Neural Control and Physiology of Sexual Function: Effect of Spinal Cord Injury

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Objective: To present the current understanding of normal anatomy, physiology, sexual physiology, pathophysiology and the consequential sexual changes and dysfunctions following a spinal cord injury (SCI). **Methods:** Narrative review of the latest literature. **Results:** Peripheral innervations of the pelvis involve 3 sets of efferent neurons coordinated through the pelvic plexus (somatic, thoracolumbar sympathetic, and sacral parasympathetic), and these are under cerebral descending excitatory and inhibitory control. SCI, depending on the level of lesion and completeness, can alter this cerebral control, affecting the psychological and reflexogenic potential for genital arousal and also ejaculation and orgasm. During arousal, nitric oxide is the main neurotransmitter for smooth muscle relaxation in both male and female erectile tissue. In men, erection, ejaculation, and orgasm are under separate neurological control and can be individually affected by SCI. **Conclusions:** Since sexual function is rated amongst the highest priorities by individuals living with SCI, methods employed to affect the neurological changes to maximize sexual neurophysiology prior to initiating medical therapies including paying attention to sexual sensate areas and visceral signals with mindfulness techniques, practicing body mapping, and sexual stimulation of sensate areas to encourage neuroplasticity. Attention should be paid to the biopsychosocial sexual contexts within which persons with SCI live to maximize their sexual and fertility rehabilitation. **Key words:** *autonomic nervous system, neuroanatomy, orgasm, sexual health, spinal cord injury*

Sexual disorders are common among individuals with spinal cord injury (SCI). However, there is still significant discomfort and lack of confidence among medical professionals in diagnosing and managing sexual dysfunctions following SCI. Recognizing the impact of neurological injury on sexual function, being familiar with possible tools for the evaluation of sexual functioning, and learning about the latest therapeutic strategies are all needed in order to be successful with management strategies for sexual dysfunction after SCI. This narrative review will address basic anatomy and physiology of sexual function, pathophysiology following SCI, and ideas for furthering sexual satisfaction prior to the addition of medical therapies.

Male Reproductive System

The male reproductive system is a network of internal and external organs that function to produce, support, transport, and deliver viable sperm for

reproduction via copulation or sexual intercourse. The internal organs consist of the testes, the ductal system (epididymis and vas deferens), and bilateral seminal vesicles attached to the prostate. The penis is composed of erectile tissue and a unique set of helicine veins and arteries capable of expansion. The penile erectile tissue (2 corpora cavernosa and 1 corpora spongiosum) has an internal portion (2 penile crura that attach to the ischial tuberosities) and external portion (the crura join to form the external portion of the penis at the penile bulb, or base, which elongates externally into the shaft and penile glans). The tunica albuginea is a fibroelastic stocking that surrounds the penile erectile tissue. The urethra runs from the bladder through the prostate and internally along the length of the corpora spongiosum of the penis until the external urethral meatus, which is located in the distal glans of the penis. The urethra carries urine from the bladder and seminal fluid (ejaculate), which is a mixture of testicular sperm, prostatic secretions, and fluid from the seminal vesicles. The external

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penile structures, attached to the pubic bone by a suspensory ligament, can be in a flaccid or erect state. Two testicles lie within the scrotum; each is attached to a spermatic cord that contains the nerves and vessels to the testes.

Internally, the male accessory organs provide a pathway for sperm to be transported from the testes and nurtured by accessory fluids before being expelled out the end of penile urethra (antegrade ejaculation). Ejaculation is usually accompanied by pelvic floor contractions and the sexual sensation of orgasm. Spermatozoa are made within the testicles, matured in the epididymis (which acts as a sperm reservoir), and transported through the vas deferens to be mixed with nourishing seminal and prostatic fluid. Semen, or ejaculate, is usually expelled in an antegrade fashion, but it can be expelled backwards into the bladder with incomplete closure of the (dysfunctional) internal sphincter, resulting in retrograde ejaculation. Cowper's gland (bulbourethral gland) produces a clear alkaline secretion ("pre-ejaculate") with high sexual arousal, which serves to lubricate and alkalize the urethral pathway for the passage of sperm. Embryologically, the testes are developed internally at the same level as the kidneys and, therefore, share a common level of innervation (T10-L1) after their descent into the scrotal sac (lower temperature required for spermatogenesis); the scrotal skin is innervated by the somatic branches of L1-L2 and S2-S3. The function of the male reproductive system is dependent on coordinated hormonal control from the autonomic nervous system. Spermatozoa are made in the testicular Sertoli cells and testosterone is produced in the Leydig cells. These cells are activated by the anterior pituitary hormones follicle-stimulating hormone (FSH) and luteinizing hormone (LH) respectively.

Female Reproductive System

The female reproductive system consists of external and internal organs that function to provide sexual arousal and orgasm and the ability for vaginal penetration. Heterosexual intercourse allows for penile penetration, ejaculation, and sperm deposition intravaginally near the cervix. Sperm can then propel themselves through

ovulatory cervical mucus into the cervix and up into the fallopian tubes where an ovulated ovum can be fertilized, potentially resulting in uterine embryo implantation and pregnancy. The external genitalia, also called the vulva, include the mons pubis, the labia majora (outer lips of the vagina), the labia minora (the inner lips of the vagina), the vaginal opening (introitus), the urethral opening, the clitoris, and the perineum.

Similar to men, women have vascular erectile tissue (the clitoris) that becomes engorged with blood (tumescence) during sexual arousal. The clitoris is composed of an externally exposed glans, a glans hood, and 2 crura that start externally then bend like a wishbone to internally attach to the ischial rami bilaterally. The corpora cavernosa are surrounded by a thin tunica albuginea, with the corpora spongiosum anatomically separate and surrounding the urethra found lower in the vaginal opening. During sexual arousal, vasocongestion of the pelvic organs results in the formation of a vaginal transudate through the estrogenized vaginal epithelial cells (vaginal lubrication), which is accompanied by vaginal elongation and uterine elevation ("tenting"). Near orgasm, the outer third of the vagina forms an orgasmic platform. At orgasm, the pelvic floor muscles rhythmically contract.¹ These physiological responses during female arousal (clitoral tumescence, vaginal lubrication, and uterine elevation) accommodate and assist comfortable vaginal penetration during sexual activities such as heterosexual intercourse.

Genital Arousal

Genital arousal in men and women is a dynamic process that involves the coordination of neuronal circuits that result in a final vascular event, creating an elongated, rigid erection in men and tumescence of the clitoris in women from their flaccid or detumescent states. The key to creating an erectile response is the dominance of parasympathetic activity that results in cavernosal smooth muscle relaxation over the tonic, sympathetically driven smooth muscle and blood vessel contraction responsible for flaccidity.² Cavernosal smooth muscle is controlled by balancing neurotransmitters responsible for contraction (norepinephrine, endothelin,

angiotensin, and vasopressin) and smooth muscle relaxation (acetylcholine, nitric oxide [NO], vasoactive intestinal peptide, prostaglandins, and calcitonin gene-related peptide).^{2,3} For either penile or clitoral erection to occur, the sacral component of the parasympathetic nervous system (S2-S4) is crucial. NO, the primary neurotransmitter responsible for penile erection and clitoral engorgement, is released at nerve endings during sexual arousal and sexual stimulation (neuronal NO or nNO) and is also made in the endothelium itself (endothelial NO or eNO).³ With the release of nNO at the nerve endings of the genital nerve fibers during sexual arousal, smooth muscle relaxation is initiated. The health of the endothelium will determine the contribution of eNO to the erectile process. Once smooth muscle relaxation occurs, vascular resistance is decreased and blood inflows through the cavernosal and helicine arteries, engorging the sinusoids; expansion and elongation of the corporal structures occurs. Therefore tumescence of erectile tissue in both men and women requires a neural input (nNO), which is then maintained by the sheer stress of the blood on the endothelium (release of eNO).

In the smooth muscle cell, NO activates guanylyl cyclase, which increases 3',5'-cyclic guanosine monophosphate (cGMP) levels in the smooth muscle cell of genital erectile tissue. The activity of calcium and potassium channels and intracellular contractile proteins that affect corpus cavernosum smooth muscle relaxation is regulated by cGMP. Phosphodiesterase type 5 (PDE5) is responsible for the destruction of cGMP and the eventual return of smooth muscle contraction. These intracellular mechanisms are targeted for pharmaceutical intervention to promote erection, such as the PDE5 inhibitors (PDE5i) used for erectile dysfunction.⁴

It is important to note that the tunica albuginea stretches over the expanding corporal bodies and increases the intracavernosal pressure. This expansion and increase in intracavernosal pressure compresses the subtunical veins against the tunica albuginea, causing entrapment of blood in the corpora cavernosa, referred to as the veno-occlusive mechanism. The male penis becomes rigid and erect, and the clitoris becomes tumescent.

Striated muscle contraction of the pelvic floor further adds to the intracavernosal pressure in the male and penile rigidity. Clitoral tumescence is less rigid than penile erection because the tunica is thinner and only surrounds the corpora cavernosal bodies. Detumescence will occur with sympathetic stimulation, causing smooth muscle contraction, reduction in arterial blood flow, release of the veno-occlusive mechanism, and eventual flaccidity.

Since genital arousal includes neuromuscular tension and vasocongestion to the pelvis, resulting in penile erection in males and clitoral tumescence, vaginal transudate (vaginal lubrication), and vaginal accommodation (elongation of the vagina and uterine tenting) in women,¹ a basic understanding of descending autonomic control is necessary. Genital arousal requires dominance of parasympathetic output in the terminal nerve endings in erectile tissue in men and women over the tonic sympathetic tone (responsible for detumescence) through the pelvic nerve, a final common pathway, that receives inputs from the medial preoptic area and genitals.⁵ With parasympathetic stimulation, proerectile neurotransmitters, particularly through the NO-cGMP pathway, promote smooth muscle relaxation and tumescence. However, the sympathetic nervous system also has a proerectile component, as demonstrated by lesions of the paravertebral sympathetic chain in humans and stimulation of the hypogastric nerve, and the sympathetic role may be "unmasked" following sacral spinal injury.⁶

Three types of genital arousal are apparent: psychogenic, reflexogenic, and nocturnal (rapid eye movement or REM sleep). Arousal triggered by sexual thoughts generated from the 5 senses or by sexual fantasy sends psychogenic impulses down the spinal cord and modulates the spinal erection centers of T11-L2 (psychogenic) and S2-S4 (reflexogenic).⁷ When the S2-S4 center is inaccessible following sacral SCI, the T11-L2 thoracolumbar centre becomes the dominant pathway for transmission of psychogenic signals of erection.⁸ Psychogenic erections in men with lumbosacral lesions are often of poorer quality, as these erections may result from inhibition of the tonic sympathetic tone and/or from the relaxation of the penile sinusoidal cavities, rather than true

vasodilation and penile rigidity.⁹ Through fMRI studies, some authors have concluded that the vagus nerve can convey sensory information from cervical stimulation in women with complete SCI,¹⁰ but this has been relatively unexplored.³

Reflexogenic genital arousal is produced by tactile stimulation to the genitals resulting in afferent stimuli to the spinal cord (afferent input), with some signals following an ascending tract (sensory perception) and others activating the autonomic nuclei S2-S4 resulting in cavernosal nerve activation, smooth muscle relaxation, and tumescence (efferent output).⁷ In complete spinal cord lesions above the T10, impulses fail to reach the psychogenic arousal center of T11-L2 and cannot ascend the spinal cord to add to sensory perception. Reflexogenic arousal therefore dominates. Sacral parasympathetic neurons are important in the preservation of reflex erection.⁷ Furthermore, the more cranial the complete SCI above thoracolumbar outputs, the more sensitive the reflexogenic response due to sacral reflexes free of descending control.

Nocturnal erections occur mostly during REM sleep, which is triggered within the pontine reticular formation. PET scans suggest that differential activation in specific brain areas (ie, activation of cholinergic neurons versus the silencing of adrenergic and serotonergic neurons) may be responsible for nocturnal erections during REM sleep.¹¹ Nocturnal erections have not been well studied after SCI.

Activation of both the autonomic and the somatic neural pathways occurs during the erection phase.⁶ Contractions of the pelvic floor muscles, particularly the ischiocavernosus muscle, increases penile rigidity. Intactness of the bulbocavernosus reflex is confirmatory for reflexogenic genital arousal potential.

Ejaculation

Ejaculation, the process of external semen expulsion, is primarily a sympathetic phenomenon (T10-L2). It is a highly coordinated muscular and neurological event that deposits semen in the prostatic urethra via the ejaculatory duct in the prostate (seminal emission) and then

ejects this fluid via the urethra out the external meatus by rhythmic contraction of the pelvic floor muscles around the urethra (propulsatile ejaculation). Ejaculation involves specific afferent sensory pathways; cerebral and spinal integrative, autonomic, and somatic centers; and efferent pathways.¹² Furthermore, a spinal generator of ejaculation, identified and unaltered after SCI in spinalized rats,¹³ is located at the L3 and L4 spinal segments.¹⁴ It is also most likely retained in men with SCI, as evidenced by the success of penile vibratory stimulation (PVS) in men with lesions higher than T6 and the relative ineffectiveness of PVS in those men with lesioned lumbar segments.¹⁵ Propulsatile ejaculation has a parasympathetic component (accessory gland peristalsis) as well as a somatic component (pelvic floor contraction).

Orgasm

Orgasm is the release of pelvic vasocongestion and neuromuscular tension most often felt locally in the genital area and experienced as pleasurable in the brain. Even though it is a neurologic phenomenon, the neural circuits are not well understood. Since the definition and interpretation of orgasm remains neurophysiologically unclear, most studies of orgasm in persons with SCI are reliant on subject self-report, physiological measures (particularly of heart rate and blood pressure), or documented phenomenological experiences.¹⁶

Genitally induced orgasm appears to require an intact sacral reflex, but orgasm can be experienced from stimulation outside the genital region (nongenital stimulation, such as nipples and ears) and with psychogenic fantasy alone (as in sleep). The perceptual experience of orgasm, once achieved, does not vary much between men and women and can be modulated by several factors, primarily psychological as opposed to physical, such as emotional intimacy.¹⁷ In the majority of men, orgasm accompanies ejaculation; however, these 2 events require activation of different complex neurophysiologic circuits. Separation of timing of orgasm from ejaculation, or having one process without the other, can occur. It is also possible to have orgasm or ejaculation without a penile erection or even penile structures.

Neurological Control of Sexual Responses

Peripheral innervations of the pelvis involve 3 sets of efferent neurons coordinated through the pelvic plexus: somatic, thoracolumbar sympathetic, and sacral parasympathetic.⁵ Since there is cerebral descending excitatory and inhibitory control over the pelvic innervations, sexual responses are modulated by minute-to-minute cerebral influences and neurotransmitter alterations.¹⁸ The motor and sensory somatic control is via the pudendal nerve, and the 2 components of the autonomic system are via the pelvic nerve (sacral parasympathetic) and hypogastric nerve (thoracolumbar sympathetic). The somatic afferent (motor) pathways are essential for initiation of the pelvic floor muscles contractions. The afferent (sensory) pathway involved in sensory innervations of the genitalia includes the hypogastric, pelvic, and vagus nerves¹⁹ and those responsible for mechanosensitivity (sensation of touch and pressure), thermosensitivity (sensation of temperature), and chemosensitivity (ie, irritants), all of which contribute to sexual interpretation of genital stimulation. It should be remembered that the whole body is sensitive to sexual touching, with erogenous hotspots consisting of genitals, breasts, and anus²⁰. This becomes important after neurological injury or insensate genitalia.

The autonomic nervous system is composed of the sympathetic and parasympathetic systems, which are integrated functionally within the central nervous system and provide balanced regulation of most of the visceral organs.²¹ Both divisions of the autonomic nervous system have preganglionic and postganglionic neurons that are interposed between the central nervous system and target organs. The cell bodies of the preganglionic neurons in the gray matter of the brain or spinal cord and their axons, called preganglionic fibers, travel within the ventral roots of the spinal cord or cranial nerves. The preganglionic fibers synapse with the postganglionic neurons in autonomic ganglia. The axons of these neurons, postganglionic fibers, innervate target organs. Sympathetic preganglionic neurons reside in the spinal gray matter of the thoracic and upper lumbar segments (T1-L2)²² and synapse on postganglionic sympathetic neurons of

the spinal paravertebral sympathetic chain ganglia and the celiac, superior, and inferior mesenteric ganglia (prevertebral ganglia). The sympathetic nervous system innervates the heart, blood vessels, respiratory tract, sweat glands, sexual organs, bowel, and bladder (**Figure 1**). Parasympathetic preganglionic neurons originate in the brainstem (cranial nerves III, VII, IX, X) and in the sacral spinal cord segments (S2-S4). In addition to the sacral parasympathetic control of the pelvic organs, cranial nerve X (vagus) contributes to innervation of the uterus and vaginal wall.¹⁰ The parasympathetics do not innervate the peripheral vasculature except for the pelvic organs. The bladder, reproductive organs, and lower portion of the gut receive parasympathetic innervation from S2-S4.

Male and female sexual organs receive sympathetic innervation via the hypogastric nerve and parasympathetic innervation via the pelvic nerve. Psychogenic erection and vaginal lubrication are mediated via the sympathetic fibers in conjunction with the parasympathetic, whereas reflex erection and vaginal lubrication are mediated by the parasympathetic nervous system.^{23,24} Ejaculation is a more complicated phenomenon and requires coordination of the sympathetic (T11-L2) and parasympathetic (S2-4) spinal centers in addition to the somatic nervous system via the pudendal nerve (S2-5).²⁵⁻²⁷ More detailed information on this topic can be found in previously published manuscripts.^{28,29}

Sexual Dysfunctions After SCI

It is well established that physiological, psychological, social, and emotional factors can contribute to male and female sexual dysfunctions. Sexual desire or libido is especially complex and is a combination of biological (ie, hormone and neurotransmitter) and psychological (the physical, emotional, and contextual sexual payoff associated with being sexual) factors. Kaplan defines libido as “the experience of specific sensations that motivate the individual to initiate or become responsive to sexual stimulation.”^{30(p42)} If a person with disability experiences a change in drive, it is most often a reduction and can be termed low sexual desire or

Figure 1. Innervation of the pelvic organs and male genitalia. In the pelvic organs and genitalia, there are 3 main tissue types: secretory, erectile, and striated muscle. The majority of the autonomic innervation to these tissues comes from the bilateral pelvic ganglia, which contains both sympathetic and parasympathetic neurons. Parasympathetic preganglionic neurons originate in the sacral cord (S2-S4) and travel in the pelvic nerve to the pelvic ganglia. Sympathetic innervation originates in the lower thoracic and lumbar cord (T10-L2) and travels via the hypogastric nerves to innervate the pelvic ganglia; sympathetic nerves also travel to the pelvic plexus via the pelvic nerve, which is mixed sympathetic and parasympathetic. These plexuses form a diffuse neural network on either side of the prostate (males) or cervix (females). In both sexes, the largest nerve exiting from the pelvic plexus is the cavernous nerve (also called penile nerve in males). Only innervation of the seminal vesicles is illustrated here. Somatic innervation of the striated perineal muscles, which include the ischiocavernosus, bulbocavernosus, and levator ani, originates in the sacral spinal cord segments (S2-S4). Afferent information from the pelvic organs is relayed to the spinal cord via the “genito-spinal” nerves (pelvic, hypogastric, and pudendal; only the pelvic nerve is illustrated here), and sensory pathways ascend bilaterally in the dorsal quadrant of the spinal cord. Reprinted with permission from Krassioukov AV, Biering-Sorensen F, Donovan W, et al. International Standards to document remaining Autonomic Function after Spinal Cord Injury (ISAFSCI), First Edition 2012. *Top Spinal Cord Inj Rehabil.* 2012;18(3):290. Copyright © 2012 Thomas Land Publishers, Inc.

low libido. If this is a temporary reduction as a result of a traumatic experience without causing distress to the person, it is not usually regarded as pathological. Sexual drive should slowly resume to baseline as the person accommodates to his or her disability, but iatrogenic causes such as medications or continuing factors such as pain or depression can continue to negatively influence sexual drive. In rare cases, sexual drive may be increased (resulting in hypersexuality) due to changes within neuronal brain circuits accompanying concomitant brain injury or the use of some medications. Clinically, other sexual dysfunctions include genital arousal disorders (disorders of male penile erection [erectile dysfunctions] and female vaginal lubrication and accommodation), ejaculatory dysfunctions in men (including anejaculation, disordered ejaculation, and premature or delayed ejaculation), and orgasmic disorders (alterations to intensity or duration and anorgasmia). Sexual problems in both men and women can also include pain with sexual arousal, with ejaculation/orgasm, or during sexual intercourse or penetrative activities (dyspareunia). Problems of sexual interest, genital arousal, and ejaculation and dyspareunia can also lead to fertility issues in both sexes.

Sexual Changes Following SCI

Evaluation of sexual dysfunctions following SCI has been focus of numerous studies using animal models³¹⁻³³ and in individuals with SCI.^{9,34-38} These studies expand our understanding of the neurophysiologic changes in sexual responses following SCI and provide an excellent model for a holistic and reintegrated approach to sexual rehabilitation. Furthermore, as evident from literature, individuals with SCI rate the recovery of sexual functions among their highest priorities for improving of quality of life.³⁹

The degree of sexual dysfunction varies significantly among individuals with SCI and depends on the level and the severity of injury.⁴⁰⁻⁴² After complete SCI above the lumbosacral spinal cord center (usually above T10), reflexogenic arousal should be preserved whereas psychogenic arousal will not (due to the lesion interrupting

the pathways from the brain to the T10-L2 spinal segment, the level responsible for psychogenic arousal).⁴⁰ Conversely, complete lesions interrupting the sacral reflexogenic pathways will result in the reliance on psychogenic arousal to promote genital arousal in men and women. However, as evident from the clinical practice, men with sacral SCI who are reliant on psychogenic arousal can induce activation of the sympathetic hypogastric pathways through mental arousal; activation of the sympathetic pathways may trigger unwanted seminal emission and penile detumescence. Men and women with varying SCI levels and incompleteness will experience various capacities to utilize their psychogenic or reflexogenic pathways.

The degree of preservation of combined light touch and pinprick sensation within the T11-L2 dermatomes is helpful in predicting those persons with SCI who are capable of psychogenic arousal.⁴² The presence of a positive bulbocavernosus reflex is indicative of an intact sacral reflex, boding well for the reflexogenic genital arousal capacity. Although more than three-fourths of men with SCI can obtain an erection, firmness and reliability in sexual situations can be a problem, necessitating the use of erection enhancement drugs in some cases. Ejaculatory disorders (primarily anejaculation) are highly prevalent (over 90%), and thus fertility is a major issue for men with SCI.⁴³ Natural ejaculation is most likely to occur in men with incomplete conus medullaris or cauda equina lesions and is least likely in men with complete supraconal lesions.⁴⁴ However, vibrostimulation-assisted ejaculation is more reliable in complete supraconal lesions.⁴⁵ Following SCI, orgasm is attainable in 40% to 45% of men (most often with ejaculation but not always)^{42,43} and in approximately 50% of women.⁴⁶ Lack of genital sensation, and especially lower motor neuron injury affecting the sacral segments, makes it significantly less likely that a person will reach a genitally triggered orgasm.⁴⁷ Even though sexual satisfaction after injury is lower in both men and women after SCI, intimacy remains a major quality of life issue for the majority of people living with SCI.^{39,48} Furthermore, there is no clinically agreed upon SCI measurement tool for sexual

health outcomes to adequately assess the complex issue of sexual health and satisfaction.⁴⁹

SCI sexual health is affected by direct and indirect consequences of neuronal injury and also by the social, cultural, and personal contexts that can affect a person's ability to perceive sexual activities as pleasurable and rewarding. An alteration in autonomic and somatic nervous systems following SCI can affect the individual from head to toe, impacting various organs and systems and affecting sexuality and fertility. As with able-bodied peers, having or not having a partner also contributes significantly to sexual life outlook. Sexual and fertility rehabilitation must therefore take into consideration the wider biopsychosocial contexts in which persons with SCI live.

Potential Therapeutic Avenues to Alter Sexual Pathophysiology

Any preservation of remaining somatic and autonomic pathways will be critical in reestablishing sexual function already familiar to the person with SCI prior to their injury. Sexuality is a complex mind-body experience, so the neurotransmitters involved in sexual function and pleasure should be respected in any treatments of SCI-related problems and iatrogenic causes of sexual dysfunction or dissatisfaction should be avoided if at all possible. However, sexual recovery is the sole responsibility of the person with SCI; they must be willing to embark upon the journey of sexual rediscovery.

Are there ways to neurologically alter the remaining neurophysiology after SCI? There are 2 ways to affect the sensory contribution in remaining sensate or altered sensate areas. One is the mind's ability to focus and appreciate the remaining sensation to its fullest. Techniques such as mindfulness and fantasy can be used, and sensate receptors found in other erogenous areas (anus, rectum, ears, nipples, or other areas found to be sensitive) can be incorporated. Promoting mindfulness and awareness of sexual signals, even from nongenital sources, is being recognized as an important treatment tool in sexual dysfunction.⁵⁰

Theoretically, another approach for altering the neurophysiology is to enhance the local

vasodilatation of the genital or other erogenous areas by the use of medications that increase topical sensation, such as botanical preparations, or underlying vasocongestion, such as PDE5i. Medications such as PDE5i are presently widely used for male erectile disorders, enhancing the subjective arousal (even if not the objectively measured arousal) of some persons after SCI. However, PDE5i appears to have no clinically meaningful benefit in sexual responsiveness in women with SCI (similar to other populations of women).⁵¹ To date there is no known work specifically on these medication therapies in persons with SCI. However, anecdotal information exists, and some studies have suggested benefits. For example, it was found that the use of vardenafil to enhance erectile quality in men with SCI also increased their chance of ejaculation and orgasm and increased sexual satisfaction rates.⁵²

With the loss of recognizable sensory pathways for sexual arousal, persons with SCI must use a process of body mapping to scan areas of remaining sensation that have the potential to initiate sexual arousal. These latent pathways (ears, nipples, neck, and hair) can lead to heightened arousal not previously noted from these sites prior to injury. Utilizing the principles of neuroplasticity, the use of fantasy and mental sexual arousal (with or without a partner), concomitant with consistent and repetitive stimulation of a body part, may allow that body part to become a source of sexual arousal. This process requires extensive time and repetition, similar to motor rehabilitation, but it has the capacity to go beyond somatic limitations. By example, persons with SCI have taught conservative medical practice the benefit of this form of neuroplasticity. Only preliminary work on sexual neuroplasticity has been done to date.²⁶

Conclusion

Changes in sexual functioning are not just organic in nature; outside influences (spinal cord-related or not) affect the thoughts, willingness, and actual physiological sexual functioning after SCI. Persons with SCI are people *first*, and persons with SCI *second*. They will have positive and negative sexual capacities prior to

their injury that may resolve or worsen after SCI. In general, after SCI, acquired sexual problems may feel overwhelming; incorporating a “new body” into a new sexual life takes motivation and persistence. However, from a physiological point of view, sexuality can continue to expand and grow unlike the definitive plateau of somatic and autonomic nerve recovery. Therefore individuals can have an expanded and full sexual

life despite motor, sensory, bladder, bowel, or other SCI-related impediments. Conditions leading to neuroplasticity appear to play an important role in the ability of individuals to have positive and pleasurable sexual experiences after SCI.

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