Delayed orgasm and anorgasmia

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Delayed orgasm/anorgasmia defined as the persistent or recurrent difficulty, delay in, or absence of attaining orgasm after sufficient sexual stimulation, which causes personal distress. Delayed orgasm and anorgasmia are associated with significant sexual dissatisfaction. A focused medical history can shed light on the potential etiologies, which include medications, penile sensation loss, endocrinopathies, penile hyperstimulation, and psychological etiologies. Unfortunately, there are no excellent pharmacotherapies for delayed orgasm/anorgasmia, and treatment revolves largely around addressing potential causative factors and psychotherapy. (Fertil Steril® 2015;104:1082–8. ©2015 by American Society for Reproductive Medicine.)

Key Words: Delayed orgasm, anorgasmia, delayed ejaculation, retarded orgasm, inhibited orgasm

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Delayed orgasm (DO) and anorgasmia (AO) have been described as one end of a spectrum of orgasm timing disorders with the other end being premature ejaculation (1). Delayed orgasm and anorgasmia are defined as the persistent or recurrent difficulty, delay in, or absence of attaining orgasm after sufficient sexual stimulation, which causes personal distress. Delayed orgasm has also been termed retarded orgasm, inhibited orgasm, retarded ejaculation, and/or inhibited ejaculation. We believe that DO is the correct term as some men fail to ejaculate for medical reasons but still experience orgasm (retroperitoneal surgery, radical prostatectomy). One of the major concerns with DO and in particular AO, young males or men with reproductive interest, is the failure to inseminate and therefore male factor infertility. Men with DO may develop anxiety and frustration, which may lead to other sexual problems such as erectile dysfunction and loss of sex drive. It is critically important to understand that orgasm is an entirely separate process from ejaculation, although they are designed to occur simultaneously.

In the clinical setting, most men with failure to ejaculate (retrograde ejaculation, failure of emission both addressed elsewhere in this issue) experience orgasm (although a man with failure to ejaculate for medical reasons may also have DO or AO). However, men with AO will not ejaculate.

DEFINITION

The best definition is probably that of the World Health Organization 2nd Consultation on Sexual Dysfunction that defines DO as the persistent or recurrent difficulty, delay in, or absence of attaining orgasm after sufficient sexual stimulation, which causes personal distress (2). The International Consultation on Sexual Medicine defines AO as the perceived absence of orgasm, independent of the presence of ejaculation. The Diagnostic and Statistical Manual of Mental Disorders, 5th edition, defines delayed orgasm as a marked delay in ejaculation or a marked infrequency or absence of ejaculation on almost all or all occasions (75%–100% of time) of partnered sexual activity without the individual desiring delay, persisting for at least 6 months and causing significant distress to the individual (3). The sexual dysfunction is not explained by another nonsexual disorder, medication, or significant relation/life distress/stressors.

Delayed orgasm is further classified as lifelong/acquired, generalized/situational, and mild/moderate/severe. An acquired dysfunction establishes that the patient previously had normal orgasm timing. Situational dysfunction implies the man has problems in a particular scenario or scenarios, yet functioning normally in others.

There is no set time threshold for what defines DO. Time threshold for distress is dependent on the partners involved. Some men will reach orgasm with one partner in 15 minutes and have no distress, but with another partner it may cause severe distress because the partner may complain of pain with prolonged intercourse. A population-based survey established that the

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median intravaginal ejaculatory latency time (IELT) was 5.4 minutes and 2 SD above was approximately 22 minutes (4–6). A provider with a patient complaining of IELT longer than 22 minutes will theoretically qualify him for the diagnosis of DO. One should differentiate between problems with of ejaculation and orgasm.

PHYSIOLOGY OF ORGASM

Orgasm is a complex neurobiological process that comes as a result of sexual activity (physical sensation) and/or arousal (cognitive awareness). The physiology of ejaculation is discussed elsewhere. When ejaculation occurs, the brain processes the sensation of the pressure buildup within the posterior urethra (bladder neck and external urinary sphincter are closed contemporaneously) leading up to seminal fluid emission and the contraction of the periurethral musculature. This processing leads to the triggering of an orgasm.

Advances in functional neuroimaging have been able to show the location of increased brain activity during orgasm (7). Positron emission tomography imaging has demonstrated that sexual stimulation leads to increased activity in the occipitotemporal, anterior cingulated, and insular cortices, as well as bilateral activation in the substantia nigra (8). During orgasm there is a decrease in regional cerebral blood flow across the prefrontal cortex (right medial orbitofrontal, left lateral orbitofrontal, left dorsolateral) and in the left temporal lobe (fusiform gyrus, superior temporal gyrus), as well as increased activation in the left dentate cerebellar nucleus, left lateral midbrain, and right pons (8, 9).

PREVALENCE

The Diagnostic and Statistical Manual of Mental Disorders, 5th edition, states that only 25% of men routinely achieve orgasm in all sexual encounters. According to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition, the prevalence remains constant until age 50 years and then the rate steadily increases with men in their 80s complaining twice as much as men less than age 59 years (3). The increase with age is likely multifactorial and may be related to a combination of changes in penile sensitivity, increased prevalence of T deficiency, increased use of offending medications, decreased exercise tolerance, and reduced partner tolerance for prolonged sexual intercourse. In one study, the prevalence of primary DO was found to be 1.5 in 1,000 and secondary DO in men less than age 65 years was 3%–4% (10, 11). Masters and Johnson only reported on 17 cases (12), Apfelbaum reported 34 cases (13), and Kaplan reported <50 cases (14). Because this is such an uncommon complaint, the true prevalence is probably underestimated. In a study by Carani et al. (15), they assessed 48 adult men, 14 with hypothyroidism and 34 with hyperthyroidism, and DO was identified in 64% of the hypothyroid patients and 3% of the hyperthyroid patients.

PATHOPHYSIOLOGY

By definition, primary AO begins from the male's first sexual experiences and lasts throughout his life. Whereas, secondary AO is preceded by a period of normal sexual experiences before the problem manifests. A Finnish population-based, twin study found that there was no evidence of a genetic influence on DO/AO, but there was a moderate familial effect with shared environments, which accounted for 24% of the variance (16). This study included 1,196 twins and their siblings using retrospective self-reported data. Table 1 provides a summary of the different possible causes for DO.

In a study by Teloken et al. (17), the investigators performed an analysis of data on 206 patients who presented with secondary DO/AO. The etiology for their condition was divided into selective serotonin reuptake inhibitor (SSRI) use (42%), low T (21%; mean total T 268 ± 111 ng/dL), abnormal penile sensation (7%), chronic/idiosyncratic penile (hyper)stimulation (2%), and psychogenic causes (28%). Age-related hormonal declines (lower T levels) and age-related loss of peripheral nerve conduction may account for the increased onset after age 50 years (3). It has also been suggested that hormonal aberrations, such as hypothyroidism and T deficiency, may play a role in DO (1).

Endocrinopathies

The role of PRL in men is not fully understood. However, it is well understood that PRL levels at more than normal, hyperprolactinemia, may result in an inhibitory effect on sexual desire (18–20). Mild forms of hyperprolactinemia (defined as >420 mU/L or 20 ng/mL) generally do not have an impact on sexual function; however, severe hyperprolactinemia (defined as >735 mU/L or 35 ng/mL) can have significant effects on sexual function, including erectile dysfunction and T production suppression (18, 19, 21, 22). Prolactin secretion is positively influenced by PRL-releasing factors (thyroid-releasing hormone, oxytocin, vasopressin, and vasoactive intestinal peptide) (23). Serotonin is implicated in the control of PRL secretion through serotonergic inputs from the dorsal raphe nucleus stimulating PRL-releasing factors in the paraventricular nucleus (24). The SSRIs are therefore capable of causing hyperprolactinemia and lead to DO/AO (25). Corona et al. (1) identified relationships between ejaculation and PRL, TSH, and T levels. Knowing that DO and premature ejaculation represent two ends of a linear spectrum, it has been shown that PRL and TSH levels progressively increased from patients with

TABLE 1

Causes of delayed orgasm and anorgasmia.

Endocrine
Testosterone deficiency
Hypothyroidism
Hyperprolactinemia
Medication
Antidepressants
Antipsychotics
Opiods
Psychosexual causes
Hyperstimulation
Penile sensation loss

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