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17.1 Introduction

Masters and Johnson were some of the first researchers to study and describe the sexual response cycle in their 1966 book *Human Sexual Response* [1].

The cycle begins with excitement as blood rushes into the genitals, then reaches a plateau during which they are fully aroused, which leads to orgasm, and finally resolution, in which the blood leaves the genitals.

Desire and arousal are both part of the excitement phase of the sexual response.

Sexual dysfunction (SD) refers to a problem occurring during any phase of the sexual response cycle that prevents the individual or couple from experiencing satisfaction from the sexual activity.

Sexual dysfunctions are highly prevalent, affecting about 43 % of women and 31 % of men [2, 3], even if it is a topic that many people are hesitant to discuss.

Moreover, in addition to their widespread prevalence, sexual dysfunctions have been found to impact significantly on interpersonal functioning and overall quality of life in both men and women, as confirmed by a large study involving 27,500 men and women in 29 countries (aged 40–80 years), which was conducted to assess the importance of sex and the prevalence of sexual dysfunction. In the results, 82 % of men and 76 % of women agreed with the statement that “a satisfactory sex life is essential to maintaining a relationship” and the majority disagreed with the statement that “older people no longer want sex” [4].

Several factors (physical, hormonal, psychological, social) contribute to sexual dissatisfaction or dysfunction, and these factors tend to be interrelated. Age was an

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important correlate of lubrication difficulties among women and of several sexual problems, including a lack of interest in sex, the inability to reach orgasm, and erectile difficulties among men.

Factors involved in sexual functions associated with chronic diseases and cancer are reported in Table 17.1.

The traditional sexual response cycle including excitement, plateau, orgasm, and resolution set the foundation for studying and categorizing sexual dysfunctions in men and women.

Sexual dysfunction generally is classified into four categories:

- *Desire disorders—lack of sexual desire or interest in sex:* Sexual desire disorders or decreased libido (also known as hypoactive sexual desire disorder; HSDD) are characterized by reduction or absence for some period of time of sexual desire or libido for sexual activity or of sexual fantasies. The condition ranges from a

Table 17.1 Factors involved in sexual functions associated with chronic diseases and cancer

Type	Mechanisms	Examples
Direct	Change in sexual desire from disease	Typically reduced, e.g., from high prolactin and anemia of chronic renal failure [1]. May be increased, e.g., from some brain disorders [2]
	Disruptbn of genital response from disease	ED from multiple sclerosis [3], hypertension [4], orgasmic disorder from multiple sclerosis [5]
	Disruptbn of genital response from surgery	Radical prostatectomy and ED [6], radical hysterectomy and reduced genital congestion/reduced lubrication [7], orgasmic disorder after radical vulvectomy [8]
	Disruptbn of genital response from radiation	ED from vascular (and also likely nerve) damage after radiotherapy for prostate cancer [9]; vaginal stenosis and friability from radiation for pelvic cancer [10]
	Dyspareunia and disruptbn of sexual desire and response from chemotherapy	Sudden ovarian failure after chemotherapy for breast cancer [11]; testicular failure after intensive chemotherapy for hematopoietic transplantation [12]
	Disruptbn of sexual desire and response from antiandrogen treatment	GnRH therapy for prostate cancer [13]
	Disruptbn of genital response from aromatase inhibitors	Loss of sexual genital sensitivity, and exacerbation of vaginal atrophy from aromatase inhibition post breast cancer [14]
	Disruptbn of sexual desire and response from pain	Pain from any chronic condition is a potent sexual distraction
	Disruption of sexual desire and response from nonhormonal medications	Narcotics can depress desire through gonadotropin suppressbn [15]; selective serotonin reuptake inhibitors reduce desire and response [16]

Table 17.1 (continued)

Type	Mechanisms	Examples
Indirect	Reduction of self-image	Reduced by disfiguring surgeries, stomas, incontinence, altered appearance (e.g., drooping and altered faces of Parkinson's, altered skin color and muscle wasting of renal failure)
	Depressed mood	Depression and mood lability commonly accompany chronic illness; depression major determinant of sexual function in women with renal failure [17] or multiple sclerosis [18]; strong link between ED and subsequent depression [19]
	Impaired mobility	Reduced ability to caress, hug, and hold a partner; to sexually self-stimulate, to stimulate a partner, to move into positions for intercourse, to pelvically thrust in spinal cord injury, Parkinson's, brain injury, postamputation
	Reduced energy	Fatigue may take its toll on sexuality especially desire, e.g., from renal failure or chemotherapy
	Partnership difficulties	Difficulties finding a partner, dysfunction in the partner who assumes a care giver role, institutionalization, fear of becoming a burden to a partner, lack of independence. Relationship discord from stressors of living with medicalized lives (e.g., three times weekly hemodialysis)
	Sense of loss of sexuality from imposed infertility	From surgery removing gonads or uterus, from chemotherapy or radiotherapy causing gonadal failure
	Fear of sex worsening medical condition	Avoiding sex fearing a further stroke

From Basson et al. [5]
ED erectile dysfunction

general lack of sexual desire to a lack of sexual desire for the current partner, and it may have started after a period of normal sexual functioning or the person may always have had no/low sexual desire. Hypoactive sexual desire disorder has been reported in approximately 30 % of women and 15 % of men in population-based studies and is associated with a wide variety of medical and psychologic causes.

- *Arousal disorders— inability to become physically aroused or excited during sexual activity:* Sexual arousal disorders, including erectile dysfunction in men and sexual arousal disorder in women, are found in 10–20 % (arousal and lubrication disorders are reported in 8–28 % of women) and are strongly age-related in men. They are characterized by a normal desire for sex but a difficulty or inability to become aroused or maintain arousal during sexual activity.
- *Orgasm disorders—delay or absence of orgasm (climax), ejaculation disorders (for male):* Orgasmic disorder is relatively common in women, affecting about 10–25 % in community-based studies. In contrast, premature ejaculation is the most common sexual complaint of men, with a reporting rate of approximately 30 % in most studies. These conditions consist in a persistent or recurrent

difficulty in achieving orgasm after sufficient sexual arousal and ongoing stimulation (anorgasmia or delayed ejaculation).

- On the contrary, premature ejaculation (PE) occurs when a man experiences orgasm and expels semen soon after sexual activity and with minimal penile stimulation. However, there is no uniform cutoff defining “premature,” and values between 15 s and one minute are reported. Men’s typical ejaculatory latency is considered approximately 4–8 min [6].
- Another uncommon ejaculation disorder is retrograde ejaculation, which occurs when semen enters the bladder instead of going out through the urethra during ejaculation. The main reason is an incomplete or ineffective closure of the bladder neck. It can be caused by medications, health conditions, or surgeries that affect the nerves or muscles that control the bladder opening.
- *Pain disorders—pain during intercourse:* Sexual pain disorders have been reported in 10–15 % of women and less than 5 % of men. Dyspareunia (painful intercourse), vaginismus (an involuntary and painful spasm of the muscles of the vaginal wall that interferes with intercourse), and vulvodynia (burning vulvar pain not necessary related to sexual activity) are common sexual pain-related conditions. Dyspareunia may be caused by insufficient lubrication (vaginal dryness) in women. Poor lubrication may result from insufficient excitement and stimulation or from hormonal changes caused by menopause, pregnancy, or breast-feeding. It is unclear exactly what causes vaginismus, but it is thought that past sexual trauma (such as rape or abuse) may play a role.

However, there is considerable overlap between sexual dysfunctions, especially in women [7]. In patients with hypoactive sexual desire disorder, 41 % of women had at least one other sexual dysfunction and 18 % had diagnoses in all three categories (excluding pain disorders) [8].

17.2 Sexual Dysfunction and Pelvic Pain Disorders

Urinary voiding disorders have been strongly associated with sexual dysfunction. In women with urinary incontinence, 60 % had urinary incontinence during sexual intercourse, which significantly impacted their sex life [9]. Similarly, others have reported greater degrees of incontinence correlating with lower scores on the sexual function survey [10, 11].

The landmark National Health and Social Life Survey established a strong association between urinary tract symptoms and arousal disorders (odds ratio 4.2; 95 % confidence interval 2.75–5.89) and sexual pain disorders (odds ratio 7.61; 95 % confidence interval 4.06–14.26) [12]. The prevalence of sexual dysfunction secondary to sexual and pelvic pain ranges from 7 to 58 % [13].

In addition, women with genitourinary hypersensitivity disorders, classified by the International Continence Society as “genitourinary pain syndromes,” account for a large percentage of female patients who present to urogynecologic and sexual medicine practices [14].

Interstitial cystitis/painful bladder syndrome (IC/PBS) is a potential cause of SD and should be considered in the differential diagnosis for dyspareunia. Dyspareunia is estimated to occur in 49–90 % of women with IC/PBS, and urinary and pain symptoms are exacerbated following sexual activity [15].

Using the Female Sexual Distress Scale (FSDS), Peters et al. reported that IC/PBS patients had significantly more dyspareunia (74.6 % vs. 29.9 %), more fear of pain (50.2 % vs. 13.5 %), and significantly less sexual desire and ability to achieve orgasm compared with healthy controls [16].

The prevalence of SD among women affected by laxity disorders of the pelvic floor is estimated to be 25–63 %. An estimated 11 % of these women undergo pelvic surgery for their laxity disorder, and the surgical repair procedure may either enhance or further impair their sexual function [12, 17].

Coital urinary incontinence (CUI) is another significant factor that affects sexual function and may be underdiagnosed in the clinical setting. Unless women are asked directly about its occurrence, they rarely report CUI spontaneously. Bachmann et al. noted that only 3 % of women self-reported sexual disturbances (including CUI); however, the prevalence increased to 20 % after direct questioning in an outpatient setting [18].

Surgical repair of pelvic organ prolapse or stress urinary incontinence, while generally beneficial for a woman's quality of life, can also have negative effects [19]. Depending on the extent of surgical dissection, tissue damage, devascularization, and denervation involved, the result can be decreased vaginal blood flow and increased fibrosis, ultimately resulting in increased dyspareunia.

Sexual function improves in women following pelvic reconstructive surgery, but the improvement is more substantial following anterior repair either alone or in combination with a vaginal hysterectomy when compared with posterior repair [20].

For upper vaginal prolapse (uterine or vault), abdominal sacral colpopexy was associated with a lower rate of recurrent vault prolapse on examination and painful intercourse than with vaginal sacrospinous colpopexy [21].

The association between vaginal mesh and dyspareunia is well documented, especially after posterior repair, and the incidence of dyspareunia was increased associating levator myorrhaphy to posterior vaginal repair, with a de novo dyspareunia rate up to 20 % [22].

Despite the good functional results achieved by expert surgeons, large multi-center studies show that urogenital dysfunction remains a common problem after rectal cancer treatment. More than half of patients experience a deterioration in sexual function, consisting of ejaculatory problems and impotence in men and vaginal dryness and dyspareunia in women. Radiotherapy seems to have a role in the development of sexual dysfunction, without affecting urinary function [23].

Laparoscopic or robotic total mesorectal excision for rectal cancer is associated with significantly less deterioration in sexual function compared with open surgery. This effect is particularly pronounced in women [24].

Prostate cancer by itself reduces sexual desire and the frequency of sexual intercourse. Additionally, surgery or hormonal therapy to block testosterone

further increases the frequency of erectile dysfunction. Erectile dysfunction following radical prostatectomy is primarily attributable to nerve injury caused by intraoperative nerve traction, thermal injury, ischemic injury, and local inflammatory reactions [25].

Women with fecal incontinence were as likely to engage in sexual relations as women without fecal incontinence; however, sexually active women with fecal incontinence had poorer sexual function and reported more dyspareunia, fear, and avoidance of sexual activity with greater partner problems than women without fecal incontinence [26].

17.3 General Treatment Principles

Treatment of sexual dysfunction is complicated by the lack of a single causative factor, limited proven treatment options, physician unfamiliarity with available treatments, overlap of different types of dysfunction, limited availability of treatment, and limited expertise in the treatment [27].

Most types of sexual dysfunction can be corrected by treating the underlying physical or psychological problems. Sexual therapy and education (e.g., cognitive behavior therapy, individual and couple therapy, physiotherapy) form the basis of treatment in most cases. The success of treatment for sexual dysfunction depends on the underlying cause of the problem and the outcome is good for dysfunction that is related to a treatable or reversible physical condition.

Substantial advances have occurred in the understanding of the pathophysiology of erectile dysfunction that led to the development of successful oral therapies, namely, the phosphodiesterase type 5 inhibitors, even if with limitations [28].

With the advent of phosphodiesterase type 5 inhibition as oral therapy, intracavernous injection of vasoactive agents has been relegated to second-line therapy for most patients with erectile dysfunction. However, the future of this category of agents remains attracting and an ever-expanding number and combination of agents are under investigation, making intracavernous injection more appealing as greater efficacy and tolerability and more rapid onset are attained [29].

Developments in the treatment of male erectile dysfunction have led to investigation of pharmacotherapy for the treatment of female sexual dysfunction, even if the benefit of hormonal and nonhormonal drugs is less demonstrated. Testosterone improves sexual function in postmenopausal women with hypoactive sexual desire disorder, although data on its long-term safety and effectiveness are lacking. Estrogen improves dyspareunia associated with vulvovaginal atrophy in postmenopausal women. Phosphodiesterase inhibitors have been shown to have limited benefit in small subsets of women with sexual dysfunction [30].

The myriad of therapeutic modalities for the treatment of female sexual dysfunction are summarized in Table 17.2, underlining the difficulties and the variability of the treatment of these diseases.

Table 17.2 Therapeutic modalities for the treatment of female sexual dysfunction

Behavioral therapy	Pharmacological therapy to bladder	Nonpharmacological therapy to pelvic floor	Therapy to vagina and vulva
Dietary modifications	Pentosan polysulfate sodium	Physical therapy	Lubricants
Stress reduction	Hydroxyzine hydrochloride	Myofascial massage	Estrogen creams
Relationship counseling and sex therapy	Amitriptyline hydrochloride	Thiele massage	Cromolyn
Lifestyle modifications	Cyclosporin A	Pharmacological therapy to pelvic floor	Capsaicin
Planned timing for intercourse	Intravesical bladder therapy/cocktails (dimethyl sulfoxide, heparin, lidocaine, Marcaine, steroids)	Muscle relaxants (Valium suppository)	Lidocaine gel
Pre- or postcoital bathing	Antispasmodics and antimuscarinics	Trigger point injections with local anesthetics and anti-inflammatory medications	Trigger point injections with local anesthetics and anti-inflammatory medications
Changing sexual positions	Botulinum toxin A	Botulinum toxin A	
Noncoital sexual alternatives	Neuromodulation		
Pre- and postcoital ice pack applied to genital and suprapubic areas	Medical: Gabapentin, Pregabalin		
Vaginal dilators	Surgical: Peripheral and sacral nerve stimulation		

From Wehlbe et al. [31]

17.4 Electrotherapies for Sexual Dysfunctions

In this wide field, electrotherapies could have a potential role for treating specific symptoms in specific patients.

The principles of using electrical stimulation of peripheral nerves or nerve roots for restoring useful bladder, bowel, and sexual function after damage or disease of the central nervous system have been extensively reported in literature.

17.4.1 Neuromodulation for Female Sexual Dysfunction

Since the 1990s, sacral neuromodulation (SNM) is used in patients with refractory idiopathic symptoms of overactive bladder or nonobstructive urinary retention and then for fecal incontinence and constipation. During routine follow-up for SNM, a

number of patients spontaneously reported improved sexual functioning in comparison to before their SNM implantation.

The rationale of this result can be highlighted considering the common innervation of the involved structures through the pudendal and the hypogastric nerves.

However, even if the associations between lower urinary tract symptoms (LUTS) and female sexual dysfunction (FSD) are well documented, the question remains as to whether improved sexual function following SNM results from a reduction in LUTS and/or the associated improvement in quality of life (QoL) or if it occurs independently of changes in urinary function.

In the study of Gill et al. [32] in 33 patients affected by overactive bladder (OAB), a statistically significant improvement in urinary and sexual function occurred according to multiple metrics. Female Sexual Function Index (FSFI) and female sexual health questionnaire (FSHQ) were used to assess the sexual status and function of the patients, and also urinary function was assessed using validated instruments. Of the 33 patients only 10 were sexually active, and all the patients were in a menopausal status.

Validated urinary symptom and quality of life scores improved significantly. After treatment, most patients were incontinent less often with sexual activity and felt less restricted from sexual activity by fear of incontinence. Validated quantification of sexual function demonstrated significant improvements in overall sexual function, arousal, and satisfaction. In their results, improved sexual function was not significantly associated with improved urinary function after SNM despite apparent trends between the two.

Opposed to this result, studying a possible correlation between OAB symptoms improvement and sexual function improvement, Signorello et al. [33] analyzed the correlations between differences in Female Sexual Function Index (FSFI) scores and in clinical outcome and correlations between differences in FSFI, short form 36 (SF36), and incontinence quality of life index (IQoL) scores in 16 sexually active patients treated for OAB with SNM (15/16 were in menopause).

A significant correlation was found between clinical improvement and improvement in sexual function. No significant correlation was found between differences in FSFI and quality of life index (IQoL and SF36).

The authors suggest that improvement in the quality of sexual function in female patients with OAB correlates with improvement in urinary symptoms.

Pauls et al. [34] reported improvement in the FSFI score in 11 female patients with an SNM implant for LUTS. Significant improvement with regard to desire, lubrication, orgasm, satisfaction, and pain was found. No increase in score was noted for the arousal domain. Sexual arousal is a response to a sexually attractive stimulus and has both a physiological and a subjective component.

Interestingly, all patients in Pauls' study improved on the FSFI, while only three subjects reported subjective improvement in sexual functioning.

Lombardi et al. [35] presented the results of their study on the effect of SNM on sexuality in 31 female patients. In this study, both patients with idiopathic and with neurological causes for their LUTS were included. Improvement in Female Sexual Distress Scale and FSFI after SNM was found in both patient groups.

Also Yih and colleagues [36] suggest a possible positive role of SNM, observing that sexual function improves along with urinary symptoms after neuromodulation in 167 patients with voiding symptoms evaluated with FSFI and Interstitial Cystitis Symptom-Problem Indices (ICSI-PI). Improved FSFI domains included desire, orgasm, satisfaction, and pain. Of the 74 sexually inactive patients at baseline, 10 became sexually active during the follow-up.

On the contrary Ingber et al. [37] used the FSFI on 54 female patients (27 sexually inactive) affected by OAB or painful bladder syndrome (PBS), who were scheduled to receive a neuromodulation implant. They administered the FSFI before and after 6 months from implantation, and they found no significant improvement in female sexual function.

Both neuromodulation by SNM and by pudendal implant techniques were used in their study.

Pudendal nerve stimulation for SD was also described in the paper of Peters [38], focusing on the technique and according to the original technique described by Spinelli [39].

Also the study of van Voskuilen et al. [40] on 8 patients treated with SNM for urgency symptoms (6 patients), urinary retention (1), and fecal incontinence (1) did not show a clear effect of SNM on sexual function, although there seems to be an improvement in orgasm scores. However, the lack of response on psychological questionnaires (5 questionnaires: Questionnaire for Screening for Sexual Dysfunctions, the Golombok-Rust Inventory of Sexual Satisfaction, the Symptom Checklist-90, the Maudsley Marital Questionnaire, and the McGill-Mah Orgasm Questionnaire) and the increase in vaginal pulse amplitude at plethysmography after SNM implantation could indicate that the improvement seems to be more physiologically than psychologically mediated.

Jarrett et al. [41] administered a self-written sexual questionnaire to patients with fecal incontinence before and after SNM implantation.

Nine of the 16 patients were sexually active. All nine patients reported that their sex life had been affected by fecal incontinence prior to SNM, and seven had felt benefit from implantation. The median improvement in their sex life was 40 %, and the percentage of improvement was inversely correlated to age.

Another study on the effects of SNM for LUTS on female sexual function was carried out by Zahibi et al. [42].

A characteristic of this study was that a large proportion of the patients had pelvic pain as well as LUTS. The study group chose to perform bilateral SNM and to position the electrodes epidurally in the sacral canal, thus stimulating the nerves of S2 and S4 as well as S3. A significant improvement in the FSFI was found. Results were better in patients who underwent the treatment for voiding dysfunction compared to those who had pain as their primary complaint. Domains with no significant improvement were desire and pain.

A concomitant improvement of LUTS and SD was noted even after percutaneous tibial nerve stimulation. Patients most likely to benefit were women, patients with an OAB, and subjective responders. The aspects of sexual life which mostly improved were overall satisfaction, libido, and frequency of sexual activities [43].

In front of these conflicting results, some factors need to be considered.

One of the most obvious causes for a not consistent concordance between the studies is the low number of patients tested. A reason for this might be that many patients with prolonged LUTS or fecal symptoms are not sexually active because of their complaints, and current sexual activity was not always an inclusion criterion. Considering sexually inactive patients in the measurement could be a selection bias.

Moreover, a large proportion of the group of patients who have urinary or fecal symptoms and are considered for SNM therapy are postmenopausal patients, as evident in the described papers.

Postmenopausal women report a relatively high rate of sexual dysfunction (higher than men). There is a marked decline in sexual interest and frequency of sexual activity. Lower estrogen levels after menopause may lead to changes in genital tissues and sexual responsiveness, and in this group considerable comorbidity can be found such as hysterectomy, vascular disease, diabetes, or arthrosis that might also preclude patients from engaging in sexual activity [44].

Previous pelvic surgery could be another influencing factor, as well as the lack of dedicated studies about SNM for SD, with SD as the main indication for the treatment.

There has been one case report on the use of sacral neuromodulation for the treatment of refractory vulvar vestibulitis syndrome.

Sacral neuromodulation was used, and at 6 weeks postoperatively, the patient reported that her pain had decreased from a 10/10 to a 2/10. At 24 months postimplantation, the patient continued to report decreased pain, was able to resume coitus, noted an increase in pain-free days, and a decrease in the intensity of pain during flares [45].

In another case report, a 51-year-old patient that presented with symptoms of lower urinary tract dysfunction and clitoral pain after an abdominal hysterectomy treated with SNM was described.

During test stimulation, she experienced only moderate improvement in voiding symptoms, but a striking improvement in clitoral pain symptoms. She underwent a two-stage implantation of a neurostimulator with a successful outcome after 6 months follow-up [46].

Govaert et al. [47] shows that SNM has an effect on uterus contractility. A decrease in frequency of contractions is seen with the SNM system switched on. However, as the measurements were performed in patients in the resting state, it is not clear what the effect of SNM on the uterus is while the patient has an orgasm.

Lastly, there are case reports on female patients who received lumbar epidural electrical stimulation, who have spontaneous orgasms when the simulator is turned on.

Through standard techniques, quadripolar or octopolar leads were placed in the epidural space percutaneously. The lead was maneuvered initially to an L1–L2 position and then repositioned based on feedback from the patient. The patients were allowed to utilize the device ad libitum for up to 9 days. These women described a greater frequency in sexual activity, increased lubrication, and overall satisfaction. A return of orgasmic capacity was found in 80 % (4/5) of patients

having secondary anorgasmia with an average intensity of $\geq 3/5$ while using the device. Once the device was removed, the patients returned to their previous anorgasmic status [48, 49].

17.4.2 Neuromodulation for Erectile Dysfunction

Anecdotally, most men report improved erectile functioning after neuromodulation for concomitant pelvic floor diseases. Research in monkeys, dogs, and rats has shown that electrical stimulation of the cavernous nerve results in an erection by causing increased arterial flow, relaxation of the cavernous muscles, and venous outflow restriction [50–52].

Shafik [53] implanted a cavernous nerve stimulation device in a series of 15 men for the treatment of erectile dysfunction. Cavernous nerve stimulation at a frequency of 10 Hz led to penile tumescence and an increase in intracavernous pressure but poor rigidity. When the stimulation frequency was increased to 60 Hz, penile tumescence and rigidity and intracavernous pressure increased, and full erection was achieved.

Additionally, Shafik's study demonstrates that unilateral cavernous nerve stimulation is sufficient to induce erection.

Erectile dysfunction is a recognized, common adverse consequence of radical prostatectomy as well as various other pelvic surgeries. While a host of management options have been considered to decrease this complication, neuromodulatory therapy has recently been advanced as an intervention that may be applied for this purpose. Neuromodulatory therapy offers a therapeutic approach for addressing the neuropathic changes of the penis that occurs in this context with the goal of maximally preserving erectile function postoperatively [54].

In 16 men undergoing retropubic radical prostatectomy and in 6 undergoing penile surgery for venous leakage, Lue et al. [55] applied electrical stimulation to the prostatic apex bilaterally (prostatectomy group) or to the hilum of the penis (venous surgery group). Electrical stimulation produced visible erection in 8 of the 16 prostatectomy patients and an increase in intracavernous pressure in 5 of the 6 venous surgery patients.

Burnett et al. [56] explored the feasibility of using an implantable electrode array for cavernous nerve stimulation for patients undergoing nerve-sparing prostatectomy. The implantable electrode array was placed over the neurovascular bundles (20 Hz frequency, 260 μ s pulse width, 5–60 mA amplitude up to 10 min) in 12 patients undergoing open retropubic radical prostatectomy, and penile circumference increases were measured. Six of 12 (50 %) patients demonstrated a significant increase in penile circumference after stimulation.

To determine if intraoperative stimulation of the cavernous nerves while monitoring changes in penile tumescence could be useful to map the course of these nerves and would result in an improvement in nerve sparing and erectile function after radical prostatectomy, Klotz and Herschorn [57] studied 23 patients.

A cavernous nerve stimulator and tumescence-monitoring device was used during radical prostatectomy to identify the course of the cavernous nerves and guide the surgeon in avoiding nerve damage. Nineteen of 21 patients reported erectile function preoperatively. Seventeen (89 %) of 19 patients demonstrated a tumescence response during surgery. Sixteen (94 %) of the 17 patients who demonstrated a response to nerve stimulation and for whom the surgery was guided by the tumescence response reported the ability to have erections after surgery.

The same author confirmed the results in a multicentric prospective, randomized, single-blinded study, performed on 61 patients at 6 centers [58]. Patients had elected to undergo nerve-sparing prostatectomy and had normal preoperative erectile function documented by the Sexual Function Inventory Questionnaire (SFIQ) and RigiScan parallel testing. At 1 year, there was substantial improvement in erectile function in the group in which the procedure was performed assisted by the neurostimulation mapping. This group had a mean of 15.9 min of greater than 60 % nocturnal tumescence compared to 2.1 min in the conventional nerve sparing group ($p < 0.024$).

However, as suggested by Holzbeierlein and colleagues [59], a response to neurovascular bundle stimulation does not necessarily correlate with the precise anatomical location of the cavernous nerves and that a considerable background variability related to anesthesia, surgical manipulation, and other undefined factors that may cause minor but measurable changes in penile circumference and need to be considered.

To evaluate if SNM could improve erectile function, Lombardi et al. [60] studied 22 males that underwent a permanent SNM for LUTS of neurogenic or idiopathic origin. International Index of Erectile Function (IIEF-5) was used. Postoperatively, seven of the 22 showed an improvement in their IIEF-5 scores maintained until the last follow up.

17.4.3 Functional Electrical Stimulation and Rehabilitative Techniques

The sphincteric and supportive functions of the pelvic floor are fairly well understood, and pelvic floor rehabilitation (PFR) has demonstrated effectiveness in the treatment of urinary and fecal incontinence.

However, the role of the pelvic floor in the promotion of optimal sexual function has not been clearly elucidated.

It has been proposed that the pelvic floor muscles are active in both male and female genital arousal and orgasm and that pelvic floor muscle hypotonus may impact negatively on these phases of function. Hypertonus of the pelvic floor is a significant component of sexual pain disorders in women and men. Furthermore, conditions related to pelvic floor dysfunction, such as pelvic pain, pelvic organ prolapse, and lower urinary tract symptoms, are correlated with sexual dysfunction [61].

The role of functional electrical stimulation (FES) in treating LUTS with regard to sexual dysfunction and quality of life has been reported, suggesting a remarkable enhancement in sexual health and satisfaction in all the FSFI domains, particularly evident for the desire, arousal, satisfaction, and orgasm domains [62]. The FES represented an important part of PFR, even if a complete rehabilitation program, including biofeedback, pelvic floor muscular exercises, and vaginal cones, should be considered [63].

The FSFI, administered before and after transvaginal electrical stimulation, showed a significant improvement in desire, lubrication, sexual satisfaction, and pain, whereas arousal and orgasm domains were not significantly affected [64].

The normalization of muscle tonus provided by PFR could be one of the possible explanations of these outcomes. As a result, rehabilitation represents the basis for satisfying orgasmic sensation [65]. In fact, ischiocavernous attachment to the clitoral hood results in clitoral engorgement; the bulbocavernous muscle, when contracted, places pressure on the deep dorsal vein of the clitoris, preventing venous escape.

Additionally, specific rehabilitation programs can improve arousal, reducing the inhibition caused by leakage during orgasm [66].

Women with vulvar pain, dyspareunia, or vaginismus have limited ability to function sexually and often present with musculoskeletal and neurological findings appropriately addressed by a trained physiotherapist [67].

Some benefits of the rehabilitative approach, performed with a portable electromyographic biofeedback instrumentation for daily, at-home, biofeedback-assisted pelvic floor muscle rehabilitation exercises, were reported for the treatment of vulvar vestibulitis syndrome [68].

Confirming these results, in the study of McKay and colleagues [66], 29 patients with moderate to severe vulvar vestibulitis syndrome were analyzed. Each patient was given a computerized electromyographic assessment of pelvic floor muscles and provided with a portable electromyographic home trainer biofeedback device, and specific instructions were given to perform biofeedback-assisted pelvic floor muscle rehabilitation exercises. Fifteen of the 29 treated patients (51.7 %) demonstrated markedly decreased introital tenderness, and 14 of them (93.3 %) were able to resume sexual activity without discomfort. Nine patients (31.0 %) demonstrated a significant decrease in introital tenderness and pain, and six of the nine (66.7 %) resumed sexual activity. Thus, 20 of the 29 women (69 %) became sexually active. Following completion of treatment, 24 (88.9 %) reported negligible or mild pain.

17.4.4 Electroejaculation for Ejaculatory Dysfunction

Ejaculatory dysfunction is an uncommon cause of male infertility; however, in some groups, such as patients with spinal cord injury, anejaculation is very prevalent and is the major cause of infertility [69]. Other causes of anejaculatory infertility include retroperitoneal lymph node dissection, diabetic neuropathy, multiple sclerosis, transverse myelitis, and psychogenic anejaculation.

Regaining sexual function is the highest priority among paraplegics [70]. However, only 10 % of men with spinal cord injury (SCI) can father children without medical assistance, owing to potential impairments in erection, ejaculation, and semen quality [71].

Before the 1980s, the options for retrieving sperm from men with SCI were limited, owing to a lack of safe, consistent, and effective methodologies. Electroejaculation (EEJ) was first described in humans by Learmonth in 1931 [72]. Horne et al. [73] in 1948 reported the first use of EEJ in SCI persons, resulting in successful ejaculation in nine of 15 men. In the 1980s, the procedure of EEJ was commercialized for semen retrieval in humans [74]. Adapted from devices used in veterinary medicine, EEJ came into wide use for retrieval of sperm from anejaculatory men, the majority of whom were men with SCI [75–77].

Electroejaculation is carried out with an electrical probe, which is inserted rectally and is positioned with the electrodes in contact with the anterior rectal wall in the area of the prostate gland and the seminal vesicles. The electrical stimulation is administered in a wave-like pattern with voltage progressively increasing in 1–2 V increments until ejaculation occurs.

Antegrade ejaculation is not produced in a projectile fashion but rather as an intermittent release of semen during the course of the procedure. Between 15 and 35 stimulations are usually needed to ensure emptying of the semen. The voltage and current that have been reported to successfully produce ejaculation range from 5 to 25 V and 100–600 mA, respectively [78].

In the 1990s, the method of penile vibratory stimulation (PVS) became the method of first choice for semen retrieval in men with spinal cord injuries (SCIs). Because vibratory stimulation is very simple in use, less expensive, and noninvasive, it does not require anesthesia and is preferred by the patients when compared with EEJ; PVS is recommended to be the first choice of treatment in spinal cord injured men [79, 80].

The majority of spinal cord injured men are not able to produce antegrade ejaculation by masturbation or sexual stimulation. However, approximately 80 % of all spinal cord injured men with an intact ejaculatory reflex arc (above T10) can obtain antegrade ejaculation with PVS. Electroejaculation may be successful in obtaining ejaculate from men with all types of SCI, including men who do not have major components of the ejaculatory reflex arc. Furthermore, EEJ has been successfully used to induce ejaculation in men with multiple sclerosis and diabetic neuropathy. Any other conditions which affect the ejaculatory mechanism of the central and/or peripheral nervous system including surgical nerve injury may be treated successfully with EEJ. Finally, for sperm retrieval and sperm cryopreservation before intensive anticancer therapy in pubertal boys, PVS and EEJ have been successfully performed in patients who failed to obtain ejaculation by masturbation [78].

Confirming these indications, Kafetsoulis and colleagues [81] administered a survey to professionals for the evaluation of the current treatment methods for infertility in couples with SCI male partners.

Because EEJ was reserved as a second-line of treatment, it was performed only after PVS failures. Electroejaculation was performed 845 times on a total of 185

men with SCI whose level of injury ranged between C3 and L3 (4.6 procedures per patient). Of the 185 men, 175 (95 %) could ejaculate with EEJ. Of the 845 EEJ procedures, 95 % resulted in ejaculation. Of the 5 % of men who did not ejaculate with EEJ, all were patients with retained pelvic sensation who experienced pain at low voltages (1–4 V) on their first trial of EEJ and did not want to continue with further trials of EEJ under sedation or general anesthesia.

The most common reasons cited by respondents for not offering EEJ was a lack of EEJ equipment (60 % of respondents) and a lack of training in EEJ (42 % of respondents).

Sperm retrieved by EEJ is characterized by abnormal sperm motility (asthenospermia) and normal count. This was the case in patients with spinal cord injuries as well as those who suffered from psychogenic anejaculation. The asthenozoospermia may be related to increased scrotal temperature, urinary infection, stasis of seminal fluid, neural effects on physiology of the testis and epididymis, sperm autoimmunity, some factors in the seminal plasma, disordered storage of spermatozoa in the seminal vesicles, and external testicular pressure effects of the “closed-leg” position [82–84].

However, the concentration and the motility of sperm obtained by electroejaculation were not significantly different from sperm obtained naturally, suggesting a disease-related alteration rather than alterations caused by the procedure [85].

Several successful pregnancies have been reported using spermatozoa obtained by PVS or EEJ combined with assisted reproduction techniques such as intrauterine insemination or in vitro fertilization with or without intracytoplasmic sperm injection.

Ohl et al. [86] published a large study in which several aspects of EEJ in combination with assisted reproductive technology in the treatment of anejaculatory infertility were investigated. They studied 121 consecutive couples, in which 87 male partners had SCI. Intrauterine insemination was the route of sperm delivery in all insemination cycles. For those couples that did not conceive within 3–6 cycles of intrauterine insemination (IUI), gamete intrafallopian transfer (GIFT) or in vitro fertilization (IVF) procedures were recommended. Among couples with an SCI male partner, in 479 completed cycles of EEJ with IUI, 41 pregnancies were obtained. This represents an 8.6 % pregnancy rate (PR) per cycle and 32.2 % PR per couple.

Chung et al. [87] reported their experience in EEJ combined with IUI and IVF-ET. A group of 26 men participated, 23 SCI patients and 3 patients who had retroperitoneal lymph node dissection for testicular cancer. Female partners received 50 mg/day (days 3–7) clomiphene citrate during IUI cycles to improve PRs. Electroejaculation was performed on the day of insemination, and both antegrade and retrograde specimens were processed by swim-up technique. A total of 50 IUIs were performed in 10 couples, resulting in 5 pregnancies in 3 couples, with 2 couples conceiving twice. This constitutes a PR of 10 % per IUI and 30 % per couple.

In spite of the lower fertilization rate in psychogenic patients, combination of EEJ and ICSI gives adequate results to couples with psychogenic anejaculation similar to the results obtained for SCI patients [88].

The overall pregnancy rate per cycle from those studies averages about 25 %. It should be noted that this rate is similar to the pregnancy rate per cycle during natural procreation in healthy couples wanting to become pregnant (25–30 %) [89], although assisted ejaculation procedures and reproduction techniques are required for SCI men and their partners. If assisted ejaculation procedures fail or yield insufficient motile or viable spermatozoa for assisted reproductive techniques, surgical procedures of sperm retrieval are indicated.

17.4.5 Acupuncture for Sexual Dysfunction

Although new pharmaceutical agents have been identified for male erectile problems, sexual desire, and orgasm disorders, individuals with sexual dysfunction often seek alternative therapies, including traditional Chinese medicine.

Acupuncture is an ancient Chinese healing method. Despite its existence for many centuries, many Western physicians still tend to dismiss its efficacy by arguing that this is caused by a placebo effect. However, such an argument is no longer in line with current neuroscientific knowledge of the underlying mechanisms of actions of acupuncture.

It has been shown that manipulation of strictly defined energetic trigger points results in potent sensory stimulation that produces various changes in the central and peripheral nervous system.

Interestingly, various studies have shown that acupuncture facilitates the release of endogenous opioids in the central nervous system and that several classes of molecules, such as neurotransmitters, cytokines, and growth factors, are possible mediators for specific acupuncture effects [90]. Particularly interesting is the effect of acupuncture in the release of β -endorphin, an endogenous opioid that influences a variety of hypothalamic and autonomic functions and involved in the regulation of pain perception, stress response, mood, and immune functions [91].

Moreover, acupuncture has been reported to alleviate the sensory discriminative aspect and affective component of pain [92]. Physiologically, the afferent stimulation of acupuncture has been attributed to its effect on A- δ and C-fiber sensory nerve fibers and activation of descending pain-inhibiting pathways. As these nerve fibers are sensitive to the light touch of mechanoreceptors in the skin, further research of acupuncture may perhaps lead to better insight into the still unanswered question of why some men only ejaculate rapidly intravaginally, whereas their ejaculation time is seemingly undisturbed during masturbation. Interestingly, neuroimaging studies in humans have validated that acupuncture modulates a widely distributed network of brain regions involved in pain perception and have shown that the amygdala, insula, and hypothalamus modulation may demonstrate some acupuncture specificity [93].

Acupuncture therapy has been used by many researchers both in male and female sexual dysfunction; however, the results are conflicting. Emerging research is establishing that acupuncture may be an effective treatment modality for sexual dysfunction including impotence, loss of libido, and inability to orgasm. Moreover,

acupuncture reduces inflammation, increases sperm motility, improves semen parameters, modulates the immune system, and improves sexual and ejaculatory dysfunction in male infertility [94].

Khamba et al. [95] shown a significant improvement in all areas of sexual functioning as well as in both anxiety and depressive symptoms among male affected by SD secondary to antidepressants (including selective serotonin reuptake inhibitors (SSRIs) and serotonin noradrenaline reuptake inhibitors (SNRIs)). Female patients reported a significant improvement in libido and lubrication.

Aydin et al. [96] examined the effects of acupuncture and hypnotic suggestions, compared with placebo, in the treatment of male sexual dysfunction. Men who received placebo had a 43–47 % improvement in sexual function. The success rates of acupuncture and hypnotic suggestions were 60 and 75 %, respectively.

Although the improvement was not statistically significant compared to the placebo, the authors suggested that treatment with acupuncture could be used as an adjuvant therapy in nonorganic male sexual dysfunction.

Kho et al. [97] investigated the use of acupuncture in erectile dysfunction (ED) and found that acupuncture did not influence the profile of the stress and sex hormones but did improve the quality of erection and restored the sexual activity in some patients, with an overall effect of 39 %. An improvement of the quality of erection was experienced by 15 % of patients, while 31 % reported an increase in their sexual activity.

In contrast, Yaman et al. [98] reported a success rate of acupuncture of 69 % in 29 patients, all patients being diagnosed with psychogenic erectile dysfunction.

However, in the review by Lee et al. [99] that analyzed the four studies of acupuncture therapy for ED, one randomized controlled trial showed beneficial effects of acupuncture compared with sham acupuncture in terms of response rate [100], while another found no effects of acupuncture and the remaining two studies were uncontrolled clinical trials. The authors concluded that the evidence was insufficient to suggest that acupuncture is an effective intervention for treating ED.

Few other papers which described the effect of acupuncture therapy on male sexual dysfunction were focused on premature ejaculation (PE).

Chen [101] compared therapeutic effects of acupuncture and medication (oral Sildenafil 20 mg/day) on primary, simple PE and found the total effective rates were 82.1 % in the acupuncture group and 63.6 % in the medication group.

Sunay [102] compared acupuncture with paroxetine (20 mg/day) and placebo. Median scores of paroxetine, acupuncture, and placebo groups were 17.0, 16.0, and 15.5 before treatment, and 10.5, 11.0, and 16.0 after treatment, respectively ($p=0.001$, $p=0.001$, and $p=0.314$). Increases of intravaginal ejaculation latency times with paroxetine, acupuncture, and placebo acupuncture were 82.7, 65.7, and 33.1 s, respectively. Extent of ejaculation delay induced by paroxetine was significantly higher than that of acupuncture ($p=0.001$). The authors concluded that although less effective than daily paroxetine, acupuncture had a significant stronger ejaculation-delaying effect than placebo.

The authors hypothesized that the observed effectiveness of this revolutionary approach to PE could be due to a central effect of acupuncture on neurotransmitters such as serotonin or endorphins, which are frequently involved in sexual behavior.

However, other authors [103] suggested a possible role of a peripheral reflex. This reflex is mediated by a spinal control center, referred to as the spinal ejaculation generator (SEG). The SEG coordinates sympathetic, parasympathetic, and motor outflow to induce the two phases of ejaculation: emission and expulsion. In addition, the SEG integrates this outflow with inputs conveying biochemical or mechanical information from the accessory sex organs, producing sequential contractions of the epididymis, vas deferens, seminal vesicles, and prostate [104].

Some positive results of acupuncture were found also in the treatment of functional retrograde ejaculation [105, 106] and vestibulodynia.

Curran et al. [107] studied 8 women with vestibulodynia. A significant decrease in pain with manual genital stimulation and helplessness was found. A strong (though nonsignificant) effect for improved ability to have intercourse and sexual desire was also noted, as an improvement in perceived sexual health, reduced pain, and improved mental well-being in the majority of participants.

In the series of Powell and Wojnarowska [108], 12 patients who had not responded to conventional treatment were studied. Two patients felt so much improved that they declared themselves “cured”; three believed their symptoms had improved and wished to continue acupuncture; four felt slightly better and judged acupuncture more effective than any other treatment; and three noted no effect at all. A large part of its beneficial effect, as recognized by the authors, may come from the regular specialist contact.

According to these results, the guidelines for the management of vulvodynia of the British Society for the Study of Vulval Diseases recommended that acupuncture may be considered in the treatment of unprovoked vulvodynia (grade of recommendation C; level of evidence IIb) [109].

References

1. Masters WH, Johnson VE (1966) Human sexual response. Little, Brown, Oxford
2. Rosen RC (2000) Prevalence and risk factors of sexual dysfunction in men and women. *Curr Psychiatry Rep* 2:189–195
3. Lewis RW, Fugl-Meyer KS, Carona G, Hayes RD, Laumann EO, Moreira ED Jr, Rellini AH, Segraves T (2010) Definitions/epidemiology/risk factors for sexual factors for sexual dysfunction. *J Sex Med* 7:1598–1607
4. Laumann EO, Nicolosi A, Glasser DB, Paik A, Gingell C, Moreira E, Wang T, GSSAB Investigators' Group (2005) Sexual problems among women and men aged 40–80 y: prevalence and correlates identified in the Global Study of Sexual Attitudes and Behaviors. *Int J Impot Res* 17:39–57
5. Basson R, Rees P, Wang R, Montejo AL, Incrocci L (2010) Sexual function in chronic illness. *J Sex Med* 7:374–388
6. Strassberg DS, Perelman MA (2009) Sexual dysfunctions. In: Blaney PH, Millon T (eds) Oxford textbook of psychopathology, 2nd edn. Oxford University Press, New York, pp 399–430
7. Balon R, Segraves RT, Clayton A (2007) Issues for DSM-V: sexual dysfunction, disorder, or variation along normal distribution: toward rethinking DSM criteria of sexual dysfunctions. *Am J Psychiatry* 164:198–200

8. Segraves KB, Segraves RT (1991) Hypoactive sexual desire disorder: prevalence and comorbidity in 906 subjects. *J Sex Marital Ther* 17:55–58
9. Jha S, Strelley K, Radle S (2012) Incontinence during intercourse: myths unravelled. *Int Urogynecol J* 23:633–637
10. Aslan G, Koseog̃lu H, Sadik O, Gimen S, Cihan A, Esen A (2005) Sexual function in women with urinary incontinence. *Int J Impot Res* 17:248–251
11. Salonia A, Zanni G, Nappi RE, Briganti A, Deho F, Fabbri F, Colombo R, Guazzoni G, Di Girolamo V, Rigatti P, Montorsi F (2004) Sexual dysfunction is common in women with lower urinary tract symptoms and urinary incontinence: results of a cross-sectional study. *Eur Urol* 45:642–648
12. Laumann EO, Paik A, Rosen RC (1999) Sexual dysfunction in the United States: prevalence and predictors. *JAMA* 281:537–544
13. Hayes RD, Bennett CM, Fairley CK, Dennerstein L (2006) What can prevalence studies tell us about female sexual difficulty and dysfunction? *J Sex Med* 3:589–595
14. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, van Kerrebroeck P, Victor A, Wein A (2002) The standardization of terminology in lower urinary tract function. Report from the Standardization Sub-committee of the International Continence Society. *Am J Obstet Gynecol* 187:116–126
15. Butrick CW (2003) Interstitial cystitis and chronic pelvic pain: new insights in neuropathology, diagnosis, and treatment. *Clin Obstet Gynecol* 46:811–823
16. Peters KM, Killinger KA, Carrico DJ, Ibrahim IA, Diokno AC, Graziottin A (2007) Sexual function and sexual distress in women with interstitial cystitis: a case-control study. *Urology* 70:543–547
17. Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL (1997) Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. *Obstet Gynecol* 89:501–506
18. Bachmann GA, Leiblun SR, Grill J (1989) Brief sexual inquiry in gynecologic practice. *Obstet Gynecol* 73:425–427
19. Pauls RN (2010) Impact of gynecological surgery on female sexual function. *Int J Impot Res* 22:105–114
20. Dua A, Jha S, Farkas A, Radley S (2012) The effect of prolapse repair on sexual function in women. *J Sex Med* 9:1459–1465
21. Maher C, Feiner B, Baessler K, Schmid C (2013) Surgical management of pelvic organ prolapse in women. *Cochrane Database Syst Rev* 4, CD004014
22. Cundiff GW, Fenner D (2004) Evaluation and treatment of women with rectocele: focus on associated defecatory and sexual dysfunction. *Obstet Gynecol* 104:1403–1421
23. Lange MM, van de Velde CJ (2011) Urinary and sexual dysfunction after rectal cancer treatment. *Nat Rev Urol* 8:51–57
24. McGlone ER, Khan O, Flashman K, Khan J, Parvaiz A (2012) Urogenital function following laparoscopic and open rectal cancer resection: a comparative study. *Surg Endosc* 26:2559–2565
25. Hyun JS (2012) Prostate cancer and sexual function. *World J Mens Health* 30:99–107
26. Cichowski SB, Komesu YM, Dunivan GC, Rogers RG (2013) The association between fecal incontinence and sexual activity and function in women attending a tertiary referral center. *Int Urogynecol J* 24:1489–1494
27. Bachmann G (2006) Female sexuality and sexual dysfunction: are we stuck on the learning curve? *J Sex Med* 3:639–645
28. Shamloul R, Ghanem H (2013) Erectile dysfunction. *Lancet* 381:153–165
29. Bella AJ, Brock GB (2004) Intracavernous pharmacotherapy for erectile dysfunction. *Endocrine* 23:149–155
30. Frank JE, Mistretta P, Will J (2008) Diagnosis and treatment of female sexual dysfunction. *Am Fam Physician* 77:635–642
31. Wehbe SA, Whitmore K, Kellogg-Spadt S (2010) Urogenital complaints and female sexual dysfunction (part 1). *J Sex Med* 7:1704–1713

32. Gill BC, Swartz MA, Firoozi F, Rackley RR, Moore CK, Goldman HB, Vasavada SP (2011) Improved sexual and urinary function in women with sacral nerve stimulation. *Neuromodulation* 14:436–443
33. Signorello D, Seitz CC, Berner L, Trenti E, Martini T, Galantini A, Lusuardi L, Lodde M, Pycha A (2011) Impact of sacral neuromodulation on female sexual function and his correlation with clinical outcome and quality of life indexes: a monocentric experience. *J Sex Med* 8:1147–1155
34. Pauls RN, Marinkovic SP, Silva WA, Rooney CM, Kleeman SD, Karram MM (2007) Effects of sacral neuromodulation on female sexual function. *Int Urogynecol J Pelvic Floor Dysfunct* 18:391–395
35. Lombardi G, Mondaini N, Macchiarella A, Cilotti A, Del Popolo G (2008) Clinical female sexual outcome after sacral neuromodulation implant for lower urinary tract symptom (LUTS). *J Sex Med* 5:1411–1417
36. Yih JM, Killinger KA, Boura JA, Peters KM (2013) Changes in sexual functioning in women after neuromodulation for voiding dysfunction. *J Sex Med* 10:2477–2483
37. Ingber MS, Ibrahim IA, Killinger KA, Diokno AC, Peters KM (2009) Neuromodulation and female sexual function: does treatment for refractory voiding symptoms have an added benefit? *Int Urogynecol J Pelvic Floor Dysfunct* 20:1055–1059
38. Peters KM (2013) Pudendal neuromodulation for sexual dysfunction. *J Sex Med* 10:908–911
39. Spinelli M, Malaguti S, Giardiello G, Lazzeri M, Tarantola J, Van Den Hombergh U (2005) A new minimally invasive procedure for pudendal nerve stimulation to treat neurogenic bladder: description of the method and preliminary data. *Neurourol Urodyn* 24:305–309
40. van Voskuilen AC, Oerlemans DJ, Gielen N, Lansen-Koch SM, Weil EH, van Lankveld JJ, van den Hombergh U, Baeten CG, van Kerrebroeck PE (2012) Sexual response in patients treated with sacral neuromodulation for lower urinary tract symptoms or fecal incontinence. *Urol Int* 88:423–430
41. Jarrett ME, Nicholls RJ, Kamm MA (2005) Effect of sacral neuromodulation for faecal incontinence on sexual activity. *Colorectal Dis* 7:523–525
42. Zabihi N, Mourtzinou A, Maher MG, Raz S, Rodríguez LV (2008) The effects of bilateral caudal epidural S2–4 neuromodulation on female sexual function. *Int Urogynecol J Pelvic Floor Dysfunct* 19:697–700
43. van Balken MR, Vergunst H, Bemelmans BL (2006) Sexual functioning in patients with lower urinary tract dysfunction improves after percutaneous tibial nerve stimulation. *Int J Impot Res* 18:470–475
44. Dennerstein L, Hayes RD (2005) Confronting the challenges: epidemiological study of female sexual dysfunction and the menopause. *J Sex Med* 2(Suppl 3):118–132
45. Ramsey LB, Wright J, Fischer JR (2009) Sacral neuromodulation in the treatment of vulvar vestibulitis syndrome. *Obstet Gynecol* 114:487–489
46. Marcelissen T, Van Kerrebroeck P, de Wachter S (2010) Sacral neuromodulation as a treatment for neuropathic clitoral pain after abdominal hysterectomy. *Int Urogynecol J* 21:1305–1307
47. Govaert B, Melenhorst J, Link G, Hoogland H, van Gemert W, Baeten C (2010) The effect of sacral nerve stimulation on uterine activity: a pilot study. *Colorectal Dis* 12:448–451
48. Meloy S (2007) Neurally augmented sexual function. *Acta Neurochir Suppl* 97(pt 1):359–363
49. Meloy TS, Southern JP (2006) Neurally augmented sexual function in human females: a preliminary investigation. *Neuromodulation* 9:34–40
50. Lue TF, Schmidt RA, Tanagho EA (1985) Electrostimulation and penile erection. *Urol Int* 40:60–64
51. Lin SN, Wang JM, Ma CP, Chen HI (1985) Hemodynamic study of penile erection in dogs. *Eur Urol* 11:401–405
52. Quinlan DM, Nelson RJ, Partin AW, Mostwin JL, Walsh PC (1989) The rat as a model for the study of penile erection. *J Urol* 141:656–661
53. Shafik A (1996) Extrapelvic cavernous nerve stimulation in erectile dysfunction. *Andrologia* 28:151–156

54. Burnett AL, Lue TF (2006) Neuromodulatory therapy to improve erectile function recovery outcomes after pelvic surgery. *J Urol* 176:882–887
55. Lue TF, Gleason CA, Brock GB, Carroll PR, Tanagho EA (1995) Intraoperative electrostimulation of the cavernous nerve: technique, results and limitations. *J Urol* 154:1426–1428
56. Burnett AL, Teloken PE, Briganti A, Whitehurst T, Montorsi F (2008) Intraoperative assessment of an implantable electrode array for cavernous nerve stimulation. *J Sex Med* 5:1949–1954
57. Klotz L, Herschorn S (1998) Early experience with intraoperative cavernous nerve stimulation with penile tumescence monitoring to improve nerve sparing during radical prostatectomy. *Urology* 52:537–542
58. Klotz L, Heaton J, Jewett M, Chin J, Fleshner N, Goldenberg L, Gleave M (2000) A randomized phase 3 study of intraoperative cavernous nerve stimulation with penile tumescence monitoring to improve nerve sparing during radical prostatectomy. *J Urol* 164:1573–1578
59. Holzbeierlein J, Peterson M, Smith JAJR (2001) Variability of results of cavernous nerve stimulation during radical prostatectomy. *J Urol* 165:108–110
60. Lombardi G, Mondaini N, Giubilei G, Macchiarella A, Lecconi F, Del Popolo G (2008) Sacral neuromodulation for lower urinary tract dysfunction and impact on erectile function. *J Sex Med* 5:2135–2140
61. Rosenbaum TY (2007) Pelvic floor involvement in male and female sexual dysfunction and the role of pelvic floor rehabilitation in treatment: a literature review. *J Sex Med* 4:4–13
62. Rivalta M, Sighinolfi MC, Micali S, De Stefani S, Bianchi G (2010) Sexual function and quality of life in women with urinary incontinence treated by a complete pelvic floor rehabilitation program (biofeedback, functional electrical stimulation, pelvic floor muscles exercises, and vaginal cones). *J Sex Med* 7:1200–1208
63. Rivalta M, Sighinolfi MC, De Stefani S, Micali S, Mofferdin A, Grande M, Bianchi G (2009) Biofeedback, electrical stimulation, pelvic floor muscle exercises, and vaginal cones: a combined rehabilitative approach for sexual dysfunction associated with urinary incontinence. *J Sex Med* 6:1674–1677
64. Paradiso Galatioto G, Pace G, Vicentini C (2007) Sexual function in women with urinary incontinence treated by pelvic floor transvaginal electrical stimulation. *J Sex Med* 4:702–707
65. Rosenbaum TY, Owens A (2008) The role of pelvic floor physical therapy in the treatment of pelvic and genital pain related sexual dysfunction. *J Sex Med* 5:513–523
66. McKay E, Kaufman RH, Doctor U, Berkova Z, Blazer H (2001) Treating vulvar vestibulitis with EMG BFB of pelvic floor musculature. *J Reprod Med* 46:337–342
67. Rosenbaum TY (2005) Physiotherapy treatment of sexual pain disorders. *J Sex Marital Ther* 31:329–340
68. Glazer HI, Rodke G, Swencionis C, Hertz R, Young AW (1995) Treatment of vulvar vestibulitis syndrome with electromyographic biofeedback of pelvic floor musculature. *J Reprod Med* 40:283–290
69. Sønksen J, Biering-Sørensen F (1992) Fertility in men with spinal cord or cauda equina lesions. *Semin Neurol* 12:106–114
70. Anderson KD (2004) Targeting recovery: priorities of the spinal cord-injured population. *J Neurotrauma* 21:1371–1383
71. Elliott S (2003) Sexual dysfunction and infertility in men with spinal cord disorders. In: Lin V (ed) *Spinal cord medicine: principles and practice*. Demos Medical Publishing, New York, pp 349–365
72. Learmonth JR (1931) A contribution to the neurophysiology of the urinary bladder in man. *Brain* 54:147–176
73. Horne HW, Paull DP, Munro D (1948) Fertility studies in the human male with traumatic injuries of the spinal cord and cauda equina. *N Engl J Med* 239:959–961
74. Halstead LS, VerVoort S, Seager SW (1987) Rectal probe electrostimulation in the treatment of anejaculatory spinal cord injured men. *Paraplegia* 25:120–129
75. Bennett CJ, Seager SW, Vasher EA, McGuire EJ (1988) Sexual dysfunction and electroejaculation in men with spinal cord injury: review. *J Urol* 139:453–457

76. Martin DE, Warner H, Crenshaw TL, Crenshaw RT, Shapiro CE, Perakash I (1983) Initiation of erection and semen release by rectal probe electrostimulation (RPE). *J Urol* 129:637–642
77. Buch JP, Zorn BH (1993) Evaluation and treatment of infertility in spinal cord injured men through rectal probe electroejaculation. *J Urol* 149:1350–1354
78. Sønksen J, Ohl DA (2002) Penile vibratory stimulation and electroejaculation in the treatment of ejaculatory dysfunction. *Int J Androl* 25:324–332
79. Nehra A, Werner M, Bastuba M, Title C, Oates R (1996) Vibratory stimulation and rectal probe electroejaculation as therapy for patients with spinal cord injury: semen parameters and pregnancy rates. *J Urol* 155:554–559
80. Ohl DA, Sonksen J, Menge AC, McCabe M, Keller LM (1997) Electroejaculation versus vibratory stimulation in spinal cord injured men: sperm quality and patient preference. *J Urol* 157:2147–2149
81. Kafetsoulis A, Brackett NL, Ibrahim E, Attia GR, Lynne CM (2006) Current trends in the treatment of infertility in men with spinal cord injury. *Fertil Steril* 86:781–789
82. Chung PH, Yeko TR, Mayer JC, Sanford EJ, Maroulis GB (1995) Assisted fertility using electroejaculation in men with spinal cord injury—a review of the literature. *Fertil Steril* 64:1–9
83. Hovav Y, Shotland Y, Yaffe H, Almagor M (1996) Electroejaculation and assisted fertility in men with psychogenic anejaculation. *Fertil Steril* 66:620–623
84. Brackett NL, Davi RC, Padron OF, Lynne CM (1996) Seminal plasma of spinal cord injured men inhibits sperm motility of normal men. *J Urol* 155:1632–1635
85. Hovav Y, Almagor M, Yaffe H (2002) Comparison of semen quality obtained by electroejaculation and spontaneous ejaculation in men suffering from ejaculation disorder. *Hum Reprod* 17:3170–3172
86. Ohl DA, Wolf LJ, Menge AC, Christman GM, Hurd WW, Ansbacher R, Smith YR, Randolph JF Jr (2001) Electroejaculation and assisted reproductive technologies in the treatment of anejaculatory infertility. *Fertil Steril* 76:1249–1255
87. Chung PH, Verkauf BS, Eichberg RD, Casady L, Sanford EJ, Maroulis GB (1996) Electroejaculation and assisted reproductive techniques for anejaculatory infertility. *Obstet Gynecol* 87:22–26
88. Gat I, Maman E, Yerushalmi G, Baum M, Dor J, Raviv G, Madjar I, Hourvitz A (2012) Electroejaculation combined with intracytoplasmic sperm injection in patients with psychogenic anejaculation yields comparable results to patients with spinal cord injuries. *Fertil Steril* 97:1056–1060
89. Spira A (1986) Epidemiology of human reproduction. Mini review. *Hum Reprod* 1:111–115
90. Manni L, Albanesi M, Guaragna M, Paparo SB, Aloe L (2010) Neurotrophins and acupuncture. *Auton Neurosci* 157:9–17
91. Kaptchuk TJ (2002) Acupuncture: theory, efficacy, and practice. *Ann Intern Med* 136:374–383
92. Thomas M, Lundberg T (1994) Importance of modes of acupuncture in the treatment of chronic nociceptive low back pain. *Acta Anaesthesiol Scand* 38:63–69
93. Wu MT, Sheen JM, Chuang KH, Yang P, Chin SL, Tsai CY, Chen CJ, Liao JR, Lai PH, Chu KA, Pan HB, Yang CF (2002) Neuronal specificity of acupuncture response: a fMRI study with electroacupuncture. *Neuroimage* 16:1028–1037
94. Hu M, Zhang Y, Ma H, Ng EH, Wu XK (2013) Eastern medicine approaches to male infertility. *Semin Reprod Med* 31:301–310
95. Khamba B, Aucoin M, Lytle M, Vermani M, Maldonado A, Iorio C, Cameron C, Tsigielis D, D'Ambrosio C, Anand L, Katzman MA (2013) Efficacy of acupuncture treatment of sexual dysfunction secondary to antidepressants. *J Altern Complement Med* 19:862–869
96. Aydin S, Ercan M, Caşkurlu T, Taşçı AI, Karaman I, Odabaş O, Yılmaz Y, Ağargün MY, Kara H, Sevin G (1997) Acupuncture and hypnotic suggestions in the treatment of non-organic male sexual dysfunction. *Scand J Urol Nephrol* 31:271–274
97. Kho HG, Sweep CG, Chen X, Rabsztyrn PR, Meuleman EJ (1999) The use of acupuncture in the treatment of erectile dysfunction. *Int J Impot Res* 11:41–46

98. Yaman LS, Kilic S, Sarica K, Bayar M, Saygin B (1994) The place of acupuncture in the management of psychogenic impotence. *Eur Urol* 26:52–55
99. Lee MS, Shin BC, Ernst E (2009) Acupuncture for treating erectile dysfunction: a systematic review. *BJU Int* 104:366–370
100. Engelhardt PF, Daha LK, Zils T, Simak R, König K, Pflüger H (2003) Acupuncture in the treatment of psychogenic erectile dysfunction: first results of a prospective randomized placebo-controlled study. *Int J Impot Res* 15:343–346
101. Chen ZX (2009) Control study on acupuncture and medication for treatment of primary simple premature ejaculation. *Zhongguo Zhen Jiu* 29:13–15
102. Sunay D, Sunay M, Aydogmus Y, Bagbanci S, Arslan H, Karabulut A, Emir L (2011) Acupuncture versus paroxetine for the treatment of premature ejaculation: a randomized, placebo-controlled clinical trial. *Eur Urol* 59:765–771
103. Jannini EA, Lenzi A (2011) Sexual dysfunction: is acupuncture a therapeutic option for premature ejaculation? *Nat Rev Urol* 8:235–236
104. Abdel-Hamid IA, Jannini EA, Andersson KE (2009) Premature ejaculation: focus on therapeutic targets. *Expert Opin Ther Targets* 13:175–193
105. Xiao Y (2002) Treatment of functional retrograde ejaculation with acupuncture and TCM herbal drugs. *J Tradit Chin Med* 22:286–287
106. Chen Y (1993) Acupuncture treatment of functional non-ejaculation: a report of 70 cases. *J Tradit Chin Med* 13:10–12
107. Curran S, Brotto LA, Fisher H, Knudson G, Cohen T (2010) The ACTIV study: acupuncture treatment in provoked vestibulodynia. *J Sex Med* 7:981–995
108. Powell J, Wojnarowska F (1999) Acupuncture for vulvodynia. *J R Soc Med* 92:579–581
109. Nunn D, Mandal D, Byrne M, McLelland J, Rani R, Cullimore J, Bansal D, Brackenbury F, Kirtschig G, Wier M, British Society for the Study of Vulval Disease (BSSVD) Guideline Group (2010) Guidelines for the management of vulvodynia. *Br J Dermatol* 162:1180–1185