

Clinical physiology and pathophysiology of ejaculation and orgasm

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ABSTRACT When assessing ejaculatory or orgasmic concerns, the diagnosis can be made by assessing the individual central and peripheral components of the sexual response as well as the capacity for release of the tonic central inhibition that controls the ejaculation reflex. The causes of ejaculatory disorders are many and varied, but the negative effects for patients are consistent. In this first of two articles in this issue, Dr Elliott outlines the physiologic basis of ejaculatory or orgasmic disorders by providing clinical definitions and diagnostic categories that will enhance the primary care clinician's ability to deal with these conditions.

Within the spectrum of male sexual disorders, it is most commonly erectile dysfunction that brings patients into the physician's office.¹ However, ejaculatory problems, with fast or premature ejaculation being the most prominent, are experienced by up to 40% of men in surveys.² Premature ejaculation alone affects an estimated 30% of men.³ As Dr Basson noted in February in the second article in this series,⁴ some important sexual history-taking questions should explore all areas of the sexual response surrounding the presenting complaint—sexual interest, arousal, ejaculation, orgasm, and sexual pain concerns.

Most men present to the primary care office with an ejaculatory complaint that involves psychosexual issues rather than a purely medical condition. In general, younger men complain of premature ejaculation, and older men present with delayed ejaculation or inability to ejaculate. In a recent study, the prevalence of men who felt they climaxed too early, regardless of age, was about 30%, compared with less than 10% of men who were unable to achieve orgasm.⁵

Conditions seen in the primary care office that are primarily medical issues include infertility, pain with ejaculation, and hematospermia. After initial investigations, these ejaculatory problems may require referral to a specialist. Other ejaculation problems involve anejaculation, anorgasmia, and anhedonic ejaculation; these are much less common but debilitating when present.

Male sexual response

The commonly recognized order of male sexual response following sexual stimulation is erection, emission, and ejaculation accompanied by orgasm

Practice Tips

1 Assess ejaculatory and orgasmic concerns as entities separate from erection, but remain cognizant of changes in sexual interest or erection that may further clarify the etiology of the disorder.

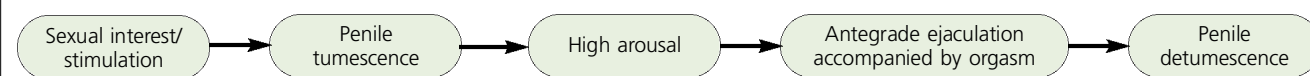
1 Identify if there is a reversible cause of dysfunction and formulate a differential diagnosis that will dictate further investigations or referral.

1 After a thorough assessment, alter those factors which may be contributing to the disorder and continue to treat to your level of comfort and expertise.

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FIGURE 1



Male sexual response

Clinical definitions

Ejaculation that occurs without orgasm and without pleasure is called *anhedonic ejaculation* or *anorgasmic ejaculation*; orgasm that occurs without ejaculation (“dry orgasm”) is usually neurogenic or anatomical in etiology. Orgasm and ejaculation may also occur out of sync—in such cases ejaculate dribbles out via gravity after orgasm versus pulsatile ejaculation occurring at the time of orgasm.

Inability to reach either orgasm or ejaculation because of a neurologic problem is usually referred to as *anejaculation*. Difficulty or failure to reach orgasm and ejaculation in those without a clear neurologic etiology is called inhibited, delayed, or *retarded ejaculation*; this is also sometimes referred to as *anorgasmia* if orgasm is not attainable at all. Personal distress from persistently reaching orgasm too quickly, defined as before or very soon after intercourse is initiated, in a partnered situation is interchangeably referred to as rapid, fast, or *premature ejaculation*.

Classification

Ejaculation disorders associated with infertility are classified according to their prospective sexual or sperm retrieval therapies.

Fast ejaculation and inhibited ejaculation, as well as any other sexual disorder functionally interfering with intravaginal semen deposition, are classified under sexual dysfunction. Failure of seminal emission and/or propulsatile ejaculation, retrograde ejaculation, or *anejaculation* all fall under neurologic disorders. Absence or blockage of the vas deferens, seminal vesicles, or ejaculatory ducts are classified as obstructive azoospermia.⁶

(Figure 1). However, this sequence is prone to alteration: although normally occurring in this order, it can miss a step, or even occur out of order. Most important, even though these events are intimately related, they can each occur independently, especially with pathologic conditions.

For example, men with generalized erectile dysfunction secondary to surgery often discover that continued manipulation of a flaccid or semi-erect penis can lead to orgasm, with or without expulsion of semen. It is therefore more useful to assess ejaculatory and orgasmic concerns

as entities separate from erection, but remain cognizant of any changes in sexual interest or erection that may further clarify the etiology of the ejaculatory/orgasmic concern.

Ejaculation

Ejaculation is the process of sperm transport from the epididymis to the urethral meatus, resulting in expulsion of semen (Figure 2). Ejaculation usually results in penile detumescence, followed by a refractory period, when arousal and/or orgasm are not possible again until a specific time has passed. Both time for detumescence and the refractory period are age- and medication-dependent.

Ejaculation occurs in two phases—seminal emission and propulsatile ejaculation: they are mediated by sympathetic and parasympathetic inputs, respectively. These phases are functionally susceptible to disease, trauma, or medications that interfere with specific neurologic functioning.

When sexual stimulation is extremely intense, autonomic impulses responsible for seminal emission exit the spinal cord at the T10-L2 level via the sympathetic chains, course into the pelvis as the hypogastric outflow, and on to the genital structures involved in ejaculation.⁷ Parasympathetic and somatic input from S2 to S4 then causes propulsatile ejaculation and antegrade ejaculation occurs (Figure 3).

Seminal emission. Seminal emission is the process of sperm transport from the epididymis to the prostatic urethra, followed by closure of the bladder neck. This component occurs at high arousal, and has an element of voluntary control. Emission begins with contraction of the vas deferens propelling sperm towards the prostate. Secretions emitted from contractions of both the seminal vesicles and prostate combine with the contents of the vas deferens as they come together in the prostatic urethra. These fluids combine to form semen.

With concomitant and intermittent closure of the external sphincter and entrapment of the seminal bolus in the prostatic urethra, increased intraprostatic pressure results. Men recognize this as a feeling of “no return,” or ejaculatory inevitability. At this point, triggering of the second phase of ejaculation occurs.

FIGURE 2 Physiology of ejaculation

Ejaculation involves sperm transport from the epididymis to the urethral meatus, resulting in expulsion of semen. The two phases of ejaculation are seminal emission and propulsatile ejaculation. Closure of the bladder neck at the time of ejaculation prevents semen from entering the bladder and may make urination difficult immediately following ejaculation. Ejaculation usually results in penile detumescence, which is followed by a refractory period.

Propulsatile ejaculation. The second phase of ejaculation consists of rhythmic contractions of the seminal vesicles, prostate, urethra, and pelvic-floor muscles that propel the seminal bolus through the urethra and out of the urethral meatus with considerable force. There is little voluntary control over this process. Closure of the bladder neck at the time of ejaculation prevents semen from entering the bladder in retrograde fashion.

Orgasm

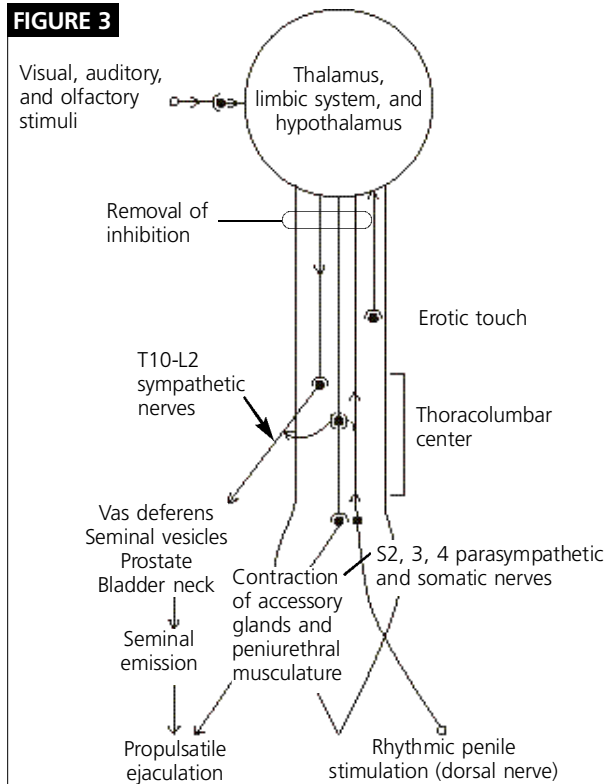
Orgasm is the brain's interpretation of ejaculatory events, even if ejaculation does not proceed in a normal fashion; men with penile amputation or loss of their prostate may still experience orgasm even if no erection or ejaculate is present. However, a small amount of testosterone appears to be crucial for attainment of orgasmic release. Orgasm is a cerebral event usually experienced at the time of ejaculation, but can occur in its absence.

Orgasm may vary in intensity and duration, depending on biopsychosocial factors, such as length of arousal, tone of the pelvic floor, duration from last orgasm, drug or alcohol use, and comfort and sexual security with partner. Orgasms are usually a combination of peripheral (genital or extra-genital) stimulation and mental "letting go." Whereas the neurologic tracts for orgasm resulting from peripheral genital stimulation are known, the cerebral and interpretative pathways for orgasm generated from central afferents are still not well understood.

Neurologically speaking, it is accepted that the pathways for ejaculation and orgasm are under a tonic inhibitory influence, and that the release of this inhibition is cerebrally mediated. Without this release, the normal orgasmic and ejaculatory reflexes cannot be expressed.

Orgasms can be initiated centrally without genital touch, as demonstrated by nocturnal emissions, or "wet dreams," and by involuntary emissions seen in neurologic disorders,⁸ but they are rare in men without genital sensation.

FIGURE 3



The ejaculation reflex

The ejaculatory reflex is under control of several divisions of the central nervous system (CNS), and must occur in a well-coordinated, organized fashion. Although both cerebral and rhythmic genital input are usually required to initiate the reflex, it may be solely initiated by CNS input, as seen in nocturnal emissions. Sensory information enters the sacral cord and proceeds up to the brain for processing, or reflexly to the thoracolumbar ejaculation center, where combined with descending cerebral input to be integrated in the cord, results in the ejaculation reflex.

Diagnostic categories

Clinically, it is easiest to think of ejaculatory problems in three broad categories following their order of prevalence; functional, neurogenic, and anatomic.

Functional. Functional ejaculatory problems present as sexual dissatisfactions, and include premature ejaculation, delayed ejaculation, and anhedonic ejaculation. Men who present with these conditions are usually neurologically intact. These ejaculatory problems are often the result of biopsychosocial factors, and treatment is dictated by the factors that are most prevalent in the assessment. For example, a young man with lifelong premature ejaculation is usually neurologically normal,

but his reflex is hypersensitive. Another man may find orgasm difficult to attain secondary to antidepressant therapy. An older man may not ejaculate during intercourse because of low androgens and/or inadequate genital stimulation; this is often experienced with age.

Neurogenic. Neurogenic disorders consist of either central or peripheral neurologic interruptions in the two-phase ejaculatory process of seminal emission and propulsive ejaculation. They are most often caused by trauma to the nerves (eg, spinal cord injury or abdominal/pelvic surgery) or by neurologic disease processes (eg, multiple sclerosis, diabetic neuropathy, and other peripheral neuropathies). These orgasmic and ejaculatory problems usually require additional medical intervention (ie, medications, mechanical stimulators, etc) and are usually referred to specialists.

Anatomic. Anatomic ejaculatory disorders may be congenital or acquired, usually interfering with ejaculation more than with orgasm, and most often present as infertility or ejaculatory pain. They are usually caused by infection, or by obstruction of the vas deferens, ejaculatory ducts, prostate, or urethra.

Formulating your approach to the patient

Ejaculation and orgasm are complicated sequential processes, so any neurologic or drug-induced interference can cause temporary or permanent problems. Your role as a primary care clinician is to identify if there is a reversible cause of ejaculation/orgasmic dysfunction, and to formulate a differential diagnosis that will dictate further investigations and/or referral. Many of the more common ejaculatory problems can be dealt with in the primary care setting with motivated physicians. However, because of the biopsychosocial complexity of these issues, you may de-

cide to refer the patient to a specialist for appropriate treatment, depending on your comfort level. However, even if you decide to refer the patient, it is necessary to do a thorough assessment as to the nature of the disorder.

In assessing ejaculatory/orgasmic concerns, the diagnosis can be made by looking at the individual central and peripheral components as well as the capacity for inhibitory release. This understanding is required to differentiate whether the concern is functional, neurologic, or anatomic, and to formulate an approach to diagnosis and a management strategy. For example, abnormalities of the central inhibitory release

mechanism are the key in diagnosis and treatment of functional difficulties (although the physiological component of functional problems must also be addressed).

For anatomic and neurologic orgasm/ejaculatory difficulties, treatment includes adapting or overcoming the additional organic pathology with various medical or operative options. While these latter cases are usually referred to specialists, the primary care physician is often left to deal with the psychological overlay caused by the dysfunction and/or its treatment.

A full sexual and medical history and a physical exam are necessary in the assessment of these concerns. ☞

Up to 40% of men in surveys report they experience ejaculatory problems, and premature ejaculation is the most prominent.

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