Standard Operating Procedures in the Disorders of Orgasm and Ejaculation

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ABSTRACT

Introduction. Ejaculatory/orgasmic disorders are common male sexual dysfunctions and include premature ejaculation (PE), inhibited ejaculation, anejaculation, retrograde ejaculation, and anorgasmia.

Aim. To provide recommendations and guidelines of the current state-of-the-art knowledge for management of ejaculation/orgasmic disorders in men as standard operating procedures (SOPs) for the treating health care professional.

Methods. The International Society of Sexual Medicine Standards Committee assembled over 30 multidisciplinary experts to establish SOPs for various male and female sexual medicine topics. The SOP for the management of disorders of orgasm and ejaculation represents the opinion of four experts from four countries developed in a process over a 2-year period.

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

Results. PE management is largely dependent upon etiology. Lifelong PE is best managed with PE pharmacotherapy (selective serotonin reuptake inhibitors and/or topical anesthetics). The management of acquired PE is etiology specific and may include erectile dysfunction (ED) pharmacotherapy in men with comorbid ED. All men seeking treatment for PE should receive basic psychosexual education. Graded behavioral therapy is indicated when psychogenic or relationship factors are present and is often best combined with PE pharmacotherapy in an integrated treatment program. Delayed ejaculation, anejaculation, and/or anorgasmia may have a biogenic and/or psychogenic etiology. Men with age-related penile hypoanesthesia should be educated, reassured, and instructed in revised sexual techniques which maximize arousal. Retrograde ejaculation is managed by education, patient reassurance, and pharmacotherapy.

Conclusions. Additional research is required to further the understanding of the disorders of ejaculation and orgasm.

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Key Words. Premature Ejaculation; Delayed Ejaculation; Anejaculation; Retrograde Ejaculation; Selective Serotonin Reuptake Inhibitor; Behavioral Therapy

Introduction

Ejaculatory dysfunction is one of the most common male sexual disorders. The spectrum of ejaculatory dysfunction extends from premature ejaculation (PE), through delayed ejaculation (DE), to a complete inability to ejaculate (known as anejaculation) and includes retrograde ejaculation.

The Anatomy and Physiology of the Ejaculatory Response

The ejaculatory reflex comprises sensory receptors and areas, afferent pathways, cerebral sensory areas, cerebral motor centers, spinal motor centers, and efferent pathways. Neurochemically, this reflex involves a complex interplay between central serotonergic and dopaminergic neurons,

with secondary involvement of cholinergic, adrenergic, oxytocinergic, and gamma aminobutyric acid (GABA) neurons.

Based upon functional, central, and peripheral mediation, the ejaculatory process is typically subdivided into three phases: emission, ejection (or penile expulsion), and orgasm. Emission consists of contractions of seminal vesicles (SVs) and the prostate, with expulsion of sperm and seminal fluid into the posterior urethra, and is mediated by sympathetic nerves (T10 to L2). Ejection is mediated by somatic nerves (S2–S4) and involves pulsatile contractions of the bulbocavernosus and pelvic floor muscles together with relaxation of the external urinary sphincter. Ejection also involves a sympathetic spinal cord reflex upon which there is limited voluntary control. The bladder neck closes to prevent retrograde flow; the bulbocavernosus, bulbospongiosus, and other pelvic floor muscles contract rhythmically, and the external urinary sphincter relaxes. Intermittent contraction of the urethral sphincter prevents retrograde flow into the proximal urethra [1]. Orgasm is the result of cerebral processing of pudendal nerve sensory stimuli resulting from increased pressure in the posterior urethra, sensory stimuli arising from the verumontanum, and contraction of the urethral bulb and accessory sexual organs.

Many neurotransmitters are involved in the control of ejaculation, including dopamine, norepinephrine, serotonin, acetylcholine, oxytocin, GABA, and nitric oxide (NO) [2]. Of the many studies conducted to investigate the role of the brain in the development and mediation of sexual functioning, dopamine and serotonin have emerged as essential neurochemical factors. Whereas dopamine promotes seminal emission/ejaculation via D2 receptors, serotonin is inhibitory. Serotonergic neurons are widely distributed in the brain and spinal cord and are predominantly found in the brainstem, raphe nuclei, and the reticular formation. Currently, multiple serotonin (5-hydroxytryptamine [5-HT]) receptors have been characterized, e.g., 5-HT1a, 5-HT1b, 5-HT2a, 5-HT2b, etc. [3]. Stimulation of the 5-HT2C receptor with 5-HT2C agonists results in delay of ejaculation in male rats, whereas stimulation of postsynaptic 5-HT1A receptors results in shortening of ejaculation latency [4], leading to the hypothesis that men with PE may have hyposensitivity of 5-HT2C and/or hypersensitivity of the 5-HT1A receptor [5,6].

PE

Definition of PE

There are multiple definitions of PE (Table 1). The first contemporary multivariate evidence-based definition of lifelong PE was developed in 2008 by a panel of international experts, convened by the International Society for Sexual Medicine (ISSM), who agreed that the diagnostic criteria necessary to define PE are time from penetration to ejaculation, inability to delay ejaculation, and negative personal consequences from PE. This panel defined lifelong PE as a male sexual dysfunction characterized by "... ejaculation which always or nearly always occurs prior to or within about one minute of vaginal penetration, the inability to delay ejaculation on all or nearly all vaginal penetrations, and the presence of negative personal consequences, such as distress, bother, frustration and/or the avoidance of sexual intimacy" [7].

This definition is supported by evidence from several controlled clinical trials that suggest that 80–90% of men with lifelong PE ejaculate within 60 seconds and the remaining 10–20% within 2 minutes (Figure 1) [17,18]. This definition should form the basis for the official diagnosis of lifelong PE. It is limited to heterosexual men engaging in vaginal intercourse as there are few studies available on PE research in homosexual men or during other forms of sexual expression. Preliminary recommendations of the American Psychiatric Association's DSM-V committee suggest a definition which parallels the definition recently adopted by the ISSM [19]. The panel concluded that there is insufficient published evidence to propose an evidenced-based definition of acquired PE (A-PE) [7]. However, recent data suggest that men with A-PE have similar intravaginal ejaculation latency times (IELTs) and report similar levels of ejaculatory control and distress, suggesting the possibility of also a single unifying definition of PE [20].

Classifications of PE

In 1943, Schapiro proposed a distinction of PE into types A and B [21]. Men with type B have always suffered from a very rapid ejaculation (or short latency), whereas in type A, the rapid ejaculation develops later in life and is often associated with erectile dysfunction (ED). In 1989, these types were, respectively, referred to as lifelong (primary) and acquired (secondary) PE [22]. Over the years, other attempts have been made to identify various classifications of PE, including several that have been incorporated into PE definitions