General

Male Sexual Dysfunction

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Male sexual dysfunction is a series of conditions, most notably including erectile dysfunction (ED), Peyronie's disease (PD), and premature ejaculation (PE), defined by impaired sexual functioning. The prevalence of male sexual dysfunction increases with age and is relatively high with greater than 50% of men aged 40 to 70 describing some degree of erectile dysfunction. Risk factors for male sexual dysfunction include age, diabetes mellitus (DM), cancer, stroke, hypertension, penile trauma, depression, anxiety, and disturbance in central serotonin neurotransmission and 5-HT postsynaptic receptor functioning. Sexual questionnaires including the International Index of Erectile Dysfunction, Sexual Health Inventory for Men, and the Premature Ejaculation Diagnostic Tool are useful in screening for these disorders. Focused history and physical can establish diagnoses. For a condition to be diagnosed as male sexual dysfunction, the patient or their partner must view their sexual functioning as impaired. Treatment of male sexual dysfunction is etiology dependent. For ED, first-line therapy is a phosphodiesterase-5 inhibitor or mental health care for psychogenic ED. More complicated cases may be treated with injections, surgery, or shockwave therapy. PD is either treated with medications for pain management, collagenase clostridium histolyticum injection, corpoplasty, plication, or shockwave therapy. PE may be treated behaviorally or with SSRIs as first line medication.

INTRODUCTION

Sexual dysfunction occurs in both men and women, and the prevalence increases with age.[1] Dysfunction can occur in various stages of the normal sexual response cycle: desire, arousal, or orgasm; it can also be due to pain.[1] Human sexual function has physiologic and psychologic components, making it quite complex.[2] Diagnosis is based on clinical findings, so a detailed sexual history and focused physical examination are critical.[1] There are multiple causes of sexual dysfunction in men, all with different risk factors and treatments. Low sexual desire includes a lack of interest in thinking about sex or in being sexual, either alone or with a partner.[2] Erectile dysfunction (ED) is the consistent or recurrent inability to attain or maintain a penile erection sufficient for sexual satisfaction.[2] Erectile dysfunction is a quite common condition. Many men do not self-report erectile dysfunction symptoms, thus physicians must ask about sexual health and function to elicit a diagnosis.[3]

Peyronie disease is an acquired structural penile abnormality that causes curvature or other deformities of the erect penis.[2] Premature ejaculation is defined as a lack of ejaculatory control that is associated with distress.[2] Delayed orgasm/anorgasmia is the persistent or recurrent difficulty, delay in, or absence of attaining orgasm after sufficient sexual stimulation.[4] Many different treatment options are available for male sexual dysfunction including cognitive/behavioral, pharmacotherapy, and surgery. The pur-

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pose of this investigation is to explore the existing literature about the causes for male sexual dysfunction, consider its epidemiology, discuss its pathophysiology and risk factors, elucidate its clinical presentation and diagnosis, and examine the various treatment modalities that have been reported.

EPIDEMIOLOGY

It is difficult to compare findings because of the different ways sexual dysfunctions are defined, the population studied, as well as the age, medical history, and socioeconomic and cultural background of patients.[5–8] Premature ejaculation and erectile dysfunction are the most common sexual dysfunctions.[5,6,9] There is a great deal of literature that has studied the prevalence of erectile dysfunction (ED) and premature ejaculation (PE).[5,9] It has been well established that the prevalence of ED is positively correlated with age; 52% of men 40 to 70 years old describe some degree of ED.[5,7,8] One cross-sectional study found a fourfold increase in the prevalence of ED in men in their 70s compared with men in their 20s.[7] Premature ejaculation (PE) has been difficult to study as there is inconsistency with how the condition is defined.[5] There is limited literature on the prevalence of interest and desire disorders in men.[5] The prevalence of decreased interest or desire was reported in the range of 15% to 25% for varying age ranges and geographic locations.[5] Levels of sexual interest appear quite stable from the late teens to approximately 60, after which prevalence drastically increased.[5,8] Most studies report prevalence rates of orgasmic dysfunction (OD) in the range of 11.8% to 19.4%.[5] The worldwide report of OD by Nicolosi et al showed a prevalence of 5% to 8% for all areas of the world except for East and Southeast Asia, where the prevalence was 10% to 15%.[5,8] There is little literature on the prevalence of dyspareunia in men. Nickel et al reported a prevalence rate of painful ejaculation in a world survey of 16.8%.[5]

PATHOPHYSIOLOGY AND RISK FACTORS

Poor health is likely to correspond with low levels of sexual interest or desire and with various sexual dysfunctions, like ED and PE. However, independent of health, age has been suggested as a key risk factor for sexual dysfunction in men.[5,8] The Health Professional Follow-up Study in 1986, which consisted of a large cohort of male dentists, optometrists, osteopaths, podiatrists, pharmacists, and veterinarians in the United States who responded to a mailed questionnaire and were followed by a questionnaire every two years.[5] One finding of this study was a 10-fold difference in relative risk for ED associated with older age, regardless of health status or previous erectile function.[5] Men with a healthy lifestyle and no chronic disease had the lowest risk for ED.[5] Comorbid conditions, such as diabetes mellitus (DM), cancer, stroke, and hypertension, were associated with increased risk for ED, in contrast, physical activity, leanness, moderate alcohol consumption, and no history of smoking were associated with decreased risk. ED is dependent upon both vascular and neural processes.[3,5,10] The internal pudendal artery supplies most of the blood flow to the penis; venous outflow occurs through a network of compressible venules.[3,10] During arousal, parasympathetic activity from the sacral spinal cord initiates a cascade of events that releases nitric oxide (NO) and increases intracellular cyclic guanosine monophosphate (cGMP)[3,10] Increases in cGMP result in vascular smooth muscle relaxation and an increase in blood flow into the corpora cavernosa.[3,10] This rapid inflow of blood leads to compression of the venule network, decreasing venous outflow, thus raising intracavernosal pressure and causing erection.[3,10] Therefore, ED can result in any process that impairs the vascular or neural pathways that contribute to erection.[3,10,11] The two most common diseases that can cause ED are atherosclerosis and diabetes.[3,10,11] Lifestyle habits such as smoking and alcohol use as well as comorbid conditions like obesity can cause an increased risk for diabetes and atherosclerosis, leading to ED. Obesity can also cause hypogonadism and hormonal changes that can lead to ED.[3,10] Diseases that impair the nervous system, such as multiple sclerosis and Parkinson disease, can disrupt the neural pathways that cause erections, causing ED.[3,10,11] Moreover, injuries to the spinal cord can also disrupt these neural pathways and cause ED. Functional and neuroanatomical studies show the relationship between the level of injury and the erectile function of men caused by impairment of the descending, facilitative, and inhibitory pathways present in the spinal cord.[12] Side effects from medications and recreational drugs like antihypertensives, anti-androgens, anticholinergics, psychotropic agents, narcotics, marijuana, and alcohol have been shown to cause ED.[3,5,10,11] The early literature about the mechanism of action for PE were based entirely on conjecture, not on systematic research studies.[13] More recent psychopharmacologic studies have led to the hypothesis that early ejaculation is a neurobiological dysfunction related to a disturbance in central serotonin neurotransmission and 5-HT postsynaptic receptor functioning.[13] Although there are many psychological factors (such as depression, poor body image, and performance anxiety) that have been explored as explanations of PE, none have been validated with controlled research.[13] It is quite possible that apprehension over PE can lead to performance anxiety and subsequent ED.[13]

CLINICAL PRESENTATION AND DIAGNOSIS

Male sexual dysfunction can be a primary or secondary disease process. When a patient first presents with symptoms of sexual dysfunction it is important to first rule out an underlying conditions or medication resulting in secondary sexual dysfunction. Underlying conditions include cardiovascular disease, peripheral artery disease, diabetes mellitus, psychological factors, testosterone deficiency, neurologic disease, chronic kidney disease, hypertension, hyperlipidemia, endocrinopathies, thyroid disease, and obstructive sleep apnea. Laboratory tests such as vital signs, complete blood count, blood urea nitrogen, creatinine, lipid panel, hemoglobin A1C, thyroid panel, and hormone levels can help identify underlying conditions resulting in symptoms of sexual dysfunction. The AUA has a grade C recommendation for the measurement of morning serum testos-

terone for all patients presenting with symptoms of ED.[14] Medications that can result in sexual dysfunction are antidepressants, antihypertensives, antiandrogens, diuretics, opioids, sympathetic blockers, and anti-parkinsonian medication.[15] Social and lifestyle choices such as eating habits, exercise, tobacco use, alcohol use, and illicit drug use should also be discussed as these can precipitate symptoms. Findings on physical exam such as gynecomastia and underdeveloped pubic hair are associated with testosterone deficiency or other endocrinopathies. A genital exam should be done to assess testicular size, presence of penile lesions or abnormalities, or previous surgery.[16] Treatment of specific conditions or discontinuing offending agents can reduce or alleviate sexual symptoms.

ERECTILE DYSFUNCTION

Many patients presenting with ED often have one of the reversible etiologies mentioned above. To establish primary diagnosis of ED, a thorough sexual history should be gathered.

Sexual history is often an uncomfortable subject for many men. It is important to establish good patient rapport in order to form an honest and effective patient-physician relationship.

Sexual questionnaires are recommended to initiate discussion.[14] Multiple validated questionaries for men include International Index of Erectile Dysfunction, Sexual Health Inventory for Men, and the Premature Ejaculation Diagnostic Tool. Questionaries give providers a score that can be used to determine cause and severity of disease.[17] These scores, though effective, should not replace a quality sexual history and physical exam.[14] A sufficient sexual history should involve questioning about sexual orientation, current sexual relationships, emotional status, symptom onset, arousal, erection, ejaculation, and pain.[16,17]

Questions related to intimate partner relationships can point towards potential psychological factors. Does the patient feel comfortable with their current partner(s)? What is their level of sexual interest or desire? Even body image can play a factor in sexual confidence.[17]

Description of erections and ejaculation is important to determine the type of dysfunction. Providers should question the patient's ability to achieve and maintain an erection, as well as the presence of nocturnal erections, erections during masturbation, and erections during intercourse. Further questions regarding ejaculation should be asked, including the presence of rapid or delayed ejaculation, amount of ejaculate, and painful ejaculation.[16] If the patient endorses nocturnal erections but denies erections during other times of intimacy, this may indicate a psychologic component.[14]

Patients who present with painful erections or painful ejaculation should be evaluated for a lower urinary tract infection, benign prostatic hyperplasia, and prostate cancer, as ED can be a common presentation for these lower urinary tract diseases.[14] The AUA suggests performing a digital rectal exam, which can reveal an enlarged or nodular prostate signifying BPH or prostate cancer. While performing a urinalysis can reveal an underlying urinary tract infection.[16,18]

SPECIFIC DIAGNOSTIC TESTING

Radiologic testing, nocturnal penile rigidity testing, vascular and neurologic functional testing, and penile Doppler ultrasound are available for further diagnostic workup of ED. These tests are not routinely indicated in the primary care setting but may be ordered by urologists or sexual medicine specialists in certain cases.[16]

PEYRONIE'S DISEASE

Peyronie's disease is characterized by abnormal healing from penile trauma. Excessive collagen deposition within the tunica albuginea results in plaque formation. The collagen plaques stiffen the tunica albuginea which can result in penile deformity, abnormal penile curvature, penile pain, painful erections, and ED.[18] It is thought that this healing cascade is activated as a result of microtrauma during intercourse or a large penile injury such as a fracture. Patients most commonly present from the ages of fifty-two to fifty-seven with new onset abnormal penile curvature and/or mild penile pain during erection.[19] Plaques are often not felt while the penis is flaccid. The most common presenting symptom is penile deformity, followed by penile pain and palpable plaques. Patients may even present with ED as their only complaint.[18,20] Patient presentations vary in the amount of pain, severity of deformity, plaque location, and rate of plaque progression. Younger patients with similar symptoms often have had a history of an inciting penile injury, such as a sports injury to the groin (6).[18]

A preliminary diagnosis of PD can be made if a patient presents with this typical symptomatology. However, the AUA reports that there is insufficient literature on diagnostic criteria for PD, but a detailed history and physical exam remain the best diagnostic indicators for disease.[18,21] Certain imaging modalities can be used to support the diagnosis. When compared to MRI and X-ray, ultrasound has shown to have the highest sensitivity for plaque detection and provides a superior view of the tunica and surrounding soft tissue. With ultrasound, there is also the added benefit of lower cost and easy accessibility.[21,22] Prior to an invasive treatment (intralesional injection treatment, surgical resection of lesion, or penile prosthesis) PD patients must undergo color doppler ultrasound during pharmaco-induced erection to assess penile vasculature.[18,23]

Once diagnosis of PD is established, disease is then classified as active or stable. The active phase occurs early in the disease process and is characterized by dynamic progressing symptoms.[18,24] Plaque formation and growth primarily occur during the active phase with varying rates, as well as varying levels of pain with or without ED. Regular and frequent physical exams are used to track plaque formation, plaque progression, curvature degree, and penile length.[18] The stable phase is reached when penile deformity remains unchanged for three consecutive months.[24]

EJACULATORY DISORDERS

The DSM-5 defines premature ejaculation (PE) as a persistent or recurrent pattern of ejaculation occurring during partnered sexual activity within approximately 1 minute

following vaginal penetration and before the individual wishes it. PE must occur in at least 75% of sexual encounters for a period of at least 6 months according to DSM-5 criteria. PE can be categorized into mild, moderate, severe based on amount of time from to ejaculation. Mild is considered 30 seconds to 1 minute; moderate is 15-30 seconds; severe is less than 15 seconds.[25] Questionnaires have been developed for PE which include Premature Ejaculation Diagnostic Tool (PEDT), the Premature Ejaculation Profile (PEP), Index of Premature Ejaculation, the Multiple Indicators of Premature Ejaculation, and the Checklist for Early Ejaculation Symptoms (AUA cited studies 65-69). However, the AUA is still uncertain of the validity of questionnaires for the clinical diagnosis of PE. Physical examination has also been found to rarely contribute to PE diagnosis (AUA). Primary diagnosis is clinical.

The DSM-5 defines delayed ejaculation (DE) as marked delay or inability to achieve ejaculation despite stimulation and desire. Similar to PE, DE must occur in at least 75% of sexual encounters for a period of at least 6 months. No specific time delay milestones have been described.

Categorizations of mild, moderate, severe are based on level of stress brought upon the patient. Neuronal testing, include assessment of anal sphincter tone and bulbocavernosal reflex, may be done in the setting of lower extremity paresthesia or history of trauma to rule out a neurologic etiology.[26] Again, primary diagnosis is largely clinical.

Regardless of the disease process, providers should discuss the impact of dysfunction on the overall well-being and establish care goals based on the patient's needs.[16]

MEDICAL TREATMENT OPTIONS

When a patient first presents with symptoms of sexual dysfunction it is important to first identity any underlying conditions, or medications that could be the cause. Underlying conditions that can result in ED include psychological factors, testosterone deficiency, diabetes mellitus, chronic kidney disease, hypertension, hyperlipidemia, and obstructive sleep apnea. Medications that can result in ED are antidepressants, antihypertensives, antiandrogens, spironolactone, opioids, sympathetic blockers, ketoconazole, and cimetidine. Additionally, modifiable risk factors for ED should be evaluated. It is a grade C recommendation by the American Urological Association (AUA) that all patients with ED maintain a healthy diet, increase their exercise and quit smoking.[14] Additionally, findings from the Prostate Cancer Prevention Trial indicated that the presence of ED has been shown to be a major risk factor for cardiovascular disease (CVD), therefore patients should be properly educated on this increased risk.[27,28]

ERECTILE DYSFUNCTION

The phosphodiesterase-5 (PDE5) inhibitors, such as sildenafil, tadalafil, and vardenafil, are the first line therapy for males with ED.[29,30] PDE5 is an enzyme that is found in the smooth muscle of the corpora cavernosa and degrades cGMP to 5'-GMP. As previously discussed, PDE5 inhibitors lead to an accumulation of cGMP, causing an inward flow of blood, resulting in a prolonged erection.[16,28,31,32] The most common side effects of PDE5 inhibitors include headaches, nasal congestion, flushing, dyspepsia, back pain and myalgias.[28,30] Additionally, PDE5 inhibitors can cause severe hypotension if combined with nitrates, therefore patients taking nitrates should not take PDE5 inhibitors. Rare side effects include Steven-Johnson Syndrome, priapism, sudden vision loss, and sudden hearing loss.[30] Intracavernosal self-injection therapy and intraurethral suppositories are options for men who cannot take PDE5 inhibitors, or those that PDE5 inhibitors are not effective. Alprostadil is a synthetic form of prostaglandin E1 which acts as a muscle relaxant that results in increased blood flow and erection. Alprostadil can be used as a monotherapy, or it can be combined with phentolamine (BiMix), or papaverine and phentolamine (TriMix). The dosing must be closely titrated in office to obtain an erection lasting no more than one hour. Side effects of the injectable form is pain at the injection site, hematoma, priapism, corporal fibrosis, and decreased efficacy over time. Side effects for the intraurethral suppositories are urethral pain, and urethral infection.[16,28,29] Vacuum erection devices (a grade C recommendation) apply negative pressure to the penis, increasing corporal blood flow, and utilize occlusive rubber cuffs to prevent venous egress.[14,16] These devices can be purchased over the counter, and can be used alone, or in combination with PDE5 inhibitors.[16]

Newer treatment options currently being investigated are the use of stem cells and platelet rich plasma (PRP). There have been several published studies evaluating the intracavernosal injection (ICI) of stem cells as a way of rescuing the damaged cavernous nerve. These studies have evaluated cells derived from human placenta, bone marrow, and adipose tissue for the treatment of ED.[28,33] Regenerative therapies with stem cells have been shown to be effective in men who have undergone radical prostatectomy for prostate cancer treatment, as well has patients experiencing ED from diabetes mellitus.[34,35] The AUA currently lists ICI with stem cell therapy as a grade C recommendation for men with ED, stating the therapy should be considered "investigational".[14] PRP injection therapy has been studied in rodent models and humans, and has been associated with the potential mechanism of action of nerve regeneration.[28,33] Platelets have been shown to be involved with tissue regeneration through various factors such as platelet-derived growth factor, transforming growth factor-beta, and insulin-like growth factor.[28] These growth factors can lead to connective tissue regeneration, which has been associated with improved erectile function.[28] The AUA currently states that the use of PRP should be considered "experimental" in the treatment of ED.[14]

PEYRONIE'S DISEASE

Peyronie's disease (PD) is caused from abnormal healing after trauma, which results in abnormal extracellular matrix production and painful penile curvature.[20] Collagenase clostridium histolyticum (CCH) breaks down collagen type I and III, which are both associated with the abnormal extracellular matrix production seen in Peyronie's disease. Intralesional injection of CCH in combination with modeling is a grade B recommendation by the AUA for reduction in

penile curvature.[36] Penile ecchymosis and swelling are common side effects associated with intralesional CCH therapy.[20] A rare but serious side effect is corporal rupture.[36] Pain associated with Peyronie's disease can be managed with nonsteroidal anti-inflammatory drugs (NSAIDs).[20,36]

EJACULATORY DISORDERS

According to the AUA, first line treatment for premature ejaculation (PE) includes daily selective serotonin reuptake inhibitors (SSRIs), on demand clomipramine or dapoxetine, and topical penile anesthetics.[37] Serotonin has an inhibitory effect on ejaculation. SSRIs can be utilized in PE to maintain high levels of serotonin, which in turn suppresses ejaculation.[38] Topical anesthetics, when applied to the glans of the penis before sexual encounters have been shown to lower the penile perception threshold, resulting in delayed ejaculation.[38] In patients who fail the first line therapy, on demand tramadol is a grade C recommendation (14).[37] Behavioral methods, such as "stop and start," and "squeeze" have been shown to augment pharmacological treatment of PE.[37,38] Currently there are no pharmacologic treatments for delayed ejaculation, and modified sexual positions are advised.[37]

Lastly, when evaluating a male with sexual dysfunction, it is important to also identify sexual dysfunction in their partner, as each can exacerbate the other. One example of this is "Couplepause". Couplepause is a term that that is used to address the sexual health needs of a couple as individuals, as it relates to menopause and andropause, and as one. Addressing sexual dysfunction as it relates to a couple as opposed to just an individual has been shown to improve sexual health outcomes and satisfaction.[39]

SURGICAL TREATMENT OPTIONS

ERECTILE DYSFUNCTION

The inflatable penile prothesis (IPP) was invented in 1973 and was initially composed of three separate pieces. There is a pair of inflatable intracorporal cylinders which are connected to a small scrotal pump and a fluid reservoir located in the abdomen.[40] There have been many advances to the prothesis to enhance patient use, to minimize complications, and to aid in implantation.[40] Penile prothesis implantation is a grade C recommendation by the AUA.[14] Infection is the most feared complication of penile prothesis implantation, with revision surgeries, and longer operative time increasing the risk of infection, while antibiotic-impregnated devices lower post-op infection rates.[41] Penile revascularization surgery due to arterial insufficiency can be achieved through microvascular arterial bypass surgery. The inferior epigastric artery is primarily harvested for anastomosis and revascularization of the dorsal penile artery.[42,43] The inferior epigastric artery can also result in revascularization when anastomosis is preformed to the deep dorsal vein, although anastomosis to the dorsal artery is preferred.[43,44] Penile arterial reconstruction is a grade C recommendation by the AUA.[14] Penile venous surgery aims to impede venous drainage in order to retain blood within the corpora cavernosa during erection. The success rates for venous surgery are poor, and reported complications include painful erection, penile curvature, penile pain and numbness, and skin necrosis.[42–44] Due to the complications and poor outcomes venous surgery is not recommended for the treatment of ED.[14]

Shockwave therapy was first introduced as a non-invasive treatment for kidney stones, but since its implementation for kidney stones the utilization of shockwave therapy for other diseases processes has expanded and includes both ED and Peyronie's disease.[45] Low-intensity extracorporal shockwave therapy (Li-ESWT) was first introduced in 2010 for the treatment of vasculogenic ED. The energy created by the shockwave initiates the release of vascular endothelial growth factor, which induces cell proliferation and results in angiogenesis, wound healing and tissue regeneration.[45] There was a study of 160 men previously classified as "PDE 5 non-responders" who underwent a total of 8-20 sessions of Li-ESWT. During each session they received between 2500 and 5000 shockwaves depending on their tolerance. The study endpoint was the ability to perform vaginal intercourse for at least 3 months after therapy utilizing the same PDE5 inhibitor regimen they were using before the procedure. 45% of patients responded and were able to perform vaginal intercourse after therapy.[46] The AUA states the use of Li-ESWT in the treatment of ED as "investigational".[14]

PEYRONIE'S DISEASE

Patients with Peyronie's disease can be eligible for surgical therapy based on the presence of stable disease.[18] Penile corporoplasty is a surgical approach that focuses on straightening the penile curvature by shortening the convex side (opposite the area of curvature). Penile excisional corporoplasty is done by excising an elliptical-shaped portion of the outer longitudinal tunica fibers opposite the point of maximal penile curvature, and then bringing the excised tissue edges together using buried sutures.[20,47] If the excision is too large, a graft may be necessary for closure.[20] Penile incisional corporoplasty is done by making two longitudinal incisions on either side of the neurovascular bundle and closing them transversely.[47] Post-operative straightening success ranges from 29% to 100% and patient satisfaction ranges from 65% to 95%.[20] The most common post-operative complication/complaint is penile shortening. The tunica albuginea plication (TAP) procedure uses multiple partial thickness incisions made through the outer longitudinal tunica fibers (not including the circular fibers) to plicate the tunica in a vertical mattress fashion.[47] Both the penile excisional and incisional corporoplasty as well as the TAP procedure are Grade C recommendations by the AUA for the improvement of penile curvature in patients with PD.[18] Penile prothesis surgery using IPP can also be offered to patients with PD who also suffer from ED.[18]

Shockwave therapy using Li-ESWT has also been studied for the use of men with Peyronie's disease. In a study conducted on 190 males with PD, 47% showed greater than 50% reduction in plaque burden, and greater than 30% improvement in penile curvature after received up to 20 sessions.[46] The AUA does not recommend the use of shockwave therapy for the reduction of penile curvature or plaque burden,

but it has been recommended (Grade B) for the improvable of penile pain.[18]

CONCLUSION

Sexual dysfunction in men is multifaceted and quite complex; both physiologic and psychologic components contribute to the disorder. The etiology of the dysfunction can occur at various stages of the sexual response cycle including arousal, orgasm, or desire. A thorough literature review shows that ED and PE are the most common causes of male sexual dysfunction. Increasing age is the greatest risk factor for the development of sexual dysfunction, but many comorbid conditions such as diabetes, hypertension, obesity, and cancer show increase the risk of sexual dysfunction. It is imperative that physicians take a detailed history of sexual health and function to find a diagnosis. Treatments include both pharmacologic and surgical approaches. PDE5 inhibitors are first line for the treatment of ED, and PRP injections are under investigation as a future option for treatment. SSRIs are first line for the treatment of ejaculatory disorders which are more psychological in nature. Penile implants and revascularization procedures can be used as a surgical approaches to ED. Moreover, corporoplasty procedures are a surgical option for patients with Peyronie's disease to straighten the curvature of the penis.

In summary, the etiology of male sexual dysfunction should be investigated for the root cause in the patient. Clinicians should perform a thorough history and physical examination before treatment of these patients to prevent misdiagnoses. After which, conservative, pharmacologic, and surgical options should be evaluated on a clinical level considering symptom severity and burden on the patient to best treat men with sexual dysfunction.

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