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Review Article

Retrograde Ejaculation: A Comprehensive Review

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Abstract

Retrograde ejaculation (RE) is a condition where semen flows backward into the bladder instead of being expelled through the urethra, significantly impacting male fertility and sexual function. It arises from neurogenic, pharmacologic, and surgical factors, with common causes including spinal cord injuries, diabetes, medications like alpha-blockers, and prostate or bladder neck surgeries. The pathophysiology involves dysfunction in the coordinated contraction of the bladder neck, ejaculatory ducts, and pelvic floor muscles. Diagnosis relies on clinical history, physical examination, and post-orgasm urinalysis. Management strategies range from pharmacological interventions, such as sympathomimetic agents, to surgical options and assisted reproductive techniques for fertility preservation. This review provides an in-depth analysis of the causes, mechanisms, and treatment options for RE to improve patient outcomes.

Keywords: Retrograde ejaculation; Ejaculatory dysfunction; Bladder neck dysfunction

Abbreviations

RE: Retrograde Ejaculation; **AUA**: American Urological Association; **ASRM**: American Society for Reproductive Medicine; **SCI**: Spinal Cord Injury; **LUTS**: Lower Urinary Tract Symptoms; **BPH**: Benign Prostatic Hyperplasia; **TURP**: Transurethral Resection of the Prostate; **RR**: Relative Risk; **PAE**: Prostate Artery Embolization; **FSH**: Follicle-Stimulating Hormone; **HbA1c**: Hemoglobin A1c; **TSH**: Thyroid-Stimulating Hormone; **SSRIs**: Selective Serotonin Reuptake Inhibitors; **TCAs**: Tricyclic Antidepressants; **RPLND**: Retroperitoneal Lymph Node Dissection; **PVS**: Penile Vibratory Stimulation; **IVF**: *In Vitro* Fertilization; **ICSI**: Intracytoplasmic Sperm Injection; **IUI**: Intrauterine Insemination.

Introduction

Retrograde ejaculation (RE) is defined by the AUA and ASRM as a condition where semen flows backward into the bladder rather than exiting through the urethra during ejaculation, resulting in reduced fertility [1]. This disorder can stem from various pathological causes.

Neurogenic factors play a critical role in RE. Nerve injuries or damage, such as those occurring in spinal cord injuries (SCI), are significant contributors. Other neurological conditions like multiple sclerosis, myelodysplasia, and diabetes mellitus (specifically uncontrolled diabetes leading to neuropathy) are also common causes. In diabetic patients, RE is linked to reduced intraurethral pressure.

Medications frequently contribute to RE. Alpha-receptor antagonists, used for treating lower urinary tract symptoms (LUTS), are well-documented in inducing RE. Drugs prescribed for hypertension, depression, and psychosis have similar side effects, interfering with the proper function of the bladder neck and internal urethral sphincter.

Surgical procedures affecting the urethra or bladder neck can also lead to RE. The most common surgical causes are treatments for benign prostatic hyperplasia (BPH), such as transurethral resection of the prostate (TURP), with studies reporting a 62–75% incidence of RE following standard TURP techniques [2]. Prostatectomy and retroperitoneal lymph node dissections are other frequent causes.

RE impacts approximately 0.3% to 2% of male infertility cases [3]. The backflow of semen into the bladder severely impedes natural conception, often requiring assisted reproductive techniques. Beyond fertility, the condition disrupts men's sexual experiences, leading to heightened anxiety and depression. These emotional challenges arise from disturbances in sexual function, reduced self-esteem, and strained intimate relationships [4]. Masters and Johnson (1989) documented that an increased seminal volume during ejaculation is associated with a heightened subjective sensation of pleasure. This effect is attributed to the distension of the urethra caused by the larger volume of seminal fluid.[5]

There is considerable debate and exploration surrounding the prevention, causes, and treatment options for retrograde ejaculation.

This paper seeks to provide a comprehensive overview of the pathophysiology of retrograde ejaculation, examining the various factors that contribute to its development. It will also discuss current strategies for its management, from medical to surgical interventions, and evaluate their effectiveness in addressing this condition. By understanding these aspects, we aim to offer a clearer perspective on how retrograde ejaculation can be prevented, treated, and managed for improved patient outcomes.

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Pathophysiology

Ejaculation Physiology

Ejaculation is classically divided into two phases: emission and expulsion. It is under the control of both the parasympathetic and sympathetic nervous systems. The neural control of emission originates in the thoracolumbar spine (T10-L2) and coordinates the actions necessary for this phase. Sympathetic efferent fibers converge into the lumbar sympathetic trunk ganglia and then proceed posteriorly to the vena cava on the right and laterally to the aorta on the left. These fibers merge to form the superior hypogastric plexus anterior to the L5 vertebra and the sacrum. Postganglionic fibers then travel to their target organs, including the bladder neck, prostate, seminal vesicles, and vas deferens, to mediate sympathetic control during emission [2].

The parasympathetic nucleus in the sacral region (S2-S4 segments) innervates the prostate and seminal vesicles, located in lamina VI. Additionally, recent research has confirmed the presence of a spinal ejaculation generator in humans within the L3-L5 spinal segments). This generator integrates sensory and motor signals crucial for ejaculation coordination [6]. Lumbar spinothalamic cells play an essential role by transmitting sensory information about sexual stimuli to the thalamus. They also consolidate neural interconnections between somatic and autonomic centers within the spinal cord. These neural pathways are organized into reflex circuits responsible for the expulsion phase of ejaculation. This involves coordinated processes such as the contraction of the bulbospongiosus muscle, relaxation of the external sphincter, and synchronized contractions of the prostate and seminal vesicles [7].

Sensory input during ejaculation is mediated by the perineal branch of the pudendal nerve, which receives signals from two distinct sets of nerve fibers. One set runs along the dorsolateral aspect of the penis, innervating the shaft and glans, while the other runs along the ventrolateral portion, innervating the urethra. Sensory axons of the afferent perineal nerve synapse on the pudendal motor neurons in the nucleus of Onuf and spinal interneurons that communicate with the thoracolumbar spinal cord (T10-L2), triggering emission. The efferent signals exit the spinal cord via the perineal nerve and terminate at the bulbospongiosus muscle, enabling somatic reflexes [7].

Retrograde Ejaculation

Studies have shed significant light on the mechanisms underlying both anterograde and retrograde ejaculation. A landmark investigation by Gil-Vernet in 1997 utilized endorectal ultrasound to provide a detailed understanding of the ejaculation process. The research revealed that during ejaculation, the verumontanum moves slightly caudally, momentarily contacting the opposing urethral wall. This finding demonstrated that ejaculation relies not only on bladder neck closure but also on the coordinated actions of the verumontanum, prostate contractions, and ejaculatory duct activity [8].

In 2005, another ultrasound study investigating retrograde ejaculation in a man with a spinal injury revealed that the ejaculation mechanism closely mirrored that of healthy individuals, with prostate contractions and bladder neck flattening observed. However, during the expulsion phase, the inframontanal urethra dilated and filled with semen, identifying the primary issue as the failure of the external sphincter to relax, ultimately resulting in retrograde ejaculation [9].

Effective semen propulsion relies on the precise coordination of several physiological processes, including the synchronized relaxation of the external urinary sphincter, the simultaneous closure of the bladder neck, and the rhythmic contractions of the striated pelvic floor muscles—particularly the bulbospongiosus muscles—working in harmony to ensure proper semen expulsion [10]

To demonstrate that the bladder neck does not play a primary role in retrograde ejaculation, researchers conducted a study involving 89 patients who underwent prostate and bladder neck resection using a technique designed to preserve anterograde ejaculation. The procedure ensured that the paracollicular tissue and the tissue situated 1 cm proximal to the verumontanum were not resected. After five years of follow-up, only 9.2% of the patients exhibited retrograde ejaculation [11]. In another study, 16 patients with bladder outlet obstruction underwent bladder neck resection while preserving the final centimeter of the supramontanal urethra. Among these patients, no cases of retrograde ejaculation were observed [12].

This suggests that the primary factor in preventing retrograde ejaculation is not the closure mechanism of the bladder neck but rather the coordinated function of several structures. These include the verumontanum, the peristaltic contractions of the ejaculatory ducts, the external sphincter muscles, and the bulbospongiosus muscle, all working together to ensure the proper expulsion of semen.

Evaluation

Evaluating ejaculatory dysfunction necessitates a comprehensive approach that incorporates situational factors and a detailed patient history. Key aspects include inquiries about nocturnal emissions and the situational variability of symptoms. A thorough review of sexual, pharmacologic, medical, and surgical history provides critical insights, particularly for identifying underlying conditions such as hypogonadism (e.g., low energy and libido), erectile dysfunction, diabetes (e.g., polyuria), psychiatric illnesses (e.g., depression), or neurological disorders (e.g., sensory abnormalities, bowel, or bladder dysfunction).

A focused physical examination is equally important, emphasizing signs of hypogonadism (e.g., atrophic testes, underdeveloped phallus), thyroid or endocrine abnormalities (e.g., gynecomastia), penile irregularities, and diabetic neuropathy (e.g., impaired peripheral sensation, obesity). Laboratory evaluations, including FSH, testosterone, HbA1c, TSH, and prolactin levels, are essential for clarifying the underlying cause. Post-orgasm urinalysis can also help confirm retrograde ejaculation.[13].

Causes of Retrograde Ejaculation

Medications

Alpha-blockers are a class of therapeutic agents commonly used as first-line treatment for bothersome BPH. These medications work by blocking alpha-1 adrenergic receptors, which exist in three subtypes: alpha-1a/c, alpha-1b, and alpha-1d. Non-selective alpha-1 blockers, such as prazosin, doxazosin, and terazosin, block all three subtypes and are referred to as alpha-1 selective blockers [14]. More

recently developed medications, such as tamsulosin, alfuzosin, and silodosin, are selective for the alpha-1A receptors found primarily in the prostate. These agents have a lower affinity for receptors in blood vessels, allowing for a more targeted therapeutic effect [15].

Alpha-blocker blockade relaxes smooth muscle, which can reduce fluid emission and potentially lead to an incompletely closed bladder neck. A study comparing common alpha-blockers to assess their likelihood of causing retrograde ejaculation suggests that terazosin, doxazosin, and alfuzosin may be associated with lower rates of ejaculatory dysfunction compared to other alpha-blockers. This could be attributed to the more nonspecific action of these agents. In contrast, drugs with higher selectivity and affinity for alpha-1a receptors are more likely to cause ejaculatory dysfunction.[16]

A recent meta-analysis showed that, compared to other α-blockers, silodosin is associated with a notable number of adverse effects, followed by tamsulosin and doxazosin. The most common adverse effects included ejaculatory dysfunction, dizziness, and hypotension. In addition to supporting our findings, studies on newer drug treatments for LUTS also confirmed that silodosin has a higher adverse effect profile than other therapies, particularly exhibiting a higher rate of ejaculatory dysfunction. [17]

Other medications, such as antipsychotics (e.g., risperidone), can cause retrograde ejaculation in some patients [18]. Additionally, antidepressants, including Selective Serotonin Reuptake Inhibitors (SSRIs) like fluoxetine, sertraline, and paroxetine, as well as Tricyclic Antidepressants (TCAs) like imipramine, have also been associated with this condition [19].

Surgeries

Anterior lumbar surgery is identified as a major cause of retrograde ejaculation (RE), with a reported incidence of up to 9.8%, according to a systematic review. The pathophysiology of RE following anterior lumbar surgery is primarily due to damage to the hypogastric plexus, which traverses the prevertebral space near the L5/S1 interspace [20]. Another surgery associated with high rates of retrograde ejaculation is prostate resection for benign prostatic hyperplasia (BPH). This procedure is linked to elevated rates of retrograde ejaculation as a complication. A study showed that, compared to the traditional procedure, transurethral resection of the prostate (TURP), newer techniques result in lower rates of retrograde ejaculation. Endoscopic treatments such as ThuLEP, Greenlight laser, and prostate artery embolization (PAE) were found to have a lower—but not statistically significant—relative risk (RR) of retrograde ejaculation compared to TURP (RR: 0.90; P=0.35; RR: 0.71; P=0.1; RR: 0.73; P=0.11) [21]. Additionally, a systematic review found that procedures like Rezum, UroLift, and PAE had minimal impact on patients' sexual function, particularly in preserving ejaculation, making them the best options for treating BPH while maintaining anterograde ejaculation [22].

Neurological Disease

Sexual dysfunction is a common complication of neurogenic diseases such as spina bifida, spinal cord injury, and multiple sclerosis, among others. Erection tends to be less affected than ejaculation. Higher spinal lesions typically preserve penile responses to reflexogenic stimulation, while lower lesions maintain psychogenic-mediated reflex erections. Emission is preserved in most patients, even those with lower or peripheral lesions. However, ejaculation is more commonly impaired, with preservation occurring in 4% of patients with higher lesions and 18% with lower lesions. Despite this, ejaculation can still be triggered in approximately 80% of patients using penile vibration and in 90% with electroejaculation. Orgasm is preserved in 40–50% of patients [2].

Diabetes

Ejaculatory dysfunction in diabetic patients can progress gradually, ranging from reduced ejaculate volume to retrograde ejaculation or anejaculation, depending on the severity of sympathetic autonomic neuropathy. This condition, like other diabetes-related complications, is closely linked to glycemic control. Studies have consistently shown that semen volume is significantly reduced in men with diabetes (both type 1 and type 2) compared to non-diabetic controls [23]. However, findings on sperm morphology and motility are inconsistent, with most studies indicating impairment in one or both parameters. Additionally, increased sperm DNA damage has been observed in diabetic patients [24].

The underlying mechanisms for altered semen parameters include endocrine disruption, such as reduced testosterone levels, and oxidative stress. Reduced semen volume may be attributed to neurological damage, as seminal vesicle atony has been documented in infertile diabetic men with neuropathy. A recent retrospective analysis highlighted a high prevalence of subfertility (51%) among men with diabetes, underscoring the significant impact of the disease on male reproductive health [25].

Management of Retrograde Ejaculation

Effective management of retrograde ejaculation RE requires a comprehensive approach that considers the patient's medical history, including coexisting conditions, medications, and previous surgeries. Risk factors for RE include diabetes mellitus, spinal cord injury (SCI), retroperitoneal lymph node dissection (RPLND), surgeries involving the prostate or bladder neck, and the use of alpha-blockers or psychotropic medications, all of which should be carefully assessed. [26].

The mechanism of sympathomimetic agents is to improve bladder neck contraction during the expulsive phase of ejaculation. Drugs such as ephedrine sulfate, imipramine hydrochloride, midodrine hydrochloride, brompheniramine maleate, and pseudoephedrine hydrochloride can support this process. Among these, imipramine is the preferred agent, particularly in patients who have undergone retroperitoneal sympathetic denervation, demonstrating high success rates [13].

A recent review of the literature revealed that treatment with sympathomimetics restored antegrade ejaculation in 28% of patients [27]. Additionally, a clinical study involving 12 patients with complete retrograde ejaculation, treated with pseudoephedrine prior to semen analysis, found that seven (58.3%) of these patients had spermatozoa recovered in the antegrade ejaculate. In the group of eight patients with partial retrograde ejaculation, seven (87.5%) treated with pseudoephedrine showed improvement in semen parameters during post-treatment analysis [28]. A recent single-arm Phase II trial with

22 evaluable patients, all ≥6 months after RPLND, demonstrated that four (18%) achieved a sperm count greater than 39 million per ejaculate (P = 0.20), and four (18%) had a semen volume exceeding 1.5 mL (P = 0.20) [29]. In a separate study comparing amoxapine to vitamin B12, the success rate was 88% (22/25) overall. Notably, the success rate was higher for amoxapine than for vitamin B12 (80%, 20/25 vs 16%, 4/25; P < 0.0001) [30].

If medical management fails, surgical options such as the Young-Dees type of bladder neck reconstruction have been shown to be successful in 4 out of 5 patients [31]. In another study, two patients with retrograde ejaculation (RE) following Y-V plasty of the bladder neck underwent a surgical procedure to reconstruct the internal vesical sphincter. After excising scar tissue and approximating the normal bladder neck muscle, both patients restored normal ejaculation, with one even successfully fathering a child [32].

Bladder neck collagen injections have also been tested as a potential method to achieve antegrade ejaculation [33]. Pharmacologically induced retrograde ejaculation (RE) can be reversed through the intermittent use of a medication that relaxes the bladder neck or by reducing the medication dose. Additionally, ejaculation with a full bladder, which promotes bladder neck closure, is a simple and effective approach to facilitating the restoration of normal ejaculation [34].

For men concerned about fertility, several sperm retrieval techniques have been explored in the literature. These methods include urinal sperm retrieval, prostatic massage, penile vibratory stimulation (PVS), electroejaculation, and vasal aspiration.

Urinary sperm retrieval, which involves postcoital voiding insemination after urinary alkalinization with an oral agent, has demonstrated promising outcomes in assisted reproductive techniques such as intrauterine insemination (IUI), *in vitro* fertilization (IVF), and intracytoplasmic sperm injection (ICSI), with per-cycle pregnancy rates ranging from 20% to 50% [35]. Similarly, prostatic massage has been effective in retrieving sperm for ICSI, and a case series found no significant difference in fertilization or pregnancy rates when comparing sperm retrieved via prostatic massage to that obtained through postejaculatory urinalysis [36]

Penile vibratory stimulation (PVS) has been effectively used in patients with various causes of anejaculation, including psychogenic anejaculation and spinal cord injury. Success rates for PVS range from 65% to 83% in larger studies, making it a promising option for inducing ejaculation in these patients [37]. In the same vein, electroejaculation, another method for overcoming anejaculation, has shown high success rates, ranging from 80% to 97%, in both psychogenic and non-psychogenic cases. The pregnancy rates following electroejaculation are also notable *in vitro* fertilization (IVF) or intracytoplasmic sperm injection (ICSI) showing pregnancy rates ranging from 6.3% to 83.3% [34]. Additionally, vasal aspiration using percutaneous, no-scalpel, or microsurgical techniques for sperm retrieval has demonstrated per-couple pregnancy rates of 60% to 71% when combined with IVF/ICSI [38].

Conclusion

Retrograde ejaculation (RE) is a complex condition shaped by neurological, surgical, and pharmacological factors, with significant implications for male fertility and quality of life. Its effective management requires identifying underlying causes, utilizing targeted pharmacological therapies, and considering surgical or assisted reproductive techniques when appropriate.

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