Ejaculation and Orgasm: Sexuality in Men with SCI

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Ejaculation and orgasm are two entities of sexual satisfaction in men after spinal cord injury (SCI). The scientific literature focuses on potentiating erection and on the sperm retrieval procedures for fertility purposes, but little has been written about the pleasurable aspects of ejaculation and the potential for orgasm in men after SCI. Men with SCI who have lower motor neuron or incomplete injuries appear to have an increased chance of ejaculating through sexual practices, whereas men who have injuries above neurological level T10 respond best to vibrostimulation. Orgasm after SCI is a local, learned spinal reflex that is interpreted via cerebral centers. In general, intense genital stimulation may be needed to elicit the subjective experience of orgasm, but extragenital stimulation or cerebral input alone can lead to orgasmic release for some men after SCI. Sexual rehabilitation includes three principles: maximization of the innate physiological potential, adaptation to limitations, and promotion of a positive outlook for sexual potential via experimentation. Key words: *ejaculation, erection, orgasm, spinal cord injury*

Many articles on male sexuality after spinal cord injury (SCI) focus either on erection dysfunction or on fertility problems. However, little attention is paid specifically to the psychophysiological aspects of sexual pleasure attainable after SCI, including the ability to ejaculate and the subjective experience of orgasm. After SCI, many men initially focus on the ability to achieve an erection adequate for sexual intercourse, even if they cannot feel the erection or the pelvic arousal per se. Earlier researchers[1,2] emphasized the vicarious pursuit of pleasure after SCI, claiming the loss of this "genitopelvic"[1(p90)] awareness resulted in a focus on the "cerebrocognitional aspect"[1(p91)] of sexual ability, with satisfaction mainly resulting from a "boost of self-esteem and pride of accomplishment at being able to satisfy the partner."[1(p91)]

However, later articles emphasized that the sexual pleasure of the person with tetraplegia or paraplegia need not be limited to the satisfaction gained by gratification of a partner.[3,4] More recently, client-oriented writings[5] promote positive sexuality and pleasure for the person with SCI. A focus on the enhancement of the remaining capacities instead of preoccupation with the lost capacities, as well as receptivity to the sexual power of emotional intimacy, may result in a more rewarding sexual life after injury.

Current literature on erection function in men with SCI is not centered on the attainment and reliability of natural erections after SCI but on methods of erection enhancement. Literature on ejaculation tends to focus

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Top Spinal Cord Inj Rehabil 2002;8(1):1–15

on sperm retrieval methodology. Erection dysfunction treatments and sperm retrieval for fertility are both very important in the sexual rehabilitation of men after SCI, but sexual pleasure is also of great significance. There is a paucity of information on orgasmic sensations accompanying ejaculation during sperm retrieval in a clinical setting, let alone sensations accompanying ejaculation at home in a more conducive, private setting. Clinicians have been told of orgasmic experiences that are unaccompanied by ejaculation.[6] No formal surveys have looked at such occurrences, but these anecdotal reports are crucial in the clinician's learning, understanding, and knowledge of sexual physiology after SCI and they ultimately direct new research. For the patient, the Internet is probably the biggest source of such information. The use of chat lines or question and answer sites to share experiences is a valuable method of learning how to obtain better arousal and release. Clearly, the area of natural arousal, release, and sexual pleasure for both men and women after SCI needs further directed, scientific research.

The scientific literature has focused primarily on self-reports and, therefore, on subjective definitions of erection, ejaculation, and orgasmic capacity. An erection that is defined as adequate for vaginal penetration in a study protocol may be quite different from an erection that is rigid and reliable enough for satisfying coital and/or noncoital sexual activities. For most men, ejaculation is considered to have occurred if there is visible confirmation of ejaculate, yet orgasmic capacity can be variably interpreted and may not be related to ejaculation at all. Furthermore, it is only by going beyond conventional scientific thinking about sexual physiology that some men with SCI have experienced their full sexual potential.

This article will concentrate on aspects of genital sexual functioning and pleasure in men after SCI, including the potential for ejaculation and orgasm and how these interrelate with erectile capacity. The neurophysiology of erection has become well elucidated over the last few years, and ejaculation physiology is not far behind. However, there is little comparable understanding of orgasm or even how orgasm is defined, especially after SCI. This article is an overview of the current literature combined with my clinical experience, observations, and theories about the mechanism and sexual interpretations of erection, ejaculation, and orgasmic function after SCI. Other articles in this issue provide more specific information on erection dysfunction and its management, sperm retrieval, and fertility.

Sexual Functioning After SCI

The 1960 article by Bors and Comarr[7] is one of the original, and most frequently cited, self-report studies on the sexual capacity of men after SCI. Their objective was to quantify sexual responses according to level of injury and completeness of SCI but not to provide information about sexual pleasure or overall sexual satisfaction. The issues of sexual and fertility rehabilitation were not as openly approached in the hospital setting then as they are today. Without the erection enhancement techniques and fertility options that are available now, sexual and paternity expectations were understandably downplayed by health professionals. Erection enhancement consisted mainly of penile prosthesis until intracavernosal injection became available in the early 1980s. Fertility was limited to those men who could ejaculate with sexual activity and whose sperm quality

was adequate for conception; this was about 5% of men in the original study.[7] It must be remembered that bladder care was less sophisticated at that time, the level and extent of the spinal injuries were not as well defined, and surgical procedures and medications used for bladder management may actually have interfered with sexual function and fertility potential.

Since the original report, other researchers have investigated the ranges of sexual capacity. The reported frequency of erections in men with SCI ranges from 54% to 95%, and frequency of ejaculation ranges from 3% to 20%.[7–9] A data summary[10] in 1977 showed erection capacity as 93% reflexogenic in patients with complete upper motor neuron (UMN) lesions, 98% reflexogenic in patients with incomplete UMN lesions, 26% psychogenic in patients with complete lower motor neuron (LMN) lesions, and 83% psychogenic in patients with incomplete LMN lesions. Bors and Comarr[7] indicated that men with incomplete injuries and UMN injuries had a better prognosis for erection as compared to complete and LMN injuries, but it must be remembered that self-reports do not always accurately reflect the full physiological response to erotic stimulation. For example, in male patients with SCI who felt they could not get erections, penile tumescence was nevertheless demonstrated when they were exposed to erotic stimulation via film, text, and fantasy.[11]

Ejaculation during sexual activity is generally reported in less than 10% of men with complete SCI and in 32% with incomplete SCI. Men with LMN lesions and more caudal lesions more commonly report ejaculation capacity.[12] The ability to have orgasm through sexual activity is poorly defined in most studies.[12] Fortunately, with the advent of vibrostimulation, ejaculation is more likely to occur, especially in patients whose injury is above the T10 neurological level. The vibrator is capable of specific frequencies and amplitudes not easily duplicated by sexual techniques. However, for some men, the reverse is true; they can ejaculate to specific sexual stimulation but not to vibrostimulation.

Ejaculation consists of two stages – seminal emission and propulsatile ejaculation – and is mediated through the T10-S4 segments of the spinal cord.

Recent self-report studies of men with SCI state that 42%–47% experience orgasm of a similar, weaker, or different quality than preinjury.[13,14] Although awareness of genital orgasm (the ability to feel genitally centered orgasmic release) is assumed to depend on intact genital afferents (upgoing lateral spinothalamic tracts) to the brain and intact efferents (downgoing corticospinal tracts) from the brain,[4] 38% of men with complete SCI reported they retained the ability to achieve orgasm.[13]

Neurology of Ejaculation and Orgasm

Ejaculation consists of two stages—seminal emission and propulsatile ejaculation—and is mediated through the T10-S4 segments of the spinal cord. Ejaculation is a spinal reflex. Like erection, this reflex is under tonic inhibition from the supraspinal centers.[15] Usually, genital stimulation in combination with central arousal acts as an afferent that removes this inhibition, allowing the natural efferent components to un-

fold. Excitatory supraspinal afferents alone can induce ejaculation as evidenced by nocturnal emissions. Alternately, if supraspinal control is lost because of complete SCI, the tonic inhibition is removed, allowing the undamaged sacral reflex to be triggered by the appropriate genital afferents, which results in ejaculation.

Stage 1 or *seminal emission* is arousal related and is under some voluntary control in the able-bodied man. This phase involves the sympathetic outflow from the presacral and hypogastric nerves (T10-L2) promoting sperm transport from the storage site in the tail of the epididymis to the more distal genital ducts. Smooth muscle peristalsis of the vas deferens and contraction of the seminal vesicles cause the spermatozoa and seminal fluid to mix with the prostatic secretions to form the seminal bolus. The seminal bolus is then deposited into the prostatic urethra via the ejaculatory ducts. Sympathetic input via the hypogastric nerve (L1,2) causes a closure of the bladder neck by stimulation of alpha-adrenergic receptors that, in turn, prevents the seminal bolus from entering the bladder (retrograde ejaculation). In addition, the external sphincter also remains closed during the seminal bolus deposition, increasing the prostatic pressure and instigating a feeling of impending release, called ejaculatory inevitability. Cerebral control of impending ejaculation is minimal at this point.[16] Increased prostatic pressure and the general vasocongestion of the pelvic organs correspond with conscious pleasurable feelings of tension in the genital area.

Stage 2 or *propulsatile ejaculation* is the process of expulsion of the ejaculatory fluid out the urethral meatus where it appears as an antegrade ejaculate. The pelvic nerve carrying parasympathetic fibers from S2-S4 causes spasmodic contractions of the seminal vesicles, prostate, and urethra, which propels the seminal bolus distally. Intermittent relaxation of the external sphincter coordinates with the three to seven rhythmic contractions (approximately 0.8 seconds apart) of the bulbospongiosus, ischiocavernosus, levator ani, and related muscles innervated by the pudendal nerve carrying the somatic fibers.[16]

It has been proposed that a neural "coordination center" for ejaculation exists in the T12–L1 region.[17] Afferent signals from S2-4 ascending to the "center" stimulate sympathetic outflow, triggering the initial stages of seminal fluid deposition and emission, and precisely direct the activation of the descending efferent parasympathetic and somatic outflow through the sacral cord to the pudendal nerve. The latter results in clonic contractions of the periurethral musculature and antegrade ejaculation.

Because propulsatile ejaculation immediately follows seminal emission, it is thought that an acute rise in intraprostatic pressure from the presence of the seminal bolus in the ejaculatory ducts and prostatic urethra may assist in triggering the switch from the sympathetic to the parasympathetic nervous system. It is still not clear if the somatic or autonomic nervous system is responsible for the orgasmic sensations; they may both provide input. Furthermore, it is still not clear, even in neurologically intact men, in what phase orgasm actually occurs.

Men with alterations to one or the other of their ejaculation phases help our understanding of orgasmic sensations. For example, men who have their prostate removed by radical prostatectomy and who no longer have antegrade ejaculation still feel pleasure during the phase of seminal emission and

experience orgasm, although it may be altered in intensity or duration.[18] Men in whom there is technically no seminal emission due to interruption of their sympathetic chain (as in retroperitoneal lymph node dissection for testicular cancer) still have the second phase triggered and experience orgasm despite not having an increased prostatic pressure from the seminal bolus deposition.[19] Men with altered somatic innervation, such as may be seen in multiple sclerosis, may have poor motor contraction of their pelvic floor and take longer to reach what is usually a blunted, less intense orgasm; ejaculate appears by gravity after orgasm has occurred, regardless of the erectile ability.[20]

Many men with SCI are anejaculatory, with neither seminal emission nor pro-pulsatile ejaculation; other men with SCI have variable capacities for ejaculation and orgasm, depending on the completeness and level of injury.[21] However, it is my observation that the sexual experiences of many men with SCI do not neatly fit the predictions stemming from current knowledge of neurology; thus, there may be additional factors besides hard-wiring that modulate the outcome of each individual's capacity or interpretation of sexual pleasure.

Changes to Ejaculation and Orgasm After SCI

Because ejaculation is a highly complex process requiring the sequential coordination of the sympathetic, parasympathetic, and somatic nervous systems, the exact alteration in ejaculatory function after SCI can be unpredictable. There can be combinations of interrupted seminal emission, inadequate closure of the bladder neck, and poor propulsatile ejaculation, all leading to various and sometimes unexpected ejaculation capacity. Physiological factors, such as current medical status, presence of bladder infections or pressure sores, and medications, may alter or attenuate ejaculation in men with SCI. Psychological influences on sexual functioning (even in complete SCI) tend to be unrecognized and/or underestimated. Both of these factors influence the reliability of ejaculation with sexual activity from one sexual attempt to the next, especially compared to preinjury. Compared with literature on the capacity for ejaculation and assisted fertility after SCI, there has been little written about the quality and predictability of ejaculation during sexual activity, let alone sexual satisfaction and orgasm.

Usually, obvious pulsatile antegrade ejaculation is triggered by vibrostimulation, because the sacral reflex is evoked. However, sometimes retrograde ejaculation occurs as evidenced when typical ejaculatory signs are present with no or small amounts of antegrade ejaculate. Unlike vibrostimulation, electroejaculation is thought to evoke only seminal emission. The semen flows from the urethra, and often more can be expressed by milking the urethra and penile bulb. More samples appear to go retrograde with electroejaculation than with vibrostimulation. Our standard protocol is to hold a balloon Foley catheter against the bladder neck during electroejaculation procedures; semen is then directed antegrade around the catheter and is collected.

The vast majority of patients who undergo monitored sperm retrieval have no feeling of pelvic arousal or accompanying orgasm with ejaculation. Even if they do not experience orgasmic release, just seeing the ejaculate—possibly for the first time since their injury—is important to their manhood and sexual

sense of self. The process of obtaining an ejaculate is not a sexual experience in the clinical setting. Many men feel unwell before ejaculation with discomforting spasm or impending autonomic dysreflexia. Men with higher lesions often get dysreflexic at ejaculation. These are not pleasant experiences. Despite this, it is my observation that approximately 5%–10% of these men experience new pleasurable sensations at ejaculation that they have not felt before through anejaculatory sexual activity. If this experience occurs with vibrostimulation, some men can then capitalize on this by learning to ejaculate at home under sexual, but safe, conditions (the risk of autonomic dysreflexia with such sexual activity can be tempered by precautionary positioning and the use of prophylactic antihypertensives). This is especially true for men with incomplete lesions; vibrostimulation with the additional cognitive arousal available in a private setting (or more accurately, the removal of remaining inhibitory control that still prevails in a clinical setting) contributes to improved outcome.

Orgasmic capacity after SCI is poorly understood. Part of the problem in discussing orgasmic capacity and satisfaction is that there is no clear definition of what constitutes "orgasm." Neurologically intact patients at the Sexual Medicine Clinic, BC Center for Sexual Medicine, describe orgasm in various ways based on their sexual experience. Even though it is possible to have orgasm without erection,[22] these men feel that their orgasmic reliability is improved with an erection and strive to attain erection before allowing themselves to actively pursue orgasm. Other men, especially those with generalized erection dysfunction secondary to medical or surgical causes, have already discovered that erection is not required to reach orgasm.[23] Clinical questioning reveals that, regardless of erection status, some men identify their seminal emission phase as "orgasm," which is followed by a lesser release phenomenon (also pleasurable) as the fluids are ejaculated with force to the external meatus during propulsatile ejaculation. These orgasms can be "drawn out" or extended in duration and intensity, or even in multiplicity, by the voluntary component of the seminal emission phase: once started, propulsatile ejaculation is under poor voluntary control. For other men, orgasm is the brain's cognitive interpretation of the pulsatile contractions of smooth and skeletal muscle during the second phase. The lay press has recently popularized exercises specific to pelvic floor awareness and strengthening as the basis for improved or multiorgasmic sensations in men.[24] Other men are not able to articulate whether their orgasmic sensation takes place primarily in the first or the second phase: one phase blends into the other with varying intensity. Each man has his own descriptive definition of the final constellation of events that constitutes orgasm.

Once the second phase is completed, a refractory period ensues. Repeat orgasms rarely occur without an interim recovery period called the *refractory period*.[23] The duration of this refractory period is widely influenced by age and sexual experience. Some men (usually young) are capable of more than one ejaculation within a relatively short period of time. They describe orgasm with each such experience and may not experience penile detumescence between orgasms. Typically, orgasm delays the refractory period. Men with premature ejaculation often ejaculate prior to partner sexual activity as a way to increase their ejaculatory latency with intercourse. As men age, the refractory period lengthens, pe-

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The fact that orgasmic experiences can occur with or without an erection, without ejaculate being produced, and even without structures (i.e., a prostate after cancer,[23] a penis after amputation for sex reassignment surgery[25]) indicates the resilience of the orgasmic capacity. It also supports the theory that orgasm is a reflex with cerebral influence and can therefore be learned and practiced. Low androgen states and medications that either influence reflex capacity (such as antispasmodics[26]) or delay orgasm (such as some antidepressants) can influence the ability to reach orgasm and/or the intensity of orgasm. SCI is one of the biggest challenges to this orgasmic resilience.

How has orgasm been defined in the scientific literature? Researchers working in the scientific study of orgasm can monitor specific physiological responses of increased heart rate, blood pressure, and pelvic floor contractions and correlate this to subjective awareness. Literature on the neurophysiology of ejaculation uses the words "sexual climax" or "orgasm" interchangeably with "ejaculation," but they are two different phenomena. Orgasm has also been described as a "combination of pleasurable physical and psychological sensations" that occurs during seminal emission and projectile ejaculation [27] or, alternately, as the "pleasurable and localized (genital) sensations at the time of seminal emission"[28] alone. Another description is that orgasm is thought to be the conscious perception of the contraction of both the smooth muscles of the internal sexual organs and the striated pelvic muscles.[29] Orgasm has also been described as a spinal cord reflex that is triggered by appropriate cerebral and sensory stimulus and that therefore can be inhibited by supraspinal suppression (conscious or subconscious).[30] As is well known in the field of sex therapy for female anorgasmia and male-inhibited ejaculation, the provision of effective, excitatory physical stimulus of sufficient intensity and duration, as well as the temporary removal of the normal tonic inhibitory cortical control on the spinal reflexes, is necessary for orgasm to be triggered. This supports the traditional view that orgasm is a pelvic reflex response and a learned reflex.

In our neurologically intact population, the intensity of pleasurable sensations seems to be dependant on psychophysiological factors such as length of abstinence, duration of arousal, strength and tone of the pelvic floor muscles, and intensity of cerebral afferents (sensory input from fantasy, memory, visual, olfactory, tactile, and taste afferents). The majority of able-bodied men experience orgasm resulting from stimulus on the penis (hand, oral, vibratory, intercourse). However, some men can experience orgasm from stimulation to nonpenile sites, such as the anus, perineum, prostate, nipples, or, rarely, by psychogenic (fantasy, memory, other cognitive sensory triggers) input alone. As mentioned before, the capacity for orgasm is rarely lost despite significant medical or surgical alterations. However, the genital orgas-

mic capacity of men after SCI is often lost or significantly altered, and therefore the "sexual pay-off" for men diminishes until they learn new pathways of arousal or adapt to their limitations. Men with SCI report becoming orgasmic from either intense genital stimulation or from learning to become highly receptive to and aroused by nongenital stimulation. The example of "eargasms" discussed by patients is one such mechanism. Other men with SCI learn alternate sexual inputs they may not have been attuned to before their injury. These include previously unrecognized erogenous zones or a new attention to emotional or spiritual connections. The combination of these new inputs with stimulation of sensate areas of the body can result in what is recognizable as an orgasm post injury. Intriguing accounts of spiritual focusing during sexual activity that may not even involve the genitalia have also triggered orgasm in some patients with SCI. Although these orgasmic experiences are different in nature and location, they can be just as sexually gratifying and meaningful, if not more, as the recognizable genitally based orgasms that were experienced before injury. However, it takes tremendous motivation and support to learn or to appreciate these new pathways.

Prior to injury, experience probably makes orgasm more reliable through neural memory or "grooving," which fosters confidence. How this translates to critical amounts of neurotransmitters required at the spinal cord level is still unclear. There may be many factors, such as the integrity of the serotonergic neurons in the spinal cord. For example, ejaculation reflexes, which were absent in cord-transected paraplegic rats, were restored with intraspinal transplants of serotonergic neurons below the level of the lesion.[31] If the reflex response remains after SCI, and the autonomic nervous system (or some yet unidentified hormonal or alternate neural transmission such as the vagus nerve[32]) is still intact, then orgasm could potentially be experienced.

The traditional view of orgasm is supported by recent evidence that women with complete SCI at T6 or above describe orgasmic experiences indistinguishable from their able-bodied counterparts.[33] The same researcher proposes that the preserved orgasmic sensory experience is partially derived from afferent autonomic innervation, which remains even in the presence of complete SCI.[34] In this group of women, only 17% of women with complete LMN dysfunction affecting the S2-S5 spinal segments were able to achieve orgasm, as compared with 59% of women with other levels and degrees of SCI, which suggests that orgasm is related to a preserved reflex.

In terms of changes in sexual desire after SCI, there are so many factors that can influence drive (depression, medical condition, lack of partner, disinterested partner, fear of incontinence, etc.) that it is difficult to say whether the biological component of drive remains intact or is altered. It is my experience that drive is initially lowered with the difficulties or preoccupation of adjusting to a new body, but within a year or two the drive often returns to preinjury level. The challenge for these men with SCI is to learn to interpret and incorporate new sexual potential that was previously not focused on. There is no evidence to suggest that there is any difference in the actual capacity to be mentally aroused pre- and post-SCI unless there is concomitant brain injury. It is likely that changes to overall libido are secondary to sexual function capacity and psychological

factors rather than to hormonal or organic brain influences.

Predictors of Ejaculation

My experience[35,36] with vibrostimulation for sperm retrieval consistently demonstrates several characteristic signs of ejaculation consistent with findings of others in the field[16,37–39]: episodic abdominal and leg contractions, generalized piloerection, scrotal wall retraction, and, usually, development of a rigid erection (ejaculation can occur with a flaccid or semi-erect penis). Ejaculation is imminent when the abdominal or leg spasms become more tonic and strong in nature (even to the point of discomfort or chest wall tightening). Erection rigidity peaks, the glans becomes acutely filled (if not so already), urethral spasms occur, the already elevated blood pressure acutely rises, and the pulse rate drops at ejaculation. Sometimes ejaculation is elusive despite these impending signs. To assist in triggering the reflex, one might extend the legs, abduct the thighs, lie flat versus upright (the latter is better for control of autonomic dysreflexia), have either a full or an empty bladder, and use erection enhancement such as sildenafil or intracavernosal injection. The vibrator placement and applied pressure is also important: I and others[20,21] have noted individual signature or trigger spots on the dorsal glans or frenulum that reliability evoke ejaculation in some men. Occasionally two vibrators may be needed to be applied at once to elicit ejaculation, but, due to the intensity of the stimulus, these trials need to be done under supervision of experienced clinicians only. Although it is well known that men with UMN lesions above T10 respond the best to vibrostimulation (these men will invariably have positive bulbocavernosis reflex), some men with incomplete lesions below T10 respond if there is enough nerve recruitment.[40] In the latter case, sympathomimetic drugs (i.e., pseudoephedrine) may help if there is no risk for autonomic dysreflexia. Arousal of the sympathetic nervous system is considered facilitatory for sexual arousal in certain patient subgroups[41]; this may be the case in men with SCI. Sometimes, men with incomplete lesions are not able to ejaculate in the clinical setting but can at home in a private sexual setting with the appropriate vibrator. Failure at the clinic is likely due to remaining tonic inhibitory control over the ejaculatory reflex.

Ejaculation can invoke negative consequences for men with SCI. Men whose injury is T6 or higher are at risk for autonomic dysreflexia with the stimulation required to reach ejaculation. Symptoms typically correlate with blood pressure, but there are exceptions. We have recorded a pressure of 220/113 in a premedicated patient with tetraplegia who was asymptomatic at ejaculation. This risk for dysreflexia during procedures can be lessened by the use of prophylactic antihypertensive drugs to blunt the high blood pressure response, reduction in the duration of vibratory stimulus (usually by applying a more aggressive amplitude or manual pressure), and removal of the vibrator at the point of impending ejaculation versus during ejaculation. Sometimes, the tendency to become dysreflexic attenuates with frequent, repeated vibrostimulation over months or years. Loss of spasticity from 1–48 hours is another side effect of ejaculation and can be seen with both vibrostimulation and electroejaculation.[42,43] Loss of spasticity may be symptomatically helpful for the patient or could hinder him, depending on the patient's

dependence on spasm for mobility and transfers. Some men experience unwanted effects on bladder management or bowel continence after sperm retrieval procedures, especially electroejaculation. Mobility, bladder, and bowel issues can all influence the man's capacity to be sexual. Other effects of sperm retrieval procedures are described elsewhere.[27,28,38,39]

Predictors of Orgasm

Compared to men with complete lesions, men with incomplete lesions who had not experienced orgasmic sensations with less genitally stimulating sexual practices had a greater chance of recognizing orgasmic sensations the first time while undergoing vibrostimulation. The unexpected potential for orgasm that is recognized in a clinical and sexually inhibiting setting can usually be enhanced by practice with the vibrator at home in a more conducive, private environment. In my experience, men with high complete lesions rarely report orgasmic experiences from vibrostimulation-induced ejaculation. Men who are technically incomplete at any level occasionally feel pleasurable or orgasmic sensations with vibrostimulation and sometimes even with electroejaculation, even if there is concomitant discomfort. It has yet to be proven whether men in this latter group have some preserved lower thoracic "coordination center" that can send information to stimulate a local cord "orgasmic" reflex. Because the chances of this are so small, patients in the Vancouver Sperm Retrieval Clinic, BC Center for Sexual Medicine, are told not to expect orgasmic sensations if they have not had them before with sexual activity. Lower expectations may actually enhance the chance of orgasmic sensation, if there is to be any, because self-consciousness about sexual experiences in a clinical setting induces cortical inhibition. However, if such sensual or orgasmic experiences do occur, they are framed in a positive light and are followed by discussions of how this may be incorporated in a personal setting with sexual activity.

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Erection, Ejaculation, and Orgasm After SCI: How Do They Intermingle?

Ejaculation is a separate neurological process from erection and/or orgasm, although there may be spinal, autonomic, and somatic pathways that are shared. Erection is primarily a parasympathetic event, but the thoracolumbar sympathetic pathways are involved. Under normal circumstances, the psychogenic and reflex arcs act synergistically to dictate the final erectile response via a common parasympathetic pathway.[44] The sympathetic nervous system is responsible for detumescence. After a complete UMN SCI, reflexogenic erections are usually possible because the sacral pathways are left intact. However, sympathetic pathways stemming from T10-L1 (thoracolumbar) have the capacity to maintain the erection independently if the sacral parasympathetic pathways are unavailable.[44] This is the explanation behind the psychogenic erectile capacity in men with LMN lesions affecting the

sacral reflexes who have lost their reflex erection.

The scientific literature on men with SCI corresponds to the experiences of women with SCI to a certain degree. Using the male analogy of pelvic arousal, it was felt that women with UMN lesions above the sacral area were capable of reflex lubrication in the same way men were capable of reflex erection and that women with sacral lesions were dependant on psychogenic lubrication as men were with psychogenic erections. Furthermore, there is some evidence that women with SCI and UMN dysfunction that affects the sacral spinal segments had preservation of their reflex genital vasocongestion.[45,46] In able-bodied women, an increase in sympathetic tone during sexual stimulation, through anxiety-provoking stimulation,[47] exercise,[48] or ephedrine,[49] contributes positively to both genital and subjective aspects of sexual response.[33] Psychogenic-mediated genital vasocongestion (as measured objectively by photoplethysmography) in women with SCI is associated with sensory preservation in the T11-L2 dermatomes where the sympathetic pathways controlling genital function originate.[33] Because able-bodied men are more subjectively aware of their arousal (erection) than women, the loss of this reliable and well-reinforced awareness after SCI may be one of the feedback loop deficiencies that prevent men with SCI from capitalizing on their orgasmic capabilities as well as women with SCI do. However, men's ultimate potential may be the same.

Discussions with patients in sexual rehabilitation reveal that men with SCI who are orgasmic either have incomplete lesions and/or have learned to incorporate or "ride" the sensory or cerebral afferents generated by stimulus of their genitalia, the hypersensitive area of their zone of injury, their nipples, breasts, or neck. Even visceral (and likely autonomic) inputs may contribute to this potential. It is this elevation of awareness that is the basis of using the senses as primary afferents ("sexual software" or the spiritual sciences[50]) that has expanded the pleasurable or even orgasmic scope for some men with SCI whose "neurological hardware" has been permanently disrupted.[51]

What is the relationship between erection and ejaculation in UMN and LMN injuries? Men with complete UMN injuries usually retain their reflex erection (it may be enhanced) due to the loss of supraspinal inhibitory control, but their ejaculatory capacity is severely compromised. These reflex erections can occur spontaneously, irrespective of sexual awareness. Reflex erections can be maintained through spasm, touch, sexual activity, or sperm retrieval methods, but they can be lost with too much stimulation, as can be seen with some positional changes in intercourse and even with vibrostimulation. Because of this, men with complete injury above T12 whose sacral reflexes are unimpaired often still need erection enhancement to improve the reliability and predictability of their reflex erections for sexual activity. These reflex erections respond well to such oral medications as the phosphodiesterase 5 (PDE5) inhibitors that are dependant on sufficient nitric oxide (NO) sources. Men with UMN lesions have this neurotransmitter available not so much through mental sexual arousal but through release of NO by touch stimulus to the penis (NO from both neuronal and endothelial sources enters the smooth muscle cell and produces the second messenger cGMP, which in turn relaxes the smooth muscle and initiates penile tumescence). PDE5 inhibitors inhibit the enzyme that de-

stroys cGMP, thereby allowing cGMP to remain longer in the cell, facilitating the erection.[52] Reflex erections are also sensitive to the intracavernosal injection of small doses of direct neurotransmitters such as prostaglandin E1 that increase the cAMP level in the smooth muscle cell[53] resulting in its relaxation and initiating erection. Although the studies of sildenafil did not show any significant effect on either the ability to ejaculate or sexual interest, it is my experience that some men with SCI who undergo vibrostimulation for sperm retrieval benefit from the maintenance of a rigid erection either through oral PDE5 inhibitors or intracavernosal injection. The continuing presence of a reliable erection may help some men with SCI ejaculate on any particular day if their ejaculation reflex is hard to attain, but this has not been verified with qualitative research. This theory is supported by one study where sildenafil treatment was associated with significant improvements in overall satisfaction, including frequency of ejaculation and orgasm.[54] Alternately, a rigid erection does not guarantee ejaculation in the men with complete UMN lesions who undergo vibrostimulation,[22] because many other factors, in particular the amplitude and speed of the vibrator,[38,55] are more predictive. Ejaculation to vibrostimulation has varying success rates; the best rate has been quoted as 95% in those men with injuries higher than T12.[38] However, rigidity of the penis and retraction of the penis away from the body have been helpful in eliciting ejaculation, possibly by potentiating the afferents of the sacral reflex. Sometimes the reflex erection will rapidly disappear with the vibratory stimulus, and the penis may remain flaccid until just prior to ejaculation where there may be acute filling of the glans alone.

Men with LMN lesions are dependent on their thoracolumbar centers to achieve psychogenic erections. Unfortunately, stimulation of the thoracolumbar pathways also elicits seminal emission. Men with LMN SCI who must maintain a high mental arousal to produce a psychogenic erection may find that an unwanted, dribbling emission has been triggered, resulting in detumescence. These men with LMN injury will also have a disruption of the second phase of ejaculation and poor somatic motor control of the striated perineal muscle group. Other men with incomplete LMN injuries may have more reliable ejaculation or even orgasmic sensation if there is enough nerve recruitment in the sacral area. Erection enhancement, usually in the form of direct neurotransmitter replacement (intracavernosal injection of prostaglandin), is often required to elicit an erection. In men with LMN lesions who cannot initiate an erection on their own, PDE5 inhibitors are less effectual,[56,57] because there is less NO at the nerve endings to enter the smooth muscle cell in the first place. Men with LMN complete lesions therefore have fewer successful options for erection enhancement.

Summary

Although erection physiology has been extensively studied in recent years, the scientific study of the sexual neurophysiology and other extraneural factors involved in ejaculation and orgasm in men with SCI is in its infancy. Further investigation into the neurophysiology of orgasm and its interpretation after SCI needs to be done. Future research should aim to provide a descriptive and physiological definition of orgasm after SCI that could then be applied to proper surveys

and well-designed clinical trials. Further study of the predictors of ejaculation and orgasm (physical examination markers, techniques of attainment, and medical or psychological factors that inhibit or facilitate the capacity) and reduction of negative consequences of sexual activity (such as autonomic dysreflexia and incontinence) would greatly assist in the sexual rehabilitation of men after SCI.

The span of sexual consequences after SCI is great, extending from the physiological loss of sexual function and fertility to the psychodynamic meaning of such changes. Men with SCI have to learn about their new sexual body. After injury, the perseverance or alteration of one sexual function may potentiate or hinder another. Men with SCI need to learn how to coordinate new body signs as facilitatory or inhibitory to their sexual functioning. As clinicians and researchers, we need to listen to what are, understandably, often vague or struggling descriptors of new sensations and responses that the patients experience. We also need to take the patients' pursuit of sexual pleasure and gratification as seriously as the more "medical" sexual function and fertility consequences.

There are three principles of sexual rehabilitation. The first is to *maximize the innate physiology*, even if that means experimenting with new mental and physical sources of sexual stimulation. A second principle is *adaptation to limitations that remain*, including acceptance of such things as erection enhancement and vibrostimulation. Men with SCI and their partners need to communicate with one another about their sexual experiences and live through these experimentations. This is the way the third principle, espousing a *positive outlook on future sexual potential*, is gained. Whether men with SCI will ultimately fulfill their erectile, ejaculatory, and orgasmic potential depends on the motivation and hope generated within themselves, by their partners, and by the interest of responsible health care clinicians who can direct and encourage them in this quest.

REFERENCES

1. Money J. Phantom orgasm in paraplegics. *Med Aspects Hum Sexuality*. 1970;Jan:90–97.
2. Herman M. Role of somasthetic stimuli in the development of sexual excitation in man: A preliminary paper. *Arch Neurol Psychiatr.* 1950;64:42.
3. Althof SH, Levine SB. Clinical approach to the sexuality of patients with spinal cord injury. *Urol Clin North Am.* 1993;20:527–534.
4. Szasz G. Sexual health care. In: Zejdlik C, ed. *Management of Spinal Cord Injury*. 2nd ed. Boston: Jones and Bartlett; 1992:175–201.
5. Ducharme SH, Gill KM. *Sexuality After Spinal Cord Injury: Answers to Your Questions.* Baltimore, MD: Paul H Brookes; 1997.
6. Higgins GE. Sexual response in spinal cord injured adults: A review of the literature. *Arch Sex Behav.* 1979;8:173–196.
7. Bors E, Comarr AE. Neurological disturbances of sexual function with special reference to 529 patients with spinal cord injury. *Urol Surv.* 1960;10:191–222.
8. Griffith ER, Tomko MA, Timms RJ. Sexual function in spinal cord injured patients: A review. *Arch Phys Med Rehabil.* 1973;54:539–543.
9. Biering-Sorenson F, Sonksen J. Penile erection in men with spinal cord or cauda equina lesions. *Semin Neurol.* 1992;12:98–105.
10. Comarr AE. Sexual function in patients with spinal cord injury. In: Pierce PS, Nickel VH, eds. *The Total Care of the Spinal Cord Injury.* Boston, MA: Little Brown; 1977:171–185.
11. Kennedy S, Over R. Psychophysiological assessment of sexual arousal of males with spinal cord injury. *Arch Sex Behav.* 1990;19:15–27.

12. Yarkony GM. Enhancement of sexual function and fertility in spinal cord injured males. *Am J Phys Med Rehabil*. 1990;69:2:81–87.
13. Alexander CJ, Sipski ML, Findley TW. Sexual activities, desire, and satisfaction in males pre- and post-spinal cord injury. *Arch Sex Behav*. 1993;22(3):217–228.
14. Phelps G, Brown M, Chen J, et al. Sexual experience and plasma testosterone levels in male veterans after spinal cord injury. *Arch Phys Med Rehabil*. 1983;64:47–52.
15. McKenna KE. Central nervous system pathways involved in the control of penile erection. *Annu Rev Sex Res*. 1999;10:157–183.
16. Shaban SF. Treatment of abnormalities of ejaculation. In: Lipshultz L, Howards S, eds. *Infertility in the Male*. St. Louis, MO: Mosby Year Book; 1991:409–427.
17. Oates RD, Kasabian NG. The use of vibratory stimulation of ejaculatory abnormalities. In: Whitehead D, Nagler H, eds. *Management of Impotence and Infertility*. Philadelphia, PA: Lippincott; 1994:314–320.
18. van Driel MF, Koeman M, Weijmar Schultz WCM, Mensink HJA. Orgasm after radical prostatectomy [Abstract D103]. *Int J Impot Res*. 1996;8(3):148.
19. Waldman TL, Eliasof B. Cancer. In: Sipski M, Alexander C, ed. *Sexual Function in People with Disability and Chronic Illness*. Gaithersburg, MD: Aspen Publishers;1997:337–354.
20. Smeltzer SC, Kelly CL. Multiple sclerosis. In: Sipski M, Alexander C, ed. *Sexual Function in People with Disability and Chronic Illness*. Gaithersburg, MD: Aspen Publishers; 1997:177–188.
21. Oates RD, Kasabian NG. The use of vibratory stimulation for ejaculatory abnormalities. In: Whitehead D, Nagler H, eds. *Management of Impotence and Infertility*. Philadephia, PA: Lippincott; 1994:314–320.
22. Szasz G, Carpenter C. Clinical observations in vibratory stimulation of the penis of men with spinal cord injury. *Arch Sex Behav*. 1989;18(6):461–474.
23. McCullough AR. Ejaculatory disorders. In: Mulcahy JJ, ed. *Male Sexual Function*. Totowa, NJ: Humana Press; 2001:351–370.
24. Chia M, Arava DA. *The Multiorgasmic Man*. New York: HarperSanFrancisco; 1996.
25. Green R. Sexual functioning in post-operative transsexuals: Male-to-female and female-to-male. *Int J Impot Res*. 1998;10(suppl 1);S22–S24.
26. Denys P, Mane M, Azouvi P, Chartier-Kastler E, Thiebaut T-B, Bussel B. Side effects of chronic intrathecal baclofen on erection and ejaculation in patients with spinal cord lesions. *Arch Phys Med Rehabil*. 1998;79:2–4.
27. Ohl DA. The use of electroejaculation for ejaculatory abnormalities. In: Whitehead ED, Nagler HM, eds. *The Management of Impotence and Infertility*. Philadelphia, PA: Lippincott; 1994:294–313.
28. Ohl D, Sonksen J. Penile vibratory stimulation and electroejaculation. In: Hellstrom W, ed. *Male Infertility and Sexual Dysfunction*. New York: Springer; 1997:219–229.
29. Kedia K, Markland C, Fraley E. Sexual function following high retroperitoneal lymphadenectomy. *J Urol*. 1975;114:237–239.
30. Marson L. Central nervous system control. In: Carson C, Kirby R, Goldstein I, eds. *Textbook of Erectile Dysfunction* Oxford: ISIS Medical Media; 1999:73–88.
31. Privat A, Mansour H, Rajaofetra N, Geffard M. Intraspinal transplants of serotonergic neurons. In: *The Adult Rat Brain Research Bulletin*. Vol. 22. Tarrytown, NY: Pergamon Press; 1989:123–129.
32. Whipple B, Gerdes CA, Komisaruk BR. Sexual response to self-stimulation in women with complete spinal cord injury. *J Sex Res*. 1996;33:231–240.
33. Sipski ML, Alexander CJ, Rosen RC. Orgasm in women with spinal cord injuries: A laboratory based assessment. *Arch Phys Med Rehabil*. 1995;76:1097–1102.
34. Sipski ML, Alexander CJ, Rosen R. Sexual arousal and orgasm in women: Effects of spinal cord injury. *Ann Neurol*. 2001;49:35–44.
35. Elliott S, Fluker M. Fertility options for men with ejaculatory disorders. *J Soc Obstet Gynaecol Can*. 2000:22(1):26–32.
36. Elliott S, Szasz G, Nigro M, Zouves C, Rines B. Vibrostimulation and electroejaculation: The Vancouver experience. *J Soc Obstet Gynaecol Can*. 1993;15(4):390–404.
37. Ohl D, Sonksen J. Penile vibratory stimulation and electroejaculation. In: Hellstrom W, ed. *Male Infertility and Sexual Dysfunction*. New York: Springer; 1997:219–229.
38. Sonksen J, Biering-Sorensen F, Kristensen JK. Ejaculation induced by penile vibratory stimulation in men with spinal cord injuries: The importance of vibratory amplitude. *Paraplegia*. 1994;32:651–660.
39. Brackett NL, Ferrel SM, Aballa TC, et al. An

analysis of 653 trials of penile vibratory stimulation in men with spinal cord injury. *J Urol*. 1998;159:1931–1934.
40. Beckerman H, Becher J, Lankhorst GJ. The effectiveness of vibratory stimulation in anejaculatory men with spinal cord injury. *Paraplegia*. 1993;31:689–699.
41. Meston CM. Sympathetic nervous system activity and female sexual arousal. *Am J Cardiol*. 2000;86(suppl):30F–34F.
42. Halstead LS, Seager SWJ. The effects of rectal probe electrostimulation on spinal cord injury spasticity. *Paraplegia*. 1991;29:43–47.
43. Stein R. Letters to the editor. *Paraplegia*. 1991;29:495–497.
44. Chuang AT, Steers WD. Neurophysiology of penile erection. In: Carson C, Kirby R, Goldstein I, eds. *Textbook of Erectile Dysfunction*. Oxford, UK: ISIS Medical Media, 1999:59–72.
45. Sipski ML, Alexander CJ, Rosen RC. Physiological parameters associated with psychogenic sexual arousal in women with complete spinal cord injuries. *Arch Phys Med Rehabil*. 1995;76:811–818.
46. Sipski ML. Spinal cord injury and sexual function: An educational model. In: Sipski M, Alexander C, ed. *Sexual Function in People with Disability and Chronic Illness*. Gaithersburg, MD: Aspen Publishers; 1997:149–176.
47. Palace EM, Gorzalka BB. The enhancing effects of anxiety on arousal in sexually dysfunctional and functional women. *J Abnorm Psychol*. 1990;99:403–411.
48. Meston CM, Gorazalka BB. The differential effects of sympathetic activation on sexual arousal in sexually functional and dysfunctional women. *J Abnorm Psychol*. 1996; 34:143–148.
49. Meston CM, Heiman JR. Ephedrine-activated physiological sexual arousal in women. *Arch Gen Psychiatry*. 1998;55:652–656.
50. Douglas N. *Spiritual Sex*. New York: Pocket Book; 1997.
51. Tepper M. Personal communication, 2001. sexualhealth.com (owned and operated by the Sexual Health Network, Inc; Pennsylvania, PA).
52. Naylor AM. Nitric oxide as a neurotransmitter in the corpus cavernosum. In: Carson DD, Kirby RS, Goldstein I, ed. *Textbook of Erectile Dysfunction*. Oxford, UK: ISIS Medical Media; 1999:117–122.
53. Spahn M, Manning M, Juenemann KP. Intracavernosal therapy. In: Carson DD, Kirby RS, Goldstein I, ed. *Textbook of Erectile Dysfunction*. Oxford, UK: ISIS Medical Media; 1999:345–354.
54. Giuliano FG, Hultling C, Masry E, et al. Randomized trial of sildenafil for the treatment of erectile dysfunction in spinal cord injury. *Ann Neurol*. 1999;46:15–21.
55. Brackett NL, Sonksen J, Lynne CM. Improved success rate in ejaculation of spinal cord injured men with high-amplitude (FERTI-CARE clinic) versus low-amplitude vibrator [abstract]. In: *ASRM Annual Meeting Program Supplement*. Birmingham, AL: ASRM; 1995:S17.
56. Ramos AS, Vidal J, Jauregui ML, et al. Efficacy, safety and predictive factors of therapeutic success with sildenafil for erectile dysfunction in patients with different spinal cord injuries. *Spinal Cord*. 2001;39:637–643.
57. Mayton MC, Derry FA, Dunsmore WW. A two-part study of sildenafil (Viagra) in men with erectile dysfunction caused by spinal cord injury. *Spinal Cord*. 1999;37:110–118.