Ejaculatory disorders and sexual function

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INTRODUCTION

Recent advances in the knowledge and treatment of erectile dysfunction have increased patient and practitioner awareness of other sexual problems, particularly ejaculatory disorders (EjD). Fertility is a major concern in the younger man, but EjD can cause considerable distress to men of all ages about their sexual function. In a recent survey of 12 815 men aged 50–80 years, 46% had an ejaculatory disturbance and 59% were 'highly bothered' by it, particularly if they also had LUTS [1]. Often sexual function in this age group is overlooked as a management factor. The most common disorder is premature ejaculation (PE), with prevalence rates of 30–40% [2,3], although in the UK, retarded ejaculation and orgasmic disorders are also a concern in 2.5% of patients with a sexual problem and attending their GP [4]. Delayed ejaculation (DE) affects ≈4% of sexually active men [5]. Retrograde ejaculation (RE) occurs in ≈75% of men after TURP and to a lesser extent after bladder neck incision, because the bladder neck fails to close [6]. This review outlines the mechanisms, causes and treatment of EjD from the perspective of male sexual dysfunction. It is timely to review ejaculatory disorders from this perspective, as the review by Hendry focused primarily on fertility issues [7]. For this review we used PubMed searches of English language papers, emphasizing the past 8 years of published work. Keywords included 'ejaculatory disorders, ejaculation, premature ejaculation, delayed ejaculation, anejaculation, retrograde ejaculation, painful ejaculation, anorgasmia'.

MECHANISM OF EJACULATION

Ejaculation comprises two phases: sympathetically mediated emission of seminal fluid into the posterior urethra, followed by somatically mediated true ejaculation with expulsion of the ejaculate. The reflex is initiated by stimulation of Krause-Finger corpuscles in the mucosa of the glans penis. These discharge as soon as a certain level of excitation is achieved. The sensory information is then transmitted via the dorsal nerve of the penis (S4) to the lumbosacral spinal cord, and is joined by sympathetic afferents from the hypogastric plexus (Fig. 1). Travelling up the spinal cord (and joined by visual, auditory and olfactory cerebral afferents) the impulses are integrated into the complex pattern of copulatory behaviour by forebrain structures that include the medial preoptic area and the paraventricular nucleus of the hypothalamus. The understanding of the neurotransmission in the hypothalamus has now advanced the medical treatment of ejaculatory disorders. Based on animal studies, dopamine has been shown to stimulate ejaculation [8], whereas serotonin (5-hydroxytryptamine, HT) inhibits it [9]. Furthermore, 16 5-HT receptor subtypes have been identified, with a balance between the facilitatory 5-HT1A and inhibitory 5-HT2C receptors being necessary [10,11]. Nitric oxide also mediates the inhibitory neurotransmission responsible for seminal emission [12].

The efferent limb of the reflex, responsible for emission, consists of sympathetic efferent fibres (T10–L2) that traverse over the pelvic brim via the hypogastric nerves to the pelvic plexus. From here, the fibres pass to, and cause, sequential contraction of the epididymis, vas deferens, seminal vesicle and prostate, with closure of the bladder neck. True ejaculation is then initiated somatically from the sacral spinal cord (S2–S4) via the pudendal nerve, causing rhythmic contractions of the bulbospongiosus and bulbocavernosus muscles, which force the ejaculate through the distal urethra.

CLASSIFICATION AND AETIOLOGY

There are several ways in which the ejaculatory process can become dysfunctional, leading to a partial or complete loss of ejaculation. These may be of either psychogenic or organic origin, and several drugs are also implicated (Table 1).

Classification of ejaculatory disorders

- Early/rapid/PE; inability to control ejaculation for a 'sufficient' length of time before penetration.
- Inhibited/delayed/retarded ejaculation; abnormal stimulation of the erect penis is necessary to achieve orgasm with ejaculation.
- Anejaculation; complete absence of antegrade or retrograde ejaculation.
- Anorgasmia (primary or secondary); the inability to reach orgasm.
- Aspermia; absent genital tract contraction.
- RE; total absence of antegrade ejaculation because semen passes backward through bladder neck into bladder.
- Haemospermia.
- Low volume ejaculate.
- Painful ejaculation.
- Lack of pleasure (anhedonia).

PE

Excessive stimulation of the ejaculatory centre in the hypothalamus is usually of cortical origin, caused by over-excitement or anxiety. The recommended definition of PE is an ejaculation that occurs within 1 min after vaginal penetration or experiencing ejaculation that occurs too early for partner satisfaction in at least half of attempts at intercourse [13,14]. The question of whether or not the cause of PE is organic, or is a result of normal sexual function associated with abnormal expectations or particular early experiences, has been raised many times. Until recently, PE was treated mostly by behavioural techniques (the 'squeeze technique' and the 'stop-start method') following the work of Masters and Johnson [15] and Semans [16], based on the notion that PE was an acquired disorder caused by initial hurried intercourse and a lack of awareness of pre-ejaculatory sensations. Hypersensitivity of the penile skin [17] and anxiety have been suggested as causal factors in PE and several other organic causes, including seminal plasma magnesium levels [18], hyperthyroxinaemia [19] and

prostatitis [20], have been associated with the condition. Strassberg *et al.* [21] proposed a psychophysiological model of PE, in which the role of anxiety is seen as variable, interacting with the somatic vulnerability of the individual to determine orgasmic latency. More recently, Waldinger [22] postulated that lifelong PE is not an acquired disorder but part of a normal biological variability of the intravaginal ejaculation latency time in men. PE is now considered to be primarily a neurobiological phenomenon that may lead to psychological distress [11]. These two elements inform treatment approaches (Table 2).

The pharmacological treatment of PE consists of 5-HT_{2C} receptor stimulation and/or 5-HT_{1A} inhibition. Treatment with serotonergic antidepressants has been shown to be very effective. Paroxetine has been shown to cause the greatest ejaculation delay [23]. Good results have been reported with SS-cream used a few hours before intercourse [24]. A case series examined by Wise and Watson [25] suggested that a novel device for treating PE based on the penile hypersensitive hypothesis (a desensitizing band) had a substantial effect on the latency period. The ring is designed to be used daily for no longer than 30 min per day for up to 6 weeks, and is not to be used for penetrative sex.

Four subtypes of biogenic and four types of psychogenic PE have been described [26]. Specific treatment is suggested for these subtypes, which can be diagnosed using an assessment exercise [26].

Effective psychosexual treatment strategies, e.g. relaxation, entrancement arousal, pubococyygeal muscle training, cognitive and behavioural pacing strategies and couple work are recommended [26]. The aim is to regain ejaculatory control and increase intimacy, pleasure and satisfaction using a combination of psychosexual skills and, when necessary, pharmacological treatments.

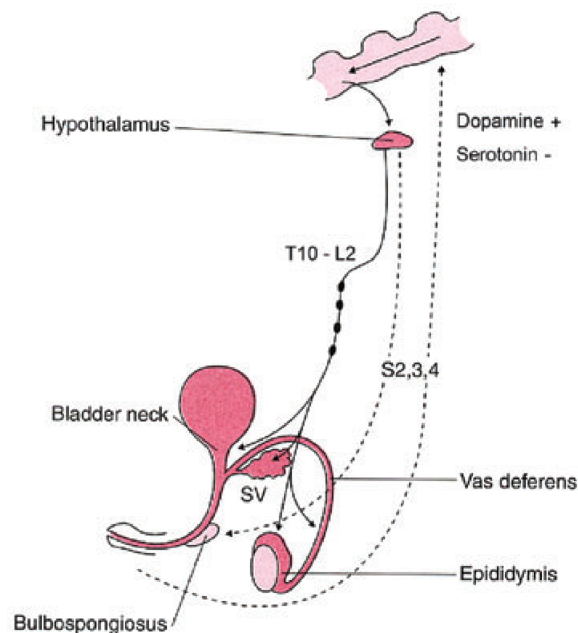

FIG. 1.
The mechanism of ejaculation.

TABLE 1 Drugs causing ejaculatory problems

Drug	Side-effect
Carbamazepine	Failure and loss of orgasm
Modifying 5HT agents (e.g. antidepressants) clomipramine	inhibit ejaculation
Anti-adrenergic agents & ganglion blockers (e.g. methyl dopa, guanethidine, monoamine oxidase inhibitors)	inhibit ejaculation
Some α-blockers (e.g. tamsulosin)	retrograde/anejaculation
SSSRIs (e.g. paroxetine, clomipramine, sertraline, fluoxetine)	inhibit ejaculation
Antipsychotics (e.g. thioridazine, clozapine, risperidone)	inhibit/retrograde
Antiandrogens (cyproterone acetate, flutamide, goserelin)	low volume ejaculate
Thiazide diuretics	retrograde ejaculation
Phenothiazines	retrograde ejaculation
Withdrawal off trifluoperazine, opiates (morphine) and ephedrine	rapid ejaculation

SSRI, selective serotonin reuptake inhibitor.

DE

An inhibition of the ejaculatory reflex results in DE, possibly with reduced or absent orgasm. Psychological causes may result from strict religious backgrounds, lack of attraction to a female partner and psychologically traumatic events. Inhibited arousal may be fairly common among men with DE with no probable somatic cause [27], suggesting that several specific relationship factors may provide strategies for enhancing arousal in these men.

The goal of behavioural therapy is to treat nonadaptive conditioning to increase the latency of ejaculation. Treatment may suggest decreasing the frequency of ejaculation outside of sexual encounters with a partner, and involves discussion of anxieties that may affect performance. Psychological work may explore themes such as inability to detach from the mother, so making commitment to another woman difficult, fear of hurting wife, punitive and controlling action over his partner and fear of pregnancy.

Additional therapy should instruct the man to spend some time focusing on the pleasurable sensations associated with caressing the penis and frenulum, alongside nongoal-orientated fantasy. Both vigorous stimulation using oils and high-speed water from a shower head can enhance arousal. The partner can become involved with super-stimulation using techniques such as hand-held vibrators, the bridging manoeuvre and partner stimulation of the perineal region or, if acceptable, digital prostatic stimulation. In

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TABLE 2 Treatments for PE

Drug	Dose	Time	Side-effects
Sertraline	50–100 mg	2–4 h before	Excessive delay
Clomipramine	25 mg	4–6 h before	Light headedness, dry mouth, nausea, dizziness, drowsiness & sleepiness, vision problems
Paroxetine	10–20 mg	Daily	Yawning (EjD rare), perspiration, fatigue, nausea, dry mouth, constipation or loose stools
Fluoxetine	10–20 mg	Every 2–3 days	nausea, diarrhoea, anxiety
Sildenafil	25–100 mg	60 min before	gastric upset, nausea, headache
Lignocaine	7.7 mg two sprays or 5% topical	10 min before	over-numbness in man and partner
Other options			
SS cream	topical	3 h before	
'Prolong' rings	rub frenulum with ring	5 min each day for 6 weeks	local skin irritation and soreness
'squeeze technique'			
'stop-start'			
double ejaculation			

TABLE 3 Treatments for DE

Drug	Dose	Time	Side-effects
Ephedrine	15–60 mg	60 min before	hypervigilance, anxiety
Pseudoephedrine	60–120 mg	120–150 min before	hypervigilance, anxiety
Desipramine	25–50 mg	30 min before	dry mouth
Yohimbine	20–45 mg	60 min before with no food	anxiety, palpitations, nausea
Cyproheptadine	4–12 mg	90 min before, (if SSRI-induced)	sedation, impaired concentration
Other options			
super stimulation/bridging technique	woman underneath holding base of penis and testicles		
vibrator to ventral surface of the penis (or prostate)	100 Hz	5–10 min before	

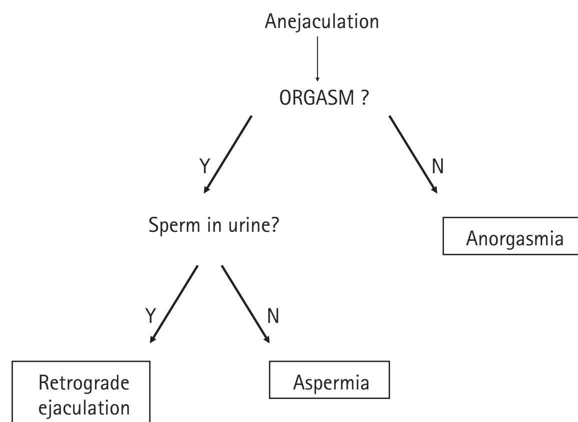

FIG. 2. Conditions presenting with anejaculation.

any sensate focus work, the man may have to be allowed to ejaculate alone, initially, with his partner watching from a distance and, eventually, intravaginally. Alcohol should be avoided in advance of sexual play (Table 3).

ANEJACULATION

Failure to ejaculate is often the presenting symptom, although the underlying diagnosis can usually be elicited from a detailed history and with simple investigations (Fig. 2).

ANORGASMIA

Anorgasmia may be divided into three categories: primary complete (has never had a normal orgasm), primary incomplete (lifelong undue delay in reaching a climax during sexual activity) or secondary (men who had normal orgasms before but then develop a failure to achieve it) [28]. Primary anorgasmia, particularly if nocturnal emissions occur, is usually caused by psychological factors, often attributed to an over-strict upbringing, be it social, cultural or religious, with repression of the normal sexual response to stimulation. Secondary anorgasmia may have an organic basis, particularly if neurological factors [29], e.g. multiple sclerosis or spinal injury, are

present. In some instances, it is the result of medication, e.g. paroxetine [30]. Anorgasmia is usually treated by using a vibrator, with or without drugs [31]. Some clinicians have experience using yohimbine, an α2-adrenergic receptor-blocking drug, to treat anorgasmia. At doses of 20–45 mg taken an hour before sexual activity, 66% (19/29) of patients became orgasmic and were able to ejaculate either during masturbation or sexual intercourse [32]. Other drugs helpful in serotonin reuptake inhibitor-induced anorgasmia are amantadine, bupropion, buspirone and cyproheptadine [33].

ASPERMIA

Aspermia is diagnosed in orgasmic patients who have no sperm in their urine sample afterward. In most cases it results from several organic causes related to damage to the sympathetic nerve supply. This is particularly relevant in patients having retroperitoneal lymph node dissections for germ cell tumours, aorto-iliac surgery and rectal excision, where the hypogastric nerves can be damaged as they traverse the pelvic brim. Lumbar sympathectomy and radical pelvic surgery are also common iatrogenic causes. Patients with autonomic neuropathy, usually from diabetes and other neurological conditions, e.g. multiple sclerosis and spinal injury, may also present because they have neurogenic loss of contraction of the epididymis, vas deferens, seminal vesicles and prostate. Treatment is indicated only to restore fertility via use of a vibrator, electro-ejaculator or conventional sperm retrieval techniques.

RE

Patients are orgasmic with anejaculation, as there is failure of the bladder neck to close during ejaculation, resulting in RE. RE can be congenital, e.g. exstrophy, imperforate anus or, more commonly, acquired after TURP and bladder neck incision in 80% and 35% of men, respectively [34]. Reports suggest that laser prostatectomy may result in less RE (one of 16 potent men after contact laser prostatectomy vs 13 of 16 after TURP) [35]. Other common causes include diabetic autonomic neuropathy, damage to the sympathetic nerves during pelvic and anterior spinal surgery [36], presenting with an isolated incompetent bladder neck, and various drugs.

TABLE 4 Treatments for RE

Drug	Dose	Time	Side-effects
Ephedrine	15–30 mg	60 min before	hypervigilance, anxiety
Pseudoephedrine	60–120 mg	120–150 min before	hypervigilance, anxiety
Imipramine	25–75 mg	three times a day	dry mouth, constipation

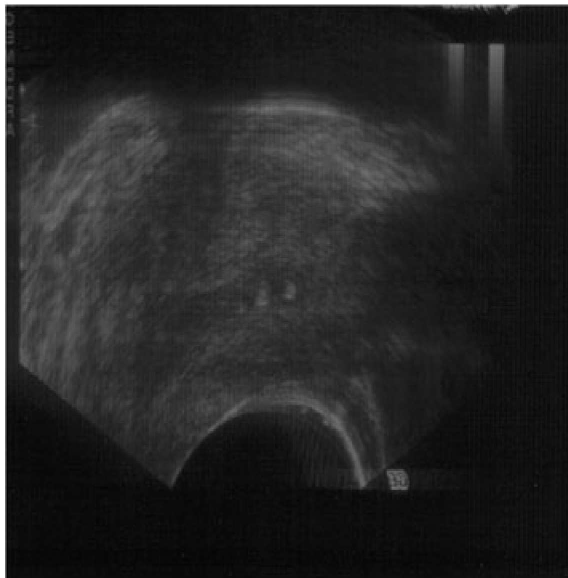

FIG. 3. TRUS findings of ejaculatory duct stones.

EjD in association with the treatment of BPH is common, at up to 30% with tamsulosin [37] (although not documented as RE or anejaculation) but less so with other uroselective α-blockers (some with no noticeable adverse ejaculatory events [38]).

The diagnosis of RE is confirmed by the presence of spermatozoa and fructose in a urine sample after orgasm, and supplemented by TRUS confirmation of an open bladder neck at rest.

Medical treatment is aimed, primarily, at stimulating the α-receptors of the bladder neck in the hope that it will contract and close. Adrenergic drugs or tricyclic antidepressants with noradrenaline re-uptake blocking action are the current drugs of choice (Table 4).

Climaxing with a full bladder is not beneficial, and the use of bladder neck bulking agents such as Macroplastique and PTFE has been only marginally successful in patients with no bladder neck scarring from previous surgery.

PAINFUL EJACULATION

This is an uncommon problem that may have psychological or organic causes, e.g. acute or chronic prostatitis. In an Internet survey of 163 men with a history of prostatitis, 69% reported pain before or after ejaculation [39]. Conventional treatment of the prostatitis, particularly if acute, is indicated, but relapses are common. Some causes of ejaculatory duct obstruction, inflammation or stones that cause pain and or haemospermia can be managed by endoscopic resection (Fig. 3).

LOW VOLUME

This is an unusual complaint and is mainly assessed during the management of infertility. A volume of <1 mL is significant for investigation. As seminal fluid formation is androgen-dependent, a deficiency resulting from hypogonadal states, or antiandrogen drugs, may present with low volume or, occasionally, absent ejaculate. Urethral strictures, ejaculatory duct obstruction, congenital anomalies of the seminal vesicles

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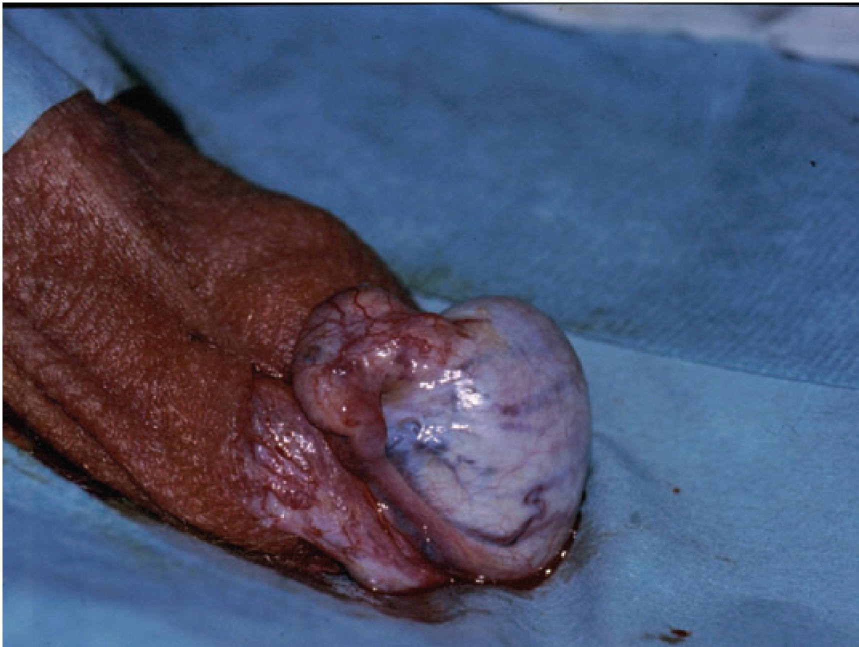
FIG. 4. Absent vas deferens associated with seminal vesicle anomalies and low ejaculate volume.



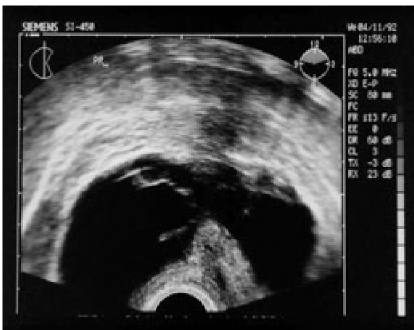


FIG. 5. TRUS of mega seminal vesicles.

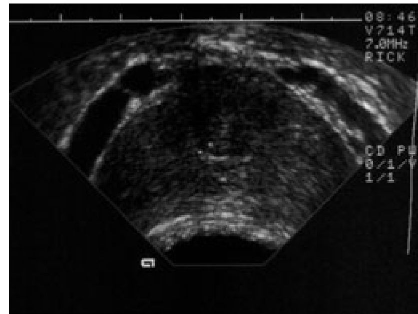


FIG. 6. TRUS showing ejaculatory duct obstruction.

(e.g. absent vas deferens) or partial neurological lesions from diabetes or surgery can also present in this way (Fig. 4).

HAEMOSPERMIA

Patients are often concerned when blood is noticed in their semen, but it is usually benign. In the younger patient (<40 years), an infective cause of the urogenital tract is likely and all that is necessary is a urine culture and a screen for sexually transmitted disease, and treated as appropriate [40]. In the older man, or if the haemospermia is persistent and with no haematuria, investigations should include TRUS to exclude cysts, calculi, dilatations, polyps of the ejaculatory duct and seminal vesicles (Fig. 5 and Fig. 6), and prostatic carcinoma. The incidence of prostatic carcinoma detected with this presentation is 1–3% [40–42].

CONCLUSION

Understanding of the ejaculatory process is greatly advanced, with both psychogenic and organic origins now being explored. Urological problems remain a frequent cause of men consulting their GP, but the direct effect that conditions like BPH and prostatitis have on sexual function is often overlooked. The additional effect that EjD have on psychological well-being is difficult to quantify. Existing tools do not specifically measure this effect but the recent development of the Male Sexual Health Questionnaire, including detailed information on male ejaculatory function, should help further our understanding. Careful evaluation of EjD is recommended to establish the cause. Both pharmacological and non-pharmacological interventions are successful in treating EjD, and allow for management in the community. However, it is important to use a dual-management strategy covering pharmacological and other approaches.

CONFLICT OF INTEREST

None declared.

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Abbreviations: **EjD**, ejaculatory disorders; **PE, DE, RE,** premature, delayed, retrograde ejaculation; **HT**, hydroxytryptamine.

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