

Kala Pohl. *Sweet Solitude*. Acrylic on canvas, 22" × 28".

Strategies to prevent or minimize sexual dysfunction in patients following surgical therapy for prostate or bladder cancer are reviewed.

Sexual Function After Surgery for Prostate or Bladder Cancer

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Background: *Compromised sexual function is often a side effect for patients following radical surgical procedures for bladder or prostate cancer.*

Methods: *The authors review the classification and physiology of sexual function and dysfunction. Moreover, they explain the possible pathophysiology directly resulting from surgery, and they discuss several approaches available to address these problems.*

Results: *Options for male sexual dysfunction, primarily erectile dysfunction resulting from radical prostatectomy or surgery for bladder cancer, range from patient education to penile prosthesis implantation. Female sexual dysfunction caused by surgical intervention for bladder cancer includes problems with libido, arousal, orgasm, and dyspareunia. Treatment options for women can include sex therapy, hormonal therapy, and preventive strategies. However, no consensus has been established on the most effective agents and time points to treat male or female sexual dysfunction following radical cystectomies or prostatectomies. The chronic intermittent treatment of erectile dysfunction following radical prostatectomy has been commonly referred to as penile rehabilitation.*

Conclusions: *Additional research is needed to obtain further data concerning sexual dysfunction in both men and women following radical pelvic surgeries. Modification of surgical techniques, the use of various treatment modalities for sexual dysfunction, and the development of new agents will help to successfully minimize or prevent damage and restore normal sexual function after local surgical therapy for prostate or bladder cancer in the future.*

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Submitted March 22, 2006; accepted April 28, 2006.

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No significant relationship exists between the authors and the companies/organizations whose products or services may be referenced in this article.

Abbreviations used in this paper: PDE = phosphodiesterase, VED = vacuum constriction device.

Introduction

Sexual dysfunction is characterized by disturbances in sexual desire and in the psychophysiologic changes associated with the sexual response cycle. Phases of this cycle — excitement, plateau, orgasm, and resolution — correspond to observable physiologic changes in men and women and have been used to define sexual dysfunction diagnoses according to the American Psychiatric Association's Diagnostic and Statistical Manual (DSM-IV) and the World Health Organization's consensus conferences.^{1,2}

In general, the most common form of sexual dysfunction in men is premature ejaculation. Male erectile dysfunction is the second most common entity and is defined as the inability to achieve or maintain a penile erection sufficient for satisfactory sexual performance.³ This condition affects an estimated 30 million men in the United States and approximately 152 million men worldwide.⁴ The etiology can be psychogenic, but organic causes predominate. Multiple risk factors, comorbidities, and iatrogenic causes are included in Table 1.

Despite perceptions that sexual dysfunction is a male-predominant condition, more women than men report some compromise in their sexual performance (43% and 31%, respectively).⁵ Research in the field of female sexual function and dysfunction has increased. Female sexual dysfunction is a complex spectrum caused by disturbances in the normal sexual response cycle. Components affecting this cycle include anatomic, physiologic, psychological, and social factors. The prevalence of female sexual dysfunction is approximately 42% in premenopausal women and 88% in postmenopausal women.⁶ Common complaints include diminished vaginal lubrication, pain and discomfort during sexual intercourse, decreased arousal, and difficulty achieving orgasm.

In general, any malignancy affecting the pelvis — from either the primary cancer or the required treatment — can eventually lead to sexual dysfunction.

Table 1. — Organic Causes of Erectile Dysfunction

Hypertension	Certain sports-related activities
Diabetes mellitus	Spinal cord injury
Dyslipidemia	Many chronic
Chronic renal disease	Neurological diseases
Smoking	Multiple endocrine disorders
Chronic alcoholism	Vascular insufficiency
Chronic marijuana use	Hormonal derangement
Chronic narcotic use	Interrupted neural pathways
Peyronie's disease	More than 300 medications
Penile trauma	Surgery and trauma
Increasing age	Hemodialysis

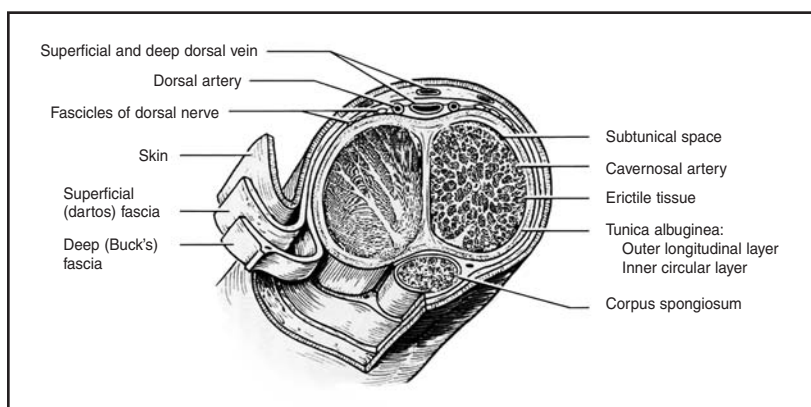


Fig 1. — Cross-section of the penis demonstrating relationships between penile layers and various components. From *Oral Pharmacotherapy for Male Sexual Dysfunction: A Guide to Clinical Management*. Broderick Ga, ed. Totowa, NJ: Humana Press; 2005. Reprinted with permission.

Gynecologic, gastrointestinal, and urologic malignancies can cause problems. Quality of life is an important component of multimodal treatment for cancer. Moreover, sexual function, being a critical quality-of-life predictor, has become an integral factor of this evaluation.

Prostate cancer is the leading cancer diagnosis in men and the third most common cause of cancer-related death in men in the United States.⁷ The lifetime risk of developing prostate cancer is 19% in the United States. Risk factors include older age, family history, race and ethnicity, and possibly dietary fat,⁸ but the etiology of this cancer remains unknown. With the widespread use of prostate-specific antigen testing and digital rectal examination as screening tools, the incidence of prostate cancer in the United States has increased.

Bladder cancer, the fifth most common cancer in the United States,⁷ typically presents as a superficial transitional cell carcinoma that is easily resectable endoscopically. However, local recurrence rates are high (66% at 5 years and 88% at 15 years), and between 10% to 30% progress to invasive cancer.^{9,10} Therapeutic options include surgery, radiation, and chemotherapy, but muscle-invasive bladder cancer typically necessitates radical cystectomy with urinary diversion.¹¹

This review describes changes in patients' sexual function after local surgical therapy for prostate or bladder cancer and discusses strategies to prevent or minimize sexual dysfunction in these patients.

Sexual Dysfunction Classification and Pathophysiology

Men

The penis is composed of three cylindrical structures, the paired corpus cavernosum (CC) and the corpus spongiosum. A cross-section of the mid-penis depicts the relationship between the various anatomic elements (Fig 1). The penis is innervated by both the autonomic and somatic nervous systems. Somatic innervation is

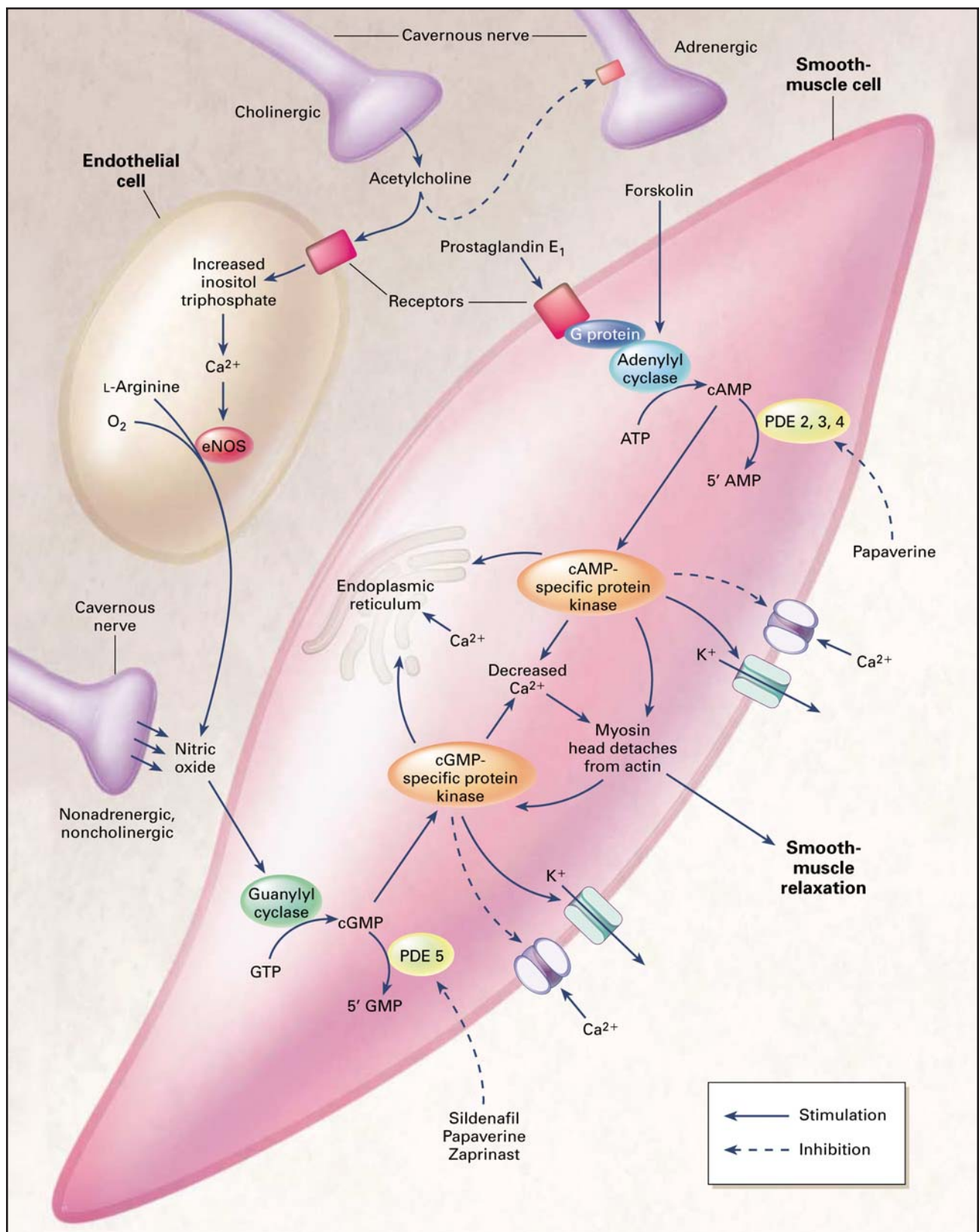


Fig 2. — Molecular mechanism of penile smooth-muscle relaxation. Cyclic AMP (cAMP) and cyclic GMP (cGMP), the intracellular second messengers mediating smooth-muscle relaxation, activate their specific protein kinases, which phosphorylate certain proteins to cause opening of potassium channels, closing of calcium channels, and sequestration of intracellular calcium by the endoplasmic reticulum. The resultant fall in intracellular calcium leads to smooth-muscle relaxation. Sildenafil inhibits the action of phosphodiesterase type 5 (PDE-5), thus increasing the intracellular concentration of cGMP. Papaverine is a non-specific phosphodiesterase inhibitor. GTP denotes guanosine triphosphate and eNOS denotes endothelial nitric oxide synthase. From Lue TF. Erectile dysfunction. *N Engl J Med.* 2000; 342:1802-1813. Copyright © 2000 Massachusetts Medical Society. All rights reserved.

derived from the S2-S4 sacral nerve roots via the pudendal nerve. These paired nerves supply the pelvis, perineum, and penis. They terminate as the dorsal nerve of the penis. Sexual stimulus releases nitric oxide from the terminal ends of the cavernosal nerves. This increases the level of guanosine 3',5'-cyclic monophosphate (cGMP) with subsequent smooth-muscle relaxation of the arteries and CC, thus allowing significant blood inflow. To maintain an erection, this blood needs to be trapped within the CC. This is achieved by the passive mechanical compression of the CC against the tunica albuginea, thus occluding the venous drainage and maintaining the erection.¹²⁻¹⁵ Physiologically, phosphodiesterase (PDE) enzymes modulate this pathway by inactivating cGMP, which results in elevated cytosolic calcium concentrations and smooth-muscle contraction. Hence, the interactions of the autonomic nervous system, coupled with the mediating transmitters, are integral in the contraction and relaxation physiology of the cavernous smooth-muscle cell (Fig 2).¹⁶⁻¹⁸

Erectile dysfunction can be caused by organic or psychogenic factors. Organic factors include vascular, neurogenic, and hormonal causes. Erectile dysfunction following pelvic surgery is usually due to a neurogenic component secondary to damage to the cavernosal nerve.¹⁹ As a consequence, the penile tissue undergoes an intense remodeling process characterized by a decrease in smooth-muscle cells and an increase in collagen synthesis that leads to reduced compliance of the CC and tunica albuginea during erection.²⁰

Women

Sexual response in both men and women can be divided into four phases on the basis of physiologic characteristics and subjective reports. These phases are excitement, plateau, orgasm, and resolution.^{21,22} In the excitement phase, female sexual arousal is the final expression of a complex process involving sexual stimulation, ascending/ descending steady control by the central nervous system, and peripheral neurovascular changes in a normal hormonal environment.²³ Physiologically, this phase begins with the engorgement of the vaginal mucosa, causing thickening of the vaginal walls and transudation of fluid into the vagina. Orgasm is defined as an altered state of consciousness associated with primarily genital but also nongenital sensory input. A so-called "orgasmic platform" in women, potentially responsible for either the genital pleasure at the peak and a possible biological basis for the greater capacity for multiple orgasms, has been suggested as the result of genital sexual arousal.^{24,25} Sensory trigger points have been advocated at the orgasmic platform level, including the clitoris and vagina. Any biological modification of these trigger points and areas can significantly affect a woman's orgasmic phase.

Female sexual dysfunction is a complex neurovascular phenomenon under psychologic and hormonal control. Various pelvic cancer pathology and/or subsequent treatment can affect each aspect of the female sexual cycle at different levels. An international consensus was established recently to develop a classification system of female sexual dysfunction. This classification system consists of four major categories: desire disorders, arousal disorders, orgasmic disorders, and sexual pain disorders.¹

Local Surgical Treatment of Prostate Cancer

Radical prostatectomy has an immediate and significant impact on erectile function. It can affect nocturnal, morning, and psychogenic erections. Bilateral nerve-sparing procedures do not guarantee preservation of sexual potency. Moreover, a significant proportion of men undergoing radical prostatectomy fail to recover to the preoperative levels of erectile function. Reported rates of recovery of erectile function after prostate surgery vary widely, ranging from 9% to 86%.²⁶⁻³⁵ This wide range of outcomes may be due to differences in assessments used to evaluate erectile function status or may be the result of the extent and precision of the nerve-sparing surgical techniques used in different institutions.

Several factors are involved in the etiology of erectile dysfunction after radical prostatectomy, even when a nerve-sparing technique is used. Direct neurogenic injury is the most obvious effect. In addition, blunt nerve damage can play an important but often overlooked role in the risk for erectile dysfunction. This category of blunt, indirect injury to the nerve can include stretch, thermal, and ischemic injuries. Also, nonneurogenic causes such as advanced age and vascular and psychologic factors can result in erectile dysfunction.^{34,36}

Iatrogenic vascular injury has been related to erectile dysfunction after radical prostatectomy.³⁷ Inadvertent injury to internal and accessory pudendal arteries that provide blood supply to the penis has been described in up to 85% of patients.^{38,39}

Atrophic and fibrotic changes of the penis occur in men who have undergone radical prostatectomy.^{40,41} Degeneration of nerve terminations occurs within the erectile tissue, as well as corporeal smooth-muscle deterioration and infiltration of the erectile tissue with collagen.^{36,42} This type of tissue destruction alters the tunical compliance needed for the maintenance of an erection and predisposes to the development of veno-occlusive erectile dysfunction. The mechanisms involved in these changes are consistent with pathways known to occur with hypoxic injury. Apoptosis is one mechanism involved in erectile tissue degeneration following penile denervation in rats.⁴³

Local Surgical Treatment of Bladder Cancer

Women

There is a paucity of research devoted to evaluating female sexual function after major urologic surgery for bladder cancer. However, a recent report indicates that during radical cystectomy in women, the neurovascular bundles (located on the lateral walls of the vagina) are usually removed or damaged by removal of the bladder, urethra, and anterior vaginal wall.⁴⁴⁻⁴⁶ In addition, significant devascularization of the clitoris often occurs with removal of the distal urethra, thus affecting subsequent sexual arousal and desire.^{44,46} Acute surgical menopause after formal radical cystectomy can also compound the problem. Hence, postoperative sexual dysfunction is common in women. Using the validated questionnaire, Female Sexual Function Index to evaluate changes in sexual function after surgery, Zippe et al⁴⁷ assessed the effects of radical cystectomy, the type of urinary diversion, and particular surgical modifications on female sexual functioning. Among 27 patients, only 13 (48%) were able to have successful vaginal intercourse, and 14 (52%) reported decreased satisfaction in overall sexual life after radical cystectomy. The authors concluded that the type of continent diversion performed does not affect sexual function. Furthermore, they recommend several surgical modifications that may improve female sexual function, including routine preservation of the distal urethra in selected diversions in an effort to preserve the clitoral neurovasculature, preservation of the anterior vaginal wall (as much as possible) to maintain vaginal lubrication and neurovascular innervations, and tubular reconstruction of the vagina (vs posterior flap rotation) to preserve vaginal depth and maintain pain-free intercourse. These surgical modifications apply only if cancer control is not compromised. An earlier study by Horenblas et al⁴⁸ evaluated the effectiveness of sparing all internal genitalia in women in addition to the urethra in appropriate candidates. The authors concluded that such surgical modifications to the radical cystectomy procedure help preserve sexual function.

Physical and emotional factors, such as a decrease in sexual attractiveness, can influence sexual life after radical cystectomy and bladder reconstruction surgery.⁴⁹⁻⁵¹ Bjerre et al⁵² evaluated the sexual profile after urinary diversion and found that almost one third of the women indicated physical problems or decreased desire and 30% felt less sexually attractive after cystectomy. This shows that sexual function is sensitive to both physical and mental effects from the treatment of bladder cancer. These psychologic and biogenic factors after radical cystectomy can make it difficult to evaluate female sexual dysfunction.^{3,45,49-51,53} Further studies with particular emphasis on postoperative management strategies are

needed to allow the surgeon to optimize postoperative sexual functioning.

Embryologically, the clitoris is the female analogous structure to the penis. Hence, it is not surprising that there are similar physiologic mechanisms involved within the corpora cavernosa of the clitoris. Nitric oxide-mediated stimulation of clitoral cavernosal smooth muscle increases blood flow and results in genital engorgement, which is important in female sexual arousal.^{15,54,55} Thus, by improving clitoral sensation and blood flow, sildenafil citrate may improve vaginal lubrication and sexual satisfaction.^{54,55} Reports in the literature regarding the use of sildenafil citrate for female sexual dysfunction are conflicting, and a clear consensus on its effectiveness has not yet been established.

Men

The efficacy of nerve-sparing techniques to preserve potency in men following cystoprostatectomy is approximately 50%, but modifications to the standard radical cystectomy procedure have been developed. Muto et al⁵⁶ reported a seminal-sparing cystectomy modification involving a posterior bladder dissection during radical cystectomy that is anterior to the seminal vesicle plane to preserve the vasa deferens, seminal vesicles, prostatic capsule, and neurovascular bundles. The authors found that normal erectile function was preserved in 95% of patients with a mean follow-up of 68 months. Their procedure was performed in patients without pathology in the bladder neck or prostate. This technique can also help preserve ejaculatory function. In a similar study by Colombo et al,⁵⁷ nerve- and seminal-sparing cystectomy offered satisfactory clinical and functional outcomes. The authors stressed this option should be considered only for young, fully potent, and socially active patients with organ-confined bladder cancer. Burday et al⁵⁸ reported good potency preservation with their prostate-sparing cystectomy series, which included patients who underwent partial or complete preservation of the prostate and neobladder formation. Their results have paralleled that of other series showing good functional outcomes after performing partial or complete preservation of the prostate during cystectomy.⁵⁹⁻⁶³ These studies re-emphasize that the risk of erectile dysfunction in cystectomy patients is related specifically to the pathology involved with surgical removal of the prostate gland.

Therapy for Sexual Dysfunction

Male Erectile Dysfunction

Several treatment modalities are available to manage sexual dysfunction (Table 2). First-line therapies include patient education, lifestyle modification, psychotherapy, oral therapy, and the use of a vacuum device. Second-

Table 2. — Management Options for Male Erectile Dysfunction

Lifestyle changes	Mechanical agent
Changing medication	Neuromodulation
Pelvic floor muscle exercise	Surgery:
Psychosexual therapy	revascularization
Hormonal therapy	venous ligation
Oral agents	nerve transplant (sural)
Topical agents	penile implant

line therapies include intraurethral alprostadil and intracavernous injection therapy, and third-line options include penile prosthesis implantation.

One group of oral agents is composed of the selective inhibitors of phosphodiesterases type-5 (PDE-5), the enzyme that breaks down the intracellular second messenger of erection, cGMP. When nitric oxide enters a vascular smooth-muscle cell, it triggers a cascade of reactions leading to the production of cGMP and subsequent smooth-muscle relaxation. The breakdown of these second messengers (cAMP and cGMP) is regulated by the set of enzymes known as PDEs (Fig 2). Thus, these oral agents enhance the natural effects of nitric oxide on corporal arterial and sinusoidal smooth muscle by inhibiting catabolism of cGMP by PDE-5.^{13,64}

For neurogenic causes of erectile dysfunction, the nerve-sparing techniques and PDE-5 inhibitors have been shown to improve the degree of erectile function. One study⁶⁵ evaluated sexual function in a series of patients who underwent a variety of nerve-sparing radical prostatectomies. Then, if indicated, they received either of two different doses of sildenafil citrate postoperatively. The authors reported that successful treatment of erectile dysfunction with sildenafil citrate after radical prostatectomy was dependent on the presence of the neurovascular bundles. Patients who underwent bilateral nerve-sparing techniques performed better than those undergoing unilateral or no nerve-sparing procedures. The response to sildenafil citrate was not related to the interval between the surgery and initiation of drug therapy but was related to dose.⁶⁵ Other studies have reported preservation of sexual function in 70% to 80% of patients treated with sildenafil citrate following radical prostatectomy.^{66,67} Vardenafil after nerve-sparing radical retropubic prostatectomy improved erection in 71.1% and 59.7% of patients taking 20 mg and 10 mg of vardenafil, respectively, during 12 weeks compared with 11.5% in the placebo group.⁶⁸ A study evaluating tadalafil in the post-prostatectomy patient showed similar efficacy.⁶⁹

A recent study by Schwartz et al⁷⁰ evaluated the histologic effects of adding sildenafil citrate during the postoperative course in prostatectomy patients. Sildenafil was given to 40 potent volunteers who were given either 50 mg or 100 mg of sildenafil citrate every other night for 6 months beginning the day of Foley

catheter removal after radical retropubic prostatectomy. A statistically significant increase in mean smooth-muscle content was seen in the high-dose group (56.85%) compared with the low-dose group (42.82%) ($P < .05$). The authors concluded that at higher doses following retropubic prostatectomy, sildenafil may increase smooth-muscle content. The effect on the return of potency is not known, but maintaining the pro-erectile ultrastructure is an integral part to rehabilitating erectile function following retropubic prostatectomy.⁷⁰ Therefore, it is appropriate to consider use of any of these oral agents to preserve the pro-erectile cyto-ultrastructure before and after surgery for bladder and prostate cancer.⁷¹

Intraurethral therapy with alprostadil, the synthetic formulation of prostaglandin PGE₁, involves inserting a vasodilatory agent into the urethra. The drug diffuses from urethra to the corpus spongiosum and then to the corpus cavernosum through venous channels. To assess the role of postoperative alprostadil in patients following prostatectomy, a study from the Walter Reed Medical Center⁷² evaluated prostatectomy patients who received doses of transurethral alprostadil in the clinic. Patients for whom a suitable dose was determined received treatment at home with active drug or placebo for 3 months. Of the 384 patients in whom radical prostatectomy was identified as a cause of erectile dysfunction, 70.3% had an erection believed sufficient for intercourse in the clinic, and 57.1% on active medication had sexual intercourse at least once at home. The overall success rate (ie, the likelihood of active treatment to lead to intercourse at home) was 40.1%. A more recent study⁷³ reported consistent efficacy of medicated urethral system for erection in the postprostatectomy patients regardless of the nerve-sparing status.

Intracavernous injections involve direct injections of papaverine, phentolamine, and alprostadil separately or in combination. The molecular mechanism of action is through inhibition of PDE-5, leading to increased cAMP and cGMP in penile erectile tissue. Advantages are high efficacy and stability at room temperature. Disadvantages include priapism (0% to 35%) and corporeal fibrosis (1% to 33%, mainly due to papaverine). Montorsi et al⁷⁴ evaluated the recovery of sexual function with postoperative intracavernosal injections of PGE-1 in prostatectomy patients. The recovery rate of spontaneous erections in patients who had early institution of postoperative PGE-1 injections was higher than those who did not. This small study prompted investigators to seek other erectile rehabilitation regimens in order to maximize the return of normal sexual function following radical prostatectomy and radical cystectomy. The concept of penile or erectile rehabilitation involves managing patients on a long-term basis involving one or more of the treatment modalities described.

The vacuum constriction device (also known as vacuum erection device, VED) consists of a plastic cylinder connected directly to a vacuum-generating source (manual or battery-operated pump). After the penis is engorged by the negative pressure, a constricting ring is applied to the base to maintain the erection. Combining intracorporeal injection with the VED may enhance the degree of tumescence.⁷⁵ A study evaluating the use of VED after radical prostatectomy showed that 92% responded to the VED (with an erection sufficient for vaginal penetration), but only 14% agreed to continue it at home.⁷⁶

The penile prosthesis remains one of the most effective treatments for all types of erectile dysfunction, especially after cavernosal nerve damage. Patient and partner satisfaction rates with the penile prostheses generally range from 60% to 80%,⁷⁷ but a common post-operative complaint is inadequate penile length. Other disadvantages of this treatment modality are the invasiveness of the procedure and inherent surgical risks.

Therapy for Female Sexual Dysfunction

Several problems can arise that have a negative effect on sexual function in women. Some of these involve problems with libido, arousal, orgasm, and dyspareunia.

Low Libido: Interest is increasing in androgens and their ability to alleviate problems of low desire.⁷⁸⁻⁸⁰ Some studies report that androgen treatment increases sexual desire and fantasies.⁸¹ In a prospective, 2-year, single-blinded, randomized trial of 34 postmenopausal women, a combination of estrogen and testosterone therapy led to greater improvements in multiple measures of sexuality than achieved by estrogen therapy alone.⁷⁸ Moreover, other studies⁸⁰ have found testosterone replacement to be successful in restoring desire and sexual responsiveness in patients who had a marked decrease in their desires as a result of surgery or chemotherapy. In addition to its benefit as a sexual motivator, testosterone maintains bone mass in both men and women. Virilizing side effects are rare but can include acne, hirsutism, and deepening of the voice.⁷⁸ Widespread use of testosterone replacement remains controversial for menopausal women, particularly in perimenopausal women.^{82,83}

Arousal: The success of vasodilating medications in male erectile dysfunction triggered the interest in its use for female sexual arousal disorders. The involvement of vascular congestion and the physiologic and biochemical similarities between the penis and the clitoris strengthened this interest and triggered several research projects. This included studies evaluating oral medications such as sildenafil citrate⁸⁴ and topical vasodilators.⁸⁵ Initially, the target population included all women with female sexual dysfunction in the hope that the broad-spectrum efficacy in male erectile dysfunction could be reproduced in female sexual dysfunction.

A multicenter, placebo-controlled, randomized, double-blind study was conducted with women using estrogen who were experiencing sexual dysfunction that included arousal disorder.⁸⁴ Results indicated that sildenafil administered on an as-needed basis for 12 weeks did not improve the sexual response in this population. Most studies on the efficacy of sildenafil citrate in female sexual arousal disorder fail to show any significant improvement, and no efforts are being made to seek approval by the US Food and Drug Administration for sildenafil citrate as a treatment option for female sexual dysfunction.

Orgasm: Treatment of orgasmic dysfunction is best managed in patients with reversible causes. One of these causes involves patients taking oral selective serotonin release inhibitors (SSRIs). Side effects of SSRIs can be managed with a number of strategies: dose adjustment, medication changes, drug holidays, drug augmentation, and most recently, administration of sildenafil citrate and other vascular drugs. Many patients who take SSRIs suffer from depression that is intrinsically or directly related to their female sexual dysfunction. Some may even suffer from depression secondary to their female sexual dysfunction. Women suffering from anorgasmia may have strong negative attitudes about sexuality and their bodies, and they may be unwilling to touch their own genitalia. Sex therapy plays a primary role in these conditions. The vibrator is the single most frequently used mechanical device with sex therapy.^{86,87} This device delivers a powerful erotic stimulus when applied to the clitoris and may be helpful for women with anorgasmia.⁸⁷

Dyspareunia: The first step in treating dyspareunia (painful coitus) is to address potential reversible causes such as vaginitis, endometriosis, and anatomic abnormalities. Topical or oral estrogens and lubricants can be used to relieve dyspareunia in patients with poor lubrication following procedures such as pelvic radiation.⁸⁸ Estrogen therapy can help alleviate symptoms such as vasomotor instability, minor psychologic disturbance, and sexual difficulties. Patients with atrophic vaginitis (fragile, thin tissues with decreased elasticity) and with poor lubrication are readily amenable to treatment with estrogen.⁷⁹

Generally, a multidisciplinary approach with input from sex therapists, psychologists, psychiatrists, urologists, and gynecologists who specialize in the field can optimize the efficacy of any treatment plan for women suffering from sexual dysfunction.

Conclusions and Future Approaches

The high prevalence of sexual dysfunction after surgical treatment for prostate or bladder cancer has increased efforts to seek effective methods to prevent

the damage from surgery and to restore normal sexual function. Montorsi et al⁷⁴ prospectively assessed the effect of postoperative intracavernous injections of alprostadil on the recovery of spontaneous erectile function after nerve-sparing radical retropubic prostatectomy. They concluded that early postoperative administration of injections increases the recovery rate of spontaneous erections after nerve-sparing radical retropubic prostatectomy. However, the optimal formula for penile rehabilitation, before and/or after radical prostatectomy, remains unclear. There is no consensus to clearly define which agents and which time points are most effective. Some clinicians believe that in the early postoperative period, intracavernosal injection therapy and VED are indicated, with the subsequent addition of a PDE-5 inhibitor once the patient obtains spontaneous erections.⁸⁹ Others believe that the concept of prophylaxis for conditioning of the vasculature of the penis is critical. Mancini et al^{90,91} studied Doppler duplex sonographic changes to compare alprostadil, sildenafil citrate, and placebo using chronic dosing for arterial conditioning. They demonstrated improvement in Doppler duplex sonographic peak systolic velocity by 30% with alprostadil and 39% with sildenafil.

The international multidisciplinary consensus panel on female sexual dysfunction in 1999¹ cited the lack of adequate experimental or clinical trial data and recognized the broad need for basic and applied research in this area. The report emphasized deficits in areas such as epidemiologic research, anatomic studies, biologic mechanisms of sexual arousal and orgasm, effects of aging and menopause, development of reproducible measurement devices, and instruments for evaluating physiologic parameters of the female sexual response in the clinical setting. Current efforts to obtain further data concerning female sexual dysfunction and the continued research in male erectile dysfunction should lead to new tools and management options that will minimize the risk of sexual dysfunction after local surgical therapy for prostate or bladder cancer.

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