behavioral interventions). These approaches may be useful in patients who have psychogenic or social components to their ED, although data from randomized trials are scanty and inconsistent. It is preferable if therapy includes both partners if the patient is involved in an ongoing relationship.

FEMALE SEXUAL DYSFUNCTION

Female sexual dysfunction (FSD) has traditionally included disorders of desire, arousal, pain, and muted orgasm. The associated risk factors for FSD are similar to those in males: cardiovascular disease, endocrine disorders, hypertension, neurologic disorders, and smoking (Table 67-4).

EPIDEMIOLOGY

Epidemiologic data are limited, but the available estimates suggest that as many as 43% of women complain of at least one sexual problem. Despite the recent interest in organic causes of FSD, desire and arousal phase disorders (including lubrication complaints) remain the most common presenting problems when surveyed in a community-based population.

PHYSIOLOGY OF THE FEMALE SEXUAL RESPONSE

The female sexual response requires the presence of estrogens. A role for androgens is also likely but less well established. In the CNS, estrogens and androgens work synergistically to enhance sexual arousal and response. A number of studies report enhanced libido in women during preovulatory phases of the menstrual cycle, suggesting that hormones involved in the ovulatory surge (e.g., estrogens) increase desire.

Sexual motivation is heavily influenced by context, including the environment and partner factors. Once sufficient sexual desire is reached, sexual arousal is mediated by the central and autonomic nervous systems. Cerebral sympathetic outflow is thought to increase desire, and peripheral parasympathetic activity results in clitoral vasocongestion and vaginal secretion (lubrication).

The neurotransmitters for clitoral corporal engorgement are similar to those in the male, with a prominent role for neural, smooth-muscle, and endothelial released nitric oxide (NO). A fine network of vaginal nerves and arterioles promotes a vaginal transudate. The major transmitters of this complex vaginal response are not certain, but roles for NO and vasointestinal polypeptide (VIP) are suspected. Investigators studying the normal female sexual response have challenged the long-held construct of a linear and unmitigated relationship between initial desire, arousal, vasocongestion, lubrication, and eventual orgasm. Caregivers should consider a paradigm of a positive emotional and physical outcome with one, many, or no orgasmic peak and release.

Although there are anatomic differences as well as variation in the density of vascular and neural beds in males and females, the primary effectors of sexual response are strikingly similar. Intact sensation is important for arousal. Thus, reduced levels of sexual functioning are more common in women with peripheral neuropathies (e.g., diabetes). Vaginal lubrication is a transudate of serum that results from the increased pelvic blood flow associated with arousal. Vascular insufficiency from a variety of causes may compromise adequate lubrication and result in dyspareunia. Cavernosal and arteriole smooth-muscle relaxation occurs via increased nitric oxide synthase (NOS) activity and produces engorgement in the clitoris and the surrounding vestibule. Orgasm requires an intact sympathetic outflow tract; hence, orgasmic disorders are common in female patients with spinal cord injuries.

TABLE 67-4 RISK FACTORS FOR FEMALE SEXUAL DYSFUNCTION

Neurologic disease: stroke, spinal cord injury, parkinsonism
Trauma, genital surgery, radiation
Endocrinopathies: diabetes, hyperprolactinemia
Liver and/or renal failure
Cardiovascular disease
Psychological factors and interpersonal relationship disorders: sexual abuse, life stressors
Medications
Antiandrogens: cimetidine, spironolactone
Antidepressants, alcohol, hypnotics, sedatives
Antiestrogens or GnRH antagonists
Antihistamines, sympathomimetic amines
Antihypertensives: diuretics, calcium channel blockers
Alkylating agents
Anticholinergics

Abbreviation: GnRH, gonadotropin-releasing hormone.

APPROACH TO THE PATIENT: Female Sexual Dysfunction

Many women do not volunteer information about their sexual response. Open-ended questions in a supportive atmosphere are helpful in initiating a discussion of sexual fitness in women who are reluctant to discuss such issues. Once a complaint has been voiced, a comprehensive evaluation should be performed, including a medical history, a psychosocial history, a physical examination, and limited laboratory testing.

The history should include the usual medical, surgical, obstetric, psychological, gynecologic, sexual, and social information. Past experiences, intimacy, knowledge, and partner availability should also be ascertained. Medical disorders that may affect sexual health should be delineated. They include diabetes, cardiovascular disease, gynecologic conditions, obstetric history, depression, anxiety disorders, and neurologic disease. Medications should be reviewed as they may affect arousal, libido, and orgasm. The need for counseling and recognizing life stresses should be identified. The physical examination should assess the genitalia, including the clitoris. Pelvic floor examination may identify prolapse or other disorders. Laboratory studies are needed, especially if menopausal status is uncertain. Estradiol, follicle-stimulating hormone (FSH), and luteinizing hormone (LH) are usually obtained, and dehydroepiandrosterone (DHEA) should be considered as it reflects adrenal androgen secretion. A CBC, liver function assessment, and lipid studies may be useful, if not otherwise obtained. Complicated diagnostic evaluations such as clitoral Doppler ultrasonography and biothesiometry require expensive equipment and are of uncertain utility. It is important for the patient to identify which symptoms are most distressing.

The evaluation of FSD previously occurred mainly in a psychosocial context. However, inconsistencies between diagnostic categories based only on psychosocial considerations and the emerging recognition of organic etiologies have led to a new classification of FSD. This diagnostic scheme is based on four components that are not mutually exclusive: (1) *hypoactive sexual desire*—the persistent or recurrent lack of sexual thoughts and/or receptivity to sexual activity, which causes personal distress; hypoactive sexual desire may result from endocrine failure or may be associated with psychological or emotional disorders; (2) *sexual arousal disorder*—the persistent or recurrent inability to attain or maintain sexual excitement, which causes personal distress; (3) *orgasmic disorder*—the persistent or recurrent loss of orgasmic potential after sufficient sexual stimulation and arousal, which causes personal distress; and (4) *sexual pain disorder*—persistent or recurrent genital pain associated with noncoital sexual stimulation, which causes personal distress. This newer classification emphasizes "personal distress" as a requirement for dysfunction and provides clinicians with an organized framework for evaluation before or in conjunction with more traditional counseling methods.