

## Disorders of Orgasm and Ejaculation in Men

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Summary of Committee. For the complete report please refer to *Sexual Medicine: Sexual Dysfunctions in Men and Women*, edited by T.F. Lue, R. Basson, R. Rosen, F. Giuliano, S. Khoury, F. Montorsi, Health Publications, Paris 2004.

### ABSTRACT

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**Introduction.** Ejaculatory/orgasmic disorders, common male sexual dysfunctions, include premature ejaculation, inhibited ejaculation, anejaculation, retrograde ejaculation and anorgasmia.

**Aim.** To provide recommendations/guidelines concerning state-of-the-art knowledge for management of ejaculation/orgasmic disorders in men.

**Methods.** An International Consultation in collaboration with the major urology and sexual medicine associations assembled over 200 multidisciplinary experts from 60 countries into 17 committees. Committee members established specific objectives and scopes for various male and female sexual medicine topics. The recommendations concerning state-of-the-art knowledge in the respective sexual medicine topic represent the opinion of experts from five continents developed in a process over a 2-year period. Concerning the Disorders of Ejaculation/Orgasm in Men Committee, there were nine experts from six countries.

**Main Outcome Measure.** Expert opinion was based on grading of evidence-based medical literature, widespread internal committee discussion, public presentation and debate.

**Results.** Premature ejaculation management is dependent upon etiology. When secondary to ED, etiology-specific treatment is employed. When lifelong, initial pharmacotherapy (SSRI, topical anesthesia, PDE5 inhibitors) is appropriate. When associated with psychogenic/relationship factors, behavioral therapy is indicated. When acquired, pharmacotherapy and/or behavioral therapies are preferred. Retrograde ejaculation, diagnosed with spermatozoa and fructose in centrifuged post-ejaculatory voided urine, is managed by education, patient reassurance, pharmacotherapy or bladder neck reconstruction. Men with anejaculation or anorgasmia have a biologic failure of emission and/or psychogenic inhibited ejaculation. Men with age-related penile hypoanesthesia should be educated, reassured and be instructed in revised sexual techniques which maximize arousal.

**Conclusions.** More research is needed in understanding management of men with ejaculation/orgasmic dysfunction.

**Key Words.** Ejaculation; Premature Ejaculation; Retrograde Ejaculation; Inhibited Ejaculation; Anejaculation; Selective Serotonin Re-Uptake Inhibitors; Psychotherapy

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*Conflicts of Interest.* Chris McMahon, Paid consultant and clinical trial investigator to Pfizer, Icos Lilly, Bayer, Glaxo-SmithKline, American Medical Systems, Johnson & Johnson.

## Introduction

Ejaculatory dysfunction is one of the most common sexual disorders in men, and extends from premature ejaculation, through inhibited ejaculation to a complete inability to ejaculate, anejaculation, and includes retrograde ejaculation.

Orgasm and ejaculation constitute the final phase of the sexual response cycle. There are three basic mechanisms involved in normal ante-grade ejaculation—emission, ejection and orgasm [1]. Ejaculatory dysfunction can result from disruption at any point in this cascade of events.

Ejaculation is a reflex comprising sensory receptors and areas, afferent pathways, cerebral sensory areas, cerebral motor centers, spinal motor centers and efferent pathways. The ejaculatory reflex is predominantly controlled by a complex interplay between central serotonergic and dopaminergic neurons with secondary involvement of cholinergic, adrenergic, nitrenergic, oxytocinergic and GABAergic neurons. Seminal emission and ejection are integrated into the complex pattern of copulatory behavior by several forebrain structures including the medial preoptic area (MPOA) and the nucleus paragigantocellularis (nPGi). Descending serotonergic pathways from the nPGi to the lumbosacral motor nuclei tonically inhibit ejaculation. Disinhibition of the nPGi by the MPOA results in ejaculation. Several brain areas are activated after ejaculation by ascending fibers from the spinal cord and may have a possible role in satiety and the post-ejaculatory refractory time.

Male rat studies demonstrate that serotonin and 5-HT receptors are involved in the ejaculatory process. As far as is currently known, 5-HT<sub>2C</sub> and 5-HT<sub>1A</sub> receptors determine the speed of ejaculation. Stimulation of 5-HT<sub>2C</sub> receptors with non-selective 5-HT<sub>2C</sub> agonists delays ejaculation in male rats whereas stimulation of post-synaptic 5-HT<sub>1A</sub> receptors resulted in shorter ejaculation latency [2]. Administration of selective serotonin re-uptake inhibitors (SSRIs) results in active blockade of presynaptic membrane 5-HT transporters, and the resultant higher synaptic cleft levels of 5-HT activate post-synaptic 5-HT<sub>2C</sub> and 5-HT<sub>1A</sub> receptors and delay ejaculation [3,4].

Waldinger et al. formulated the hypothesis that men with premature ejaculation have hyposensitivity of 5-HT<sub>2C</sub> and/or hypersensitivity of 5-HT<sub>1A</sub> receptor [3,5,6]. The hypothesis that activation of post-synaptic 5-HT receptors delays ejaculation is supported by numerous studies in

humans with different SSRIs. However, in these studies it is not obvious whether 5-HT<sub>2C</sub> and 5-HT<sub>1A</sub> receptors subtypes are also involved in human ejaculation since SSRI treatment activates many different post-synaptic subtype receptors. Further studies with selective 5-HT<sub>2C</sub> and 5-HT<sub>1A</sub> agonist and antagonists are required to further unravel the neuropharmacological mechanisms of ejaculation.

## Premature Ejaculation (PE)

Medical literature contains several one-dimensional and multidimensional operational definitions of premature ejaculation. The lack of agreement as to what constitutes premature ejaculation has hampered basic and clinical research into the etiology and management of this condition. Quantitative measures of intercourse such as the intravaginal ejaculatory latency time (IELT), the number of thrusts between penetration and ejaculation, the extent of partner sexual satisfaction, and the patient's assessment of his voluntary control over ejaculation have been described. Each of the three criteria above has been operationalized, although not always with consistency [7].

Operationalization of PE using the quantifiable and objective number of intravaginal thrusts between penetration and ejaculation has been reported by several authors [8–11], however these definitions were subjective, and not supported by normative data. Operationalization of PE using the length of time between penetration and ejaculation, the intravaginal ejaculatory latency time (IELT), forms the basis of most current clinical studies on PE. There is considerable variance of the latencies used to identify men with PE with IELTs ranging from 1 to 7 minutes and none of the definitions offer any supportive rationale for their proposed cut-off time or normative data [12–15]. The inability to control and defer ejaculation until the female partner was sexually satisfied on at least 50% of intercourse attempts was proposed as a definition of PE by Masters and Johnson [11]. An inherent problem exists in defining a man as dysfunctional based on the sexual responsiveness of his partner, as only 30% of women achieve orgasm during sexual intercourse regardless of the extent of their partner's ejaculatory control and latency.

The lack of a reliable operational definition for PE has severely limited clinical research into the understanding of PE as studies that fail to define PE offer meaningless or difficult to interpret

results. The lack of a universally accepted operationalized definition makes comparison of different studies difficult or impossible as experimental group subjects in one study may very well have been placed in the control group of a second study. The following multivariate definition of PE is proposed: "Premature ejaculation is persistent or recurrent ejaculation with minimal stimulation before, on, or shortly after penetration, and before the person wishes it, over which the sufferer has little or no voluntary control which causes the sufferer and/or his partner bother or distress."

Premature ejaculation may be classified into various subtypes based on the developmental history and response characteristics. Acquired PE and/or situational PE suggest a psychological etiology and behavioral therapy as the most appropriate initial therapy and/or relationship counseling whereas lifelong and/or global PE suggest a biogenic etiology and pharmacologic treatment as the most appropriate initial therapy. A significant number of men with PE also report having problems achieving and/or maintaining an erection, estimated as high as 30% [16].

Historically, attempts to explain the etiology of premature ejaculation have included a diverse range of biogenic and psychological theories (Table 1). Most of these proposed etiologies are not evidence based and are speculative at best. The lack of an operationalized definition for PE, and the presence of methodological problems related to the inadequate definitions used, is a common flaw in the majority of these studies.

### Treatment Of Premature Ejaculation

Men with premature ejaculation should be evaluated with a detailed medical and sexual history, a physical examination and appropriate investigations to establish the true presenting complaint and identify obvious biological causes such as genital or lower urinary tract infection (Figure 1).

**Table 1** Proposed etiologies of premature ejaculation

Psychogenic	Anxiety
	Early sexual experience
	Frequency of sexual intercourse
	Ejaculatory control techniques
	Evolutionary
	Psychodynamic theories
Biological	Penile hypersensitivity
	Hyper-excitability ejaculatory reflex
	Arousability
	Endocrinopathy
	Genetic predisposition
	5-HT receptor dysfunction

In many relationships premature ejaculation causes few if any problems. In others, the couple may reach an accommodation of the problem through various strategies—young men with a short refractory period may often experience a second and more controlled ejaculation during an episode of lovemaking. Frequently, however, premature ejaculation eventually leads to significant problems in the relationship with partners regarding the man as selfish and developing a pattern of sexual avoidance. This only worsens the severity of the prematurity on the occasions when intercourse does occur.

The cornerstones of behavioral treatment are the Seman's "stop-start" maneuver and its modification proposed by Masters and Johnson, the squeeze technique. Both are based on the theory that premature ejaculation occurs because the man fails to pay sufficient attention to pre-orgasmic levels of sexual tension [11,17]. As most men with premature ejaculation are aware of their anxiety and the sources of that anxiety tend to be relatively superficial, treatment success with these behavioral approaches is relatively good in the short term but convincing long-term treatment outcome data are lacking [18,19].

Pharmacological modulation of ejaculatory threshold represents a novel and refreshing approach to the treatment of premature. The introduction of the selective serotonin reuptake inhibitors (SSRIs) meant a revolutionary change in the approach to and treatment of premature ejaculation. Selective serotonin reuptake inhibitors encompass five compounds (citalopram, fluoxetine, fluvoxamine, paroxetine and sertraline) with a similar pharmacological mechanism of action. Although the methodology of the initial drug treatment studies was rather poor, recent double blind placebo-controlled studies demonstrated the efficacy of SSRIs and clomipramine in delaying ejaculation [20–24]. In spite of a development towards more evidence based drug treatment research, the majority of studies still lack adequate design and methodology [25]. There are three drug treatment strategies to treat premature ejaculation: 1) daily treatment with serotonergic antidepressants; 2) as-needed treatment with antidepressants; and 3) topical local anaesthetics.

Examining daily treatment with serotonergic antidepressants paroxetine, clomipramine, sertraline and fluoxetine, a meta-analysis of drug treatment studies demonstrates that paroxetine exerts the strongest ejaculation delay [60]. Ejaculation delay usually occurs within 5–10 days but may

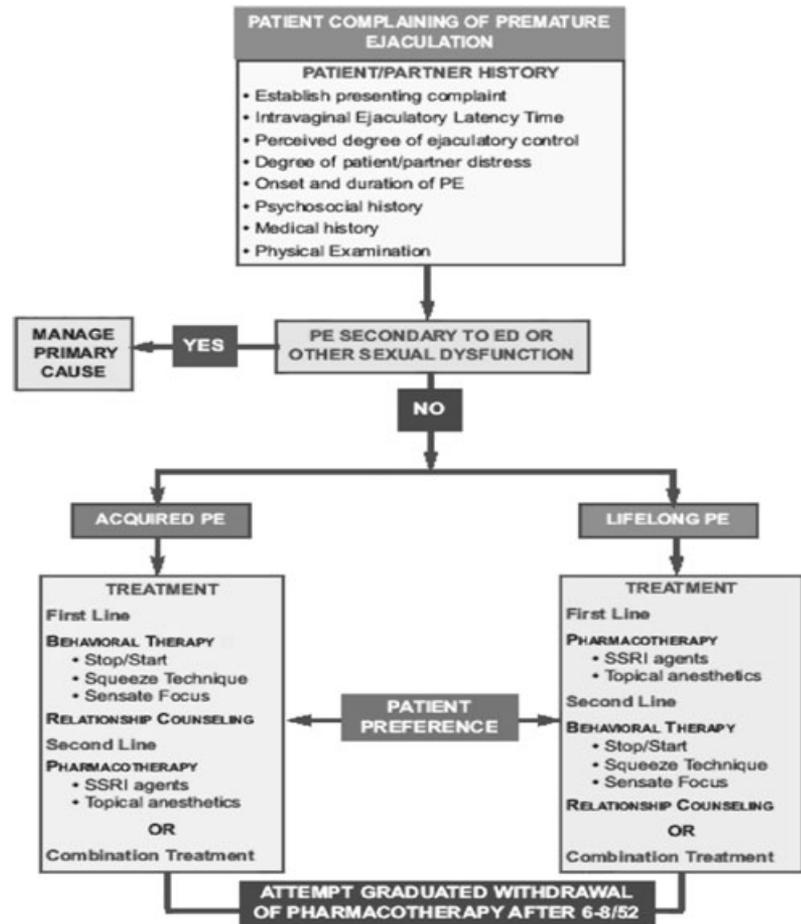


Figure 1

occur earlier. Adverse effects are usually minor, start in the first week after intake, gradually disappear within 2–3 weeks and include fatigue, yawning, mild nausea, loose stools or perspiration. Diminished libido or mild erectile dysfunction are infrequently reported.

Administration of antidepressants as needed, paroxetine, sertraline, fluoxetine and clomipramine 4–6 hours prior to intercourse, is efficacious and well tolerated, is associated with less ejaculatory delay than daily treatment and may be combined with either an initial trial of daily treatment or concomitant low dose daily treatment [26–28].

The use of topical local anesthetics such as lignocaine and/or prilocaine as a cream, gel or spray is well established. They are moderately effective in retarding ejaculation, but do so at the price of possibly causing significant penile hypoesthesia and possible transvaginal absorption, resulting in vaginal numbness and resultant female anorgasmia unless a condom is used [29–31].

Several authors have reported their experience with sildenafil citrate as a treatment for PE

[32–34]. It is unlikely that phosphodiesterase inhibitors have a significant role in the treatment of PE with the exception of men with acquired PE secondary to comorbid ED.

#### Inhibited Ejaculation, Anejaculation and Anorgasmia

The Diagnostic and Statistical Manual of Mental Disorders defines inhibited ejaculation (IE) as the persistent or recurrent difficulty, delay in, or absence of attaining orgasm following sufficient sexual stimulation, which causes personal distress [35]. Inhibited ejaculation or anejaculation can be classified as either a lifelong or acquired, or as global or situational. Any single or combination of psychological or medical disease, surgical procedure or drug which interferes with either central control of ejaculation, the afferent or efferent nerve supply to the vas, bladder neck, pelvic floor or penis, can result in inhibited ejaculation, anejaculation and anorgasmia (Table 2).

Inhibited ejaculation, like other sexual dysfunctions, is more prevalent as men age [36]. The pro-

**Table 2** Causes of inhibited ejaculation, anejaculation and anorgasmia

Psychogenic	Inhibited ejaculation
Congenital	Mullerian duct cyst Wolfian duct abnormality Prune belly syndrome
Anatomic Causes	Transurethral resection of prostate Bladder Neck Incision
Neurogenic Causes	Diabetic autonomic neuropathy Spinal cord injury Radical prostatectomy Proctocolectomy Bilateral sympathectomy Abdominal aortic aneurysmectomy Para-aortic lymphadenectomy
Infective	Urethritis Genitourinary tuberculosis Schistosomiasis
Endocrine	Hypogonadism Hypothyroidism
Medication	Alpha-methyl dopa Thiazide diuretics Tricyclic and SSRI antidepressants Phenothiazine Alcohol abuse

gressive loss of the fast conducting peripheral sensory axons which begins to be apparent in the third decade of life, and the dermal atrophy, myelin collagen infiltration and pacinian corpuscle degeneration observed in older men, may result in a degree of age-related degenerative penile hypoesthesia and difficulty in achieving the ejaculatory threshold.

IE may be associated with cultural and religious beliefs, concurrent psychopathology such as unconscious aggression and unexpressed anger, insufficient sexual arousal, preconditioning for inhibited ejaculation due to a preference for masturbation over partnered sex, fear of pregnancy or sexually transmissible disease [11,37–41]. Apfelbaum observed that some males achieve erections sufficient for intercourse despite a relative absence of subjective arousal and incorrectly regard themselves as ready for sex and capable of achieving orgasm [37]. This same process is the likely cause of increased anecdotal clinical reports of inhibited ejaculation in patients using pharmacologic treatments for ED [41,42].

The ability to ejaculate is severely impaired by spinal cord injury (SCI) and is dependent upon the level and completeness of SCI [43]. Unlike erectile capacity, the ability to ejaculate increases with descending levels of spinal injury. Less than 5% of patients with complete upper motor neuron lesions retain the ability to ejaculate.

### Treatment of Inhibited Ejaculation, Anejaculation and Anorgasmia

Men with inhibited ejaculation, anejaculation and/or anorgasmia should be evaluated with a detailed medical and sexual history, a physical examination and appropriate imaging and/or electrophysiological investigations to establish the true presenting complaint, identify obvious biological causes such as medication, diabetes mellitus or recent pelvic surgery, and uncover sufficient detail to establish the optimal treatment plan. The exact site of ejaculatory duct obstruction may be identified by transrectal ultrasonography, vasography or by percutaneous puncture of the seminal vesicles. Four neurophysiological tests are routinely used—pudendal somatosensory evoked potentials, pudendal motor evoked potentials, sacral reflex arc testing and sympathetic skin responses. Treatment should be etiology specific and address the issue of infertility in men of a reproductive age.

Although multiple psychodynamic and behavioral treatments for IE have been suggested, empirical evidence to support treatment efficacy is lacking [11,44–49]. Most reports are uncontrolled case reports with treatment ranging from a few brief sessions of sex education to the nearly 2 years of multiple-modality treatment in more complex multiple etiologic cases.

There are multiple reports in the literature of the use of a variety of drugs in the treatment of inhibited ejaculation [50–59]. The drugs facilitate ejaculation by either a central dopaminergic or anti-serotonergic mechanism of action. There are no published placebo controlled studies and most reports are anecdotal case reports/series that deal with the treatment of SSRI induced ejaculatory dysfunction (Table 3). While medical treatment may not always produce normal ejaculation, it may convert a patient with lack of emission into one with retrograde ejaculation.

### Conclusions

Men with premature ejaculation secondary to erectile dysfunction, other sexual dysfunction or genitourinary infection should receive appropriate etiology specific treatment. Men with lifelong premature ejaculation should be initially managed with pharmacotherapy. Men with significant contributing psychogenic or relationship factors may benefit from concomitant behavioral therapy. Recurrence of premature ejaculation is highly likely to occur following withdrawal of treatment. Men with acquired premature ejaculation can be

**Table 3** Adjunctive drug therapy for SSRI-induced sexual dysfunction

Drug	Symptom	Dosage	
		As needed	Daily
Amantadine	Anorgasmia Decreased libido Erectile dysfunction	100–400 mg (for 2 days prior to coitus)	75–100 mg bid or tid
Bupropion	Anorgasmia	75–150 mg	75 mg bid or tid
Buspiron	Anorgasmia Decreased libido Erectile dysfunction	15–60 mg	5–15 mg bid
Cyproheptadine	Anorgasmia Decreased libido Erectile dysfunction	4–12 mg	On demand
Yohimbine	Anorgasmia Decreased libido Erectile dysfunction	5.4–10.8 mg	5.4 mg tid

treated with pharmacotherapy and/or behavioral therapy according to patient/partner preference. Restoration of ejaculatory control in men with acquired premature ejaculation is likely to occur following completion of treatment but is the exception in men with lifelong premature ejaculation. Behavioral therapy may augment pharmacotherapy to enhance relapse prevention.

Men who never achieve orgasm and ejaculation are suffering from either a biogenic failure of emission and/or psychogenic inhibited ejaculation. Management involves identification of the etiology and disease specific treatment. Men who occasionally achieve orgasm and ejaculation are usually suffering from psychogenic inhibited ejaculation or penile hypoanesthesia secondary to age-related degeneration of the afferent penile nerves. The former is managed with behavioral therapy and/or psychotherapy. Men with age-related penile hypoanesthesia should be educated, reassured and be instructed in revised sexual techniques which maximize arousal.

The majority of men who always achieve orgasm but never experience prograde ejaculation or have a greatly reduced prograde ejaculatory volume have retrograde ejaculation. The presence of spermatozoa and fructose in centrifuged post-ejaculatory voided urine confirms the diagnosis. Management involves education and reassurance of the patient, pharmacotherapy or, in rare cases, bladder neck reconstruction. The absence of spermatozoa suggests congenital absence or agenesis of the testis or vas/vasa or acquired ejaculatory duct obstruction. Management involves investigation by ultrasonic or radiological imaging to identify the site of obstruction and disease specific treatment.

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