[71] (p. 273). Screening issues have been identified to detect patients for whom penile prosthesis implantation is planned but who might benefit also from preventive counseling.[73] These include the following factors:

- Concern about the importance of penile size in sexual activity
- Disinterest in foreplay
- Low sexual desire in either partner
- Premature ejaculation
- Untreated vaginal atrophy in the woman

Indications for Referral for Consultation or Continuing Care by a Specialist

The treatment of solo men, or couples, in which the man has an acquired and generalized form of erectile dysfunction requires attention to both physical and psychological etiological issues. The more the etiologies are known, the more specific will be the treatment, as well as the kind of health professional needed to provide the necessary form of care. Referral for medical specialist *consultation* may be useful in, for example, the following specific and defined circumstances:

- Endocrine disorders
- Diabetes
- Cardiovascular disorders (including hypertension)
- Major depression

Referral to a urologist for continuing care might be beneficial in instances of "venous leak."

When complex, expensive, or physically invasive diagnostic procedures are necessary to clarify the etiology, consultation with a urologist who is knowledgeable about erectile disorders is required. Buvat reminded clinicians of " . . . Cochran's aphorism: 'before doing a test, decide what to do if it is (a) positive and (b) negative. If both answers are the same, don't do the test."[12]

When the etiology is unknown (or the etiology known but not responsive to specific therapy), treatment is nonspecific. The advent of safe and effective oral therapies will likely result in many more men with erectile dysfunction being identified and cared for on a primary care basis than at present. However, liberal use should be made of other health professionals (especially urologists and sex therapists) when cases are treatment-resistant (for the purpose of consultation and possible implementation of other nonspecific treatment approaches).

Summary

Impotence is a term that is widely used and accepted but falls short of being helpful for two reasons: (l) confusion, since several conditions are grouped in the same category

and (2) even more confusion, since the disorders have nothing to do with power (the origin of the word "potency").

The prevalence of erectile disorders is 40% of men at age 40 and 66% at age 70. The resources needed to treat this widespread problem are substantial and will become even more substantial as the aging population increases.

In the same way that different cardiac disorders manifest in similar ways despite having several origins, so do erectile disorders. While the chief manifestation of an erectile disorder is a soft penis rather than a hard one, the pattern of erection function matters when considering etiology ("psychogenic" and "organic") and treatment. An erectile problem that is generalized (exists in all situations: with a partner, in the morning, and with masturbation) suggests a different etiological and treatment direction than one that is situational (erections are unimpaired in some situations). Likewise, it matters if erection problems have always existed, since the man has been sexually involved with others (lifelong) or developed more recently (acquired).

Causes of erectile disorders (often more than one) include the following:

- Medical disorders (endocrine, cardiovascular, neurological)
- Drugs
- Elevated blood lipids
- Cigarette smoking
- Psychiatric disorders
- Relationship problems
- Anxiety

In any investigation of an erectile disorder, history-taking is essential, a physical examination is necessary (although the yield is low), and laboratory tests are required if the pattern of erectile dysfunction even hints at being generalized.

Treatment of erectile disorders are sometimes specific to the etiology (e.g., replacing a hormone that exists in insufficient amounts) and sometimes nonspecific, for example, oral medications (sildenafil [Viagra], psychotherapy, or intracavernosal injections. Counseling intervention ranges from being central (sex therapy) to being an adjunct (e.g., information about the use of vacuum erection devices).

The prevalence of erectile disorders makes primary care health professionals central to the care of men (and couples) with this disorder. Their task will likely be made easier by the introduction of safe and effective oral therapies. However, even with the advent of new forms of care there still will be treatment-resistant patients and couples, and in those instances, liberal use should be made of other approaches and specialists.

POSTSCRIPT

When a patient takes no action after treatment suggestions are made for erectile difficulties, clinicians should not necessarily be surprised or discouraged.

A 57-year-old divorced computer analyst was seen because of long-standing (about ten years) erectile difficulties that appear when alone in masturbation and on the occasional times he is sexually active with a partner. He experienced a myocardial

infarction four years before the referral. He lived alone for 25 years after a marriage that lasted three years. The longest relationship he had with a woman since then (he was not romantically or sexually interested in men) was four months and that was about 20 years before. Since then he had a few dates but none in the previous ten years because he felt that women would expect him to do what he felt was not possible, that is, to sexually "perform." Discontented with the suggestions made at the end of a thorough assessment, he insisted on special diagnostic vascular procedures about which he had read. He was unwilling to consider oral medications, intracavernosal injections, or VEDs and was angry about the suggestion of psychiatric care as part of a treatment "package." He did not appear again after two visits and canceled his last appointment.

A survey of men assessed for erectile problems in a urology clinic found that two years later over half had not followed up on recommendations.[74] Sexual and nonsexual reasons may have existed. A strong desire for return of erectile capability may not be durable after the discovery that treatment entails significant psychological and/or physical effort and discomfort. In addition, some men are quite resistant to the notion that the explanation for problems with the function of their genitalia may, in fact, lie elsewhere (e.g., the problems may be an expression of intimacy difficulties [see Appendix II]).

REFERENCES

1. Rosen RC, Leiblum SR: Erectile disorders: an overview of historical trends and clinical perspectives. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*, New York, 1992, Guildford Press, pp. 3-26.
2. Elliott ML: The use of "impotence" and "frigidity": why has "impotence" survived, *J Sex Marital Ther* 11:51-56, 1985.
3. Impotence. NIH Consensus Statement 10:1-31, 1992
4. Lue TF, Tanagho EA: Functional anatomy and mechanism of penile erection. In Tanagho EA, Lue TF, McClure RD (editors): *Contemporary management of impotence and infertility*. Baltimore,1988, Williams & Wilkins.
5. *Diagnostic and Statistical Manual of Mental Disorders, ed 4, Primary Care Version*, Washington, 1995, American Psychiatric Association.
6. Feldman HA et al: Impotence and its medical and psychosocial correlates: results of the Massachusetts male aging study, *J Urology* 151:54-61,1994.
7. Rosen RC, Leiblum SR: Erectile disorders: an overview of historical trends and clinical perspectives. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*, 1992, The Guilford Press, pp. 3-26.
8. Spector IP, Carey MP: Incidence and prevalence of the sexual dysfunctions, *Arch Sex Behav* 19:389-408, 1990.
9. Masters WH, Johnson VE: *Human sexual inadequacy*, 1970, Little, Brown and Company.
10. Althof SE, Seftel AD: The evaluation and management of erectile dysfunction, *Psych Clin N Am* 18:171,1995.
11. LoPiccolo J: Postmodern sex therapy for erectile failure. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and management*, New York, 1992, The Guilford Press, pp. 171-197.
12. Buvat J et al: Recent developments in the clinical assessment and diagnosis of erectile dysfunction, *Ann Rev Sex Res* 1:265-308,1990.

13. O'Leary MP et al: A brief male sexual function inventory for urology, *Urol* 46:697-706, 1995.
14. Schover LR, Jensen SB: *Sexuality and chronic illness: a comprehensive approach*, New York, 1988, The Guilford Press.
15. Slag MF et al: Impotence in medical clinic outpatients, *JAMA* 249:1736-1740, 1983.
16. Rosen RC, Leiblum SR, Spector IP: Psychologically based treatment for male erectile disorder: a cognitive-interpersonal model, *J Sex Marital Ther* 20:67-85, 1994.
17. Levine SB: Intrapsychic and interpersonal aspects of impotence: psychogenic erectile dysfunction. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*, 1992, The Guilford Press, pp. 198-225.
18. Masters WH, Johnson VE: *Human sexual response*, Boston, 1966, Little, Brown and Company.
19. Althof SE: Psychogenic impotence: treatment of men and couples. In Leiblum SR, Rosen RC (editors): *Principles and Practice of Sex Therapy: Update for the 1990s*, ed 2, New York, 1989, The Guilford Press, pp. 237-265.
20. Zilbergeld B: *The new male sexuality*, New York, 1992, Bantam Books.
21. Goldman A, Carroll JL: Educational intervention as an adjunct to treatment of erectile dysfunction in older couples, *J Sex Marital Ther* 16:127-141,1990
22. Cranston-Cuebas MA, Barlow DH: Cognitive and effective contributions to sexual functioning, *Ann Rev Sex Res* 1:119-161, 1990.
23. Turner LA et al: Self-injection of papaverine and phentolamine in the treatment of psychogenic impotence, *J Sex Marital Ther* 15(3):163-176, 1989.
24. De Amicis LA et al: Clinical follow-up of couples treated for sexual dysfunction, *Arch Sex Behav* 14:467-489, 1985.
25. Hawton K et al: Long-term outcome of sex therapy, *Behav Res Ther* 24:665-675, 1986.
26. Schiavi RC et al: Diabetes mellitus and male sexual function, *Diabetologia* 36:745-751, 1993.
27. Report of the expert committee on the diagnosis and classification of diabetes mellitus, *Diabetes Care* 20:1183-1197, 1997.
28. Nofzinger EA et al: Results of nocturnal penile tumescence studies are abnormal in sexually functional diabetic men, *Arch Internal Med* 152:114-118, 1992.
29. Meulemann EJ, Diemont WL: Investigation of erectile dysfunction: diagnostic testing for vascular factors in erectile dysfunction, *Urol Clin N Am* 22:803-819, 1995.
30. Virag R, Bouilly P, Frydman D: Is impotence an arterial disorder? *Lancet* 8422:181-184, 1985.
31. Condra M et al: Prevalence and significance of tobacco smoking in impotence, *Urol* 27:495-498, 1986.
32. Rosen MP et al: Cigarette smoking: an independent risk factor for atherosclerosis in the hypogastric-cavernosus arterial bed of men with arteriogenic impotence, *J Urol* 145:759-763, 1991.
33. Crenshaw TL, Goldberg JP: *Sexual pharmacology: drugs that affect sexual function*, New York, 1996, W.W. Norton & Company, Inc.
34. Segraves RT, Segraves KB: Aging and drug effects on male sexuality. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*, New York, 1992, The Guilford Press, pp. 96-138.
35. Schiavi RC: Chronic alcoholism and male sexual dysfunction, *J Sex Marital Ther* 16:23-33, 1990.
36. Lemere F, Smith JW: Alcohol-induced sexual impotence, *Am J Psychiatry* 130:212-213, 1973.
37. Whalley LJ: Sexual adjustment of male alcoholics, *Acta Psychiatr Scand* 58:281-298, 1978.
38. Jensen SB: Sexual function and dysfunction in younger married alcoholics: a comparative study, *Acta Psychiatr Scand* 69:543-549, 1984.
39. Snyder S, Karacan I: Effects of chronic alcoholism on nocturnal penile tumescence, *Psychosom Med* 43:423-429, 1981.
40. Bannister P, Lowowsky MS: Ethanol and hypogonadism, *Alcohol Alcoholism* 22:213-217, 1987.

41. Davidson JM, Rosen RC: Hormonal determinents of erectile function. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*, New York, 1992, The Guilford Press, pp. 72-95.
42. Buvat J, Lemaire A: Endocrine screening in 1,022 men with erectile dysfunction: clinical significance and cost-effective strategy, *J Urol* 158:1764-1767, 1997.
43. WHO Expert Committee on Diabetes Mellitus: *World Health Organization Technical Report Series 646*, Geneva, 1980 World Health Organization.
44. Buvat J et al: Is intracavernous injection of papaverine a reliable screening test for vascular impotence? *J Urol* 135:476-478, 1986.
45. Porst H: Diagnotic use and side-effects of vasoactive drugs—a report on over 2100 patients with erectile failure, *Int J Impotence Res* 2(2):222-223,1990.
46. Schiavi RC: Laboratory methods for evaluating erectile dysfunction. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*. New York,1992, The Guilford Press, pp. 141-170.
47. Schiavi RC et al: Healthy aging and male sexual function, *Am J Psychiatry* 147:766-771, 1990.
48. Nofzinger EA et al: Sexual function in depressed men, *Arch Gen Psychiatry* 50:24-30, 1993.
49. Nofzinger EA et al: Results of nocturnal penile tumescence studies are abnormal in sexually functional diabetic men, *Arch Intern Med* 152:114-118, 1992.
50. Kirkeby HJ et al. Erectile dysfunction in multiple sclerosis, *Neurology* 38,1366-1371, 1988.
51. Kirkeby HJ, Andersen AJ, Poulsen EU: Nocturnal penile tumescence and rigidity: translation of data obtained from normal males, *Int J Impotence Res* 1:115-125, 1989.
52. Levine LA, Carroll RA: Nocturnal penile tumescence and rigidity in men without complaints of erectile dysfunction using a new quantitative analysis software, *J Urol* 152:1103-1107, 1994.
53. Levine LA, Lenting EL: Use of nocturnal penile tumescence and rigidity in the evaluation of male erectile dysfunction, *Urol Clin N Am* 22:775-788, 1995.
54. Schreiner-Engel P: Therapy of psychogenic erectile disorders, *Sex Disability* 4:115-122, 1981.
55. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus, *N Engl J Med* 329:977-986, 1993.
56. McCullough DK et al: The prevalence of diabetic impotence, *Diabetologia* 18:279-283,1990.
57. Boolell M et al: Sildenafil, a novel effective oral therapy for male erectile dysfunction, *Brit J Urology* 78:257-261, 1996.
58. Lue T: Editorial Comment, *J Urol* 157:2021, 1997.
59. Boolell M et al: Sildenafil: an orally active type 5 cyclic GMP-specific phosphodiesterase inhibitor for the treatment of penile erectile dysfunction, *Int J Impotence Res* 8:47-52, 1996.
60. Wicker P: Phosphodiesterase inhibitors and male erectile dysfunction. Presented at the 1997 Annual Meeting of the Society for Sex Therapy and Research, Chicago, 1997.
61. Virag R et al: (Pfizer Central Reseach, UK): Sildenafil (Viagra), a new oral treatment for erectile dysfunction (editor): an 8 week double-blind, placebo-controlled parallel group study. Presented at the VII World Meeting of the International Society for Impotence Research, San Francisco, 1996.
62. Goldstein I et al: Oral sildenafil in the treatment of xerectile dysfunction, *N Engl J Med* 338:1397-1404, 1998.
63. Rosen RC, Ashton AK: Prosexual drugs: empirical status of the "new aphrodesiacs," *Arch Sex Behav* 22:521-543, 1993.
64. Mann K et al: Effects of Yohimbine on sexual experiences and nocturnal penile tumescence and rigidity in erectile dysfunction, *Arch Sex Behav* 25:1-16, 1996.
65. Carey MP, Johnson BT: Effectiveness of Yohimbine in the treatment of erectile disorder: four meta-analytic integrations, *Arch Sex Behav* 25:341-360, 1996.

66. Althof SE, Turner LA: Self-injection therapy and external vacuum devices in the treatment of erectile dysfunction. In Rosen RC, Leiblum SR (editors): *Erectile disorders: assessment and treatment*, New York, 1992, The Guilford Press, pp. 283-309.
67. Fallon B: Intracavernous injection therapy for male erectile dysfunction, *Urol Clin N Am* 22:833-845, 1995.
68. Donatucci CF, Lue TF: The combined intracavernous injection and stimulation test: diagnostic accuracy, *J Urol* 148:61-62, 1992.
69. Lue TF et al: Priapism: a refined approach to diagnosis and treatment, *J Urol* 166:104-108, 1986.
70. Padman-Nathan H: Treatment of men with erectile dysfunction with transurethral alprostadil, *N Eng J Med* 336:1-7,1997.
71. Melman A, Tiefer L: Surgery for erectile disorders: operative procedures and psychological issues. In Rosen RC, Lieblum SR (editors): *Erectile disorders: assessment and treatment*. New York, 1992, The Guilford Press, pp. 255-282.
72. Pederson B et al: Evaluation of patients and partners 1 to 4 years after penile prosthesis surgery, *J Urol* 139:956-958, 1988.
73. Schover LR: Sex therapy for the penile prosthesis recipient. *Urol Clin N Am* 16:91-98, 1989.
74. Tiefer L, Melman A: Adherence to recommendations and improvement over time in men with erectile dysfunction, *Arch Sex Behav* 16:301-308, 1987.