The mechanisms of human ejaculation – a critical analysis

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ABSTRACT The current mechanism for human ejaculation is a two-part model consisting of a postulated “pressure chamber” created in the prostatic urethra by initial closure of both distal and proximal sphincters with secretions loaded in by adrenergic-mediated smooth muscle contractions of the vas deferens and capsules of the internal genital organs. The pressure build up is claimed then to “trigger” the intermittent relaxation/contraction of the distal sphincter and activate the contractions of the striated pelvic musculature, especially that of the bulbocavernosus, which forcefully expels the semen along the urethra by powerful, rhythmic spurts. A number of difficulties with this model are examined critically against experimental findings which indicate that the so-called pressure chamber is unlikely to be the valid trigger for ejaculation. The most recent finding of a specialised group of lumbar sacral neurons in the spinal cord of rat that function as the spinal ejaculation generator is more likely to be the “trigger” for ejaculation but confirmation that these cells also exist in the human cord is waiting.

KEYWORDS: ejaculation, striated pelvic muscles, bulbocavernosus, prostate, seminal vesicles, vas deferens, penile glans, brain imaging, orgasm, endorectal ultrasound, spinal cord generator, urethra

Introduction

Of the four “e’s” of male sexual function viz excitation, erection, emission and ejaculation, the mechanism that has been studied the least is the last – ejaculation. Erection, being such an important aspect of male ego and sexuality, has been the most examined mechanism both experimentally and clinically. The reward for this has been extraordinarily successful, an oral pharmacologic treatment of astonishing effectiveness in three-quarters of those with erectile dysfunction. Ejaculation, however, has been until quite recently the Cinderella of male mechanisms with a very limited catalogue of experimental studies. Indeed, since Regnier De Graaf suggested in 1668 that the contraction of the striated bulbocavernosus muscle (bcm) at the base of the penis would compress the urethra and expel its contents there have
been few electromyographic studies on these muscles in humans (see Gerstenberg et al., 1990 for early references; Shafik, 1998; Shafik & El-Sibai, 2000).

Why has human ejaculation been so poorly investigated? Firstly, it is difficult to study – it doesn’t last very long – the muscular movements are rapid and are over in a few seconds for most men. Second, a large proportion of its activity is hidden well inside the abdomen and pelvis thus any imaging without X-ray cinematography posed a real problem until the advent of modern techniques of soft tissue imaging by magnetic resonance imaging (MRI) and by endorectal ultrasound. Brain imaging, able to record the changes in regional cerebral blood flow (rCBF) with functional MRI (fMRI) or positron emission transmission (PET) has allowed the black box of the brain to be opened up for study (see brief review by Levin, 2004 for references).

What has made ejaculation a target for study is the current focus on one of its disorders – that of premature, early or rapid ejaculation. This is claimed to be the most common male sexual disorder with prevalences reported in the USA of from as little of 5% to greater than 30% (Montague et al., 2004). The great range is partly due to the fact that more than one definition exists for the condition as a universal one has yet to be established. Many authors have proposed definitions starting with the now superseded Masters & Johnson’s (1970) “inability to delay ejaculation long enough for the woman to achieve orgasm fifty per cent of the time” to the American Psychiatric Association’s DSM-IV-R “the persistent or recurrent ejaculation with minimal stimulation before, on, or shortly after penetration and before the person wishes it. . . . The disturbance causing marked distress or interpersonal difficulty” and the World Health Organisation’s ICD-10 . . . . “There is an inability to delay ejaculation sufficiently to enjoy lovemaking”. These are only brief excerpts from much fuller definitions. The short ejaculatory latency, the inability to control the ejaculation and concern or distress about the situation are three fundamental criteria of the condition. Some think that fast ejaculation is a legacy of primitive man (Hong, 1984) as it allowed insemination of a greater number of females by the individual thus enhancing the chances of his genetic material being widely dispersed and it also reduced the time of his vulnerability to putative enemies. If, however, fast ejaculation was important in evolutionary terms slow ejaculators would have been replaced by the surviving fast ejaculators.

The current model of the human ejaculatory process

Remarkably a detailed, non-disputed physio-anatomical description of the mechanism of human ejaculation has still to be produced. The current model in the literature focuses on the posterior urethra and has contentious issues.

Although it is argued that emission initiates the ejaculation process, ejaculation proper starts with the end of the movement of the collected genital secretions into the prostatic urethra viz the end of the emission phase which is under the control of the hypogastric nerve (sympathetic spinal cord reflex, T10 – L2). This phase is mediated by activated adrenergic contractions of the smooth muscle in the capsules of the testes, seminal vesicles and prostate together with the contractions of the various duct smooth muscles (epididymis, vas deferens). The seminal vesicles contract by
Peristalsis (Mitsuya et al., 1960) and it is usually assumed that the seminal ducts do also but according to Brindley (1985) such contractile waves have never been observed in the vasa deferentia, rather the whole vas contracts from the cauda epididymis to the ejaculatory duct of the prostate.

Marberger (1974) was perhaps the first to describe speculatively the trigger for ejaculation as being initiated by the semen entering the posterior urethra accompanied by a closure of the neck of the bladder creating a closed so-called “pressure chamber”, a transformation controlled by the sympathetic (hypogastric nerve) and parasympathetic (pudendal nerve). This seminal entry together with the prostate’s own added secretion created distension of the “pressure chamber” and activated a series of rhythmic reflex contractions of the pelvic striated muscles. The proximal or internal prostatic sphincter contracts to prevent back-flux of semen into the bladder (retrograde ejaculation) but the distal or external sphincter relaxes, the semen is moved into the urethra (antegrade ejaculation). Pleasure is felt on this downloading. The pelvic striated muscles under the control of the pudendal nerve (parasympathetic spinal cord reflex, S2–S4), especially the bcm, then contract rhythmically and involuntarily (Shafik, 1995, 1998) which forcefully spruts the semen through the penile urethra to the outside world. Pulsating ecstatic pleasure occurs with each contraction, voluntary contractions of the bcm do not create this pleasure. The exact site of the generation of the ejaculatory pleasure is still a mystery.

According to the account of Newman et al. (1982) both sphincters of the prostate (distal and proximal) are contracted in the emission phase and the secretions pour into the posterior urethra converting it into the pressure chamber. The intermittent opening and closure of the distal sphincter (called the membranous sphincter in these early studies) aids in the propulsion of the semen through the bulbous urethra to the pendulous urethra (Kollberg et al., 1962); its importance is shown if it becomes paralysed or is incapacitated by experimental anaesthesia (Shafik, 1998), a dripping seminal emission but no ejaculation then occurs.

The feeling that men have of an impending ejaculation when fully sexually aroused is called “ejaculatory inevitability” and as the sensation increases less and less voluntary control over it occurs until a point is reached when it cannot be halted. Newman et al. (1982) stated that the feeling was due to the distension of the posterior urethra an attributed mechanism still accepted in the most recent account of ejaculation (McMahon et al., 2004).

In the model the smooth muscles perform the “loading” of semen into the duct system while the striated muscles and sphincters create the forceful pumped ejection. No study has ever correlated the contractions of the smooth musculature of the genital tract with the striated muscle contractions. However, it has been shown that the first striated muscle contraction (bcm) does not always eject semen presumably because the smooth muscle has not yet fully primed the system. The ejection of the main bulk of the semen is usually completed by 6–10 bcm contractions but in many men there are still extended further contractions presumably acting as a safety feature to ensure complete seminal ejection (Gerstenberg et al., 1990). The last fraction of the ejaculate expelled is from the seminal vesicles, interestingly sperm do not survive for long in this fraction. It has been suggested that this is a chemical defence by the
male to deactivate the sperm of any rival’s following ejaculate thus preventing being cuckolded by the female (Baker & Bellis, 1995).

The “trigger” for ejaculation - the prostatic pressure chamber concept

In the above description the tacit suggestion first proposed by Marberger in 1974 is that the “distension” of the prostatic urethra by the entering semen is the probable trigger for the ejaculation reflex. Even recent authors repeat the speculation in their account of ejaculation (Jannini et al., 2002). Is there evidence for this account? In fact rather than positive evidence for the suggestion there are four important pieces of experimental evidence against it! In brief these are:-

(i) $\alpha$–adrenergic blocking agents (phenoxybenzamine, phentolamine) prevents the discharge of semen into the urethra but they do not inhibit the initiation of ejaculation despite there being no fluid to ejaculate (dry orgasms) nor do they change significantly the subjective experience of orgasm (Brindley, 1983; Gerstenberg et al., 1990). Newman et al. (1982) claimed, however, that if the “pressure chamber” is not developed fully because of poor fluid volume (poor urethral distension?) there is a decreased sense of “inevitability”, the pleasure of release of the tension is decreased while the clonic contractions of the striated muscles are not as satisfying. Moreover, in a dry emission/ orgasm with a complete absence of secretions, they claimed the ejaculatory movements were fewer and the sensation felt “is a ghostly echo of a remembered orgasm”! The latter observation is not the conclusion of a healthy subject who during a number of normal orgasms and ejaculations then experienced a “dry one” with no semen ejaculated (Table 1).

(ii) According to the endorectal ultrasonographical study of ejaculation undertaken by Gil-Vernet et al. (1994) in a healthy 18 year-old heterosexual man,

TABLE I. Duration (by stopwatch), number of felt pelvic contractions (striated muscles) and orgasm grade (scale was 1 = poor to 10 = excellent) in a series of ejaculations obtained on different days by auto-manipulation in a healthy male subject. Note that in the “dry orgasm” where no semen was ejaculated (starred number 4) while the number of felt contractions and the duration were the least of the series the grade of orgasm was uninfluenced. See text for details

<table>
<thead>
<tr>
<th>Number</th>
<th>Duration orgasm (seconds)</th>
<th>Number of felt striated muscle contractions</th>
<th>Grade of Orgasm (1 – 10)</th>
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<td>1</td>
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<td>4* (“Dry orgasm”)</td>
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in none of his seven ejaculations studied was there any evidence of the existence of a “pressure chamber” where “semen accumulates in the infracollicular urethra (prostatic urethra) between closed internal and external sphincters before its expulsion towards the bulbous urethra.” In fact these authors reported that the emission phase and the expulsion or ejaculatory phase rather than being distinct and separate were actually “superimposed in time and were not as previously believed consecutive”. This study has generally been overlooked probably because it was based on a single subject but it suggests that all is not as simple as previous descriptions implied. Hermabessiere et al. (1999) also studied human ejaculation by continuous endorectal ultrasonography in 8 normal healthy volunteers. They constantly observed the expulsion of the contents of the seminal vesicles into the inframontanal urethra without prior ballooning of the prostatic urethra. These observations confirm the previous endorectal study in that a “pressure chamber” does not appear to be formed before prostatic contractions occur.

(iii) In a remarkable series of experiments using an inflatable balloon-tipped catheter that was inserted into the prostatic urethra of 14 healthy, conscious male volunteers Shafik and El-Sibai (2000) explored the effects of inflating the balloon with increments of saline on the electromyographic activity (emg) of the bcm recorded by a needle electrode. Initially the balloon was filled with saline in increments of 0.25 mls in the prostatic urethra then it was slowly withdrawn along the urethra and the distension was repeated in the membranous, bulbous and pendulous urethra monitoring its effects on the emg of the bcm. Balloon distension in the prostatic, membranous, or pendulous urethra did not cause any increase in the emg of the bcm. When in the bulbous urethra distension with 0.25 ml again caused no effects on the emg but with 0.5ml up to 1.5 ml there was an increased activity of the emg of the bcm (indicating contractions of the muscle), the response augmented with the increasing volume. If either the bulbous urethra or the bcm was anaesthetised the response did not occur. It was suggested that the bcm contraction was initiated by a reflex activated by large volumes of saline and that it probably reflected what occurs to propel the semen along from the bulbous urethra during ejaculation. The results also indicate that small volumes of semen would not activate the reflex and that if no fluid was present in the bulbous urethra (dry ejaculation) the reflex mechanism would not operate.

(iv) An early animal model for the study of sexual function in anaesthetised male and female rats was developed by Chung et al. (1988). Urethral stimulation by increased intraluminal pressure elicited erection, ejaculation and rhythmic contractions of the striated perineal muscles in male rats while in female rats vaginal and uterine contractions and rhythmic contractions of the perineal muscles were elicited, the activity was named the urethrogenital reflex. Because these responses showed great similarity to those seen in humans during sexual climax it was suggested as a model for this activity. Holmes and Sachs (1991) however, while they could block the contractions
activated by the intraurethral pressure in anaesthetised rats by intra-urethral lavage with a local anaesthetic (1% lidocaine) they could not block the contractions of the bcm during normal coitus in intact rats even when they had anaesthetised the whole length of the urethra with lidocaine. They concluded that urethral stimulation by the ejaculate does not contribute to the activation of the striated muscle component of the ejaculation reflex in copulating male rats.

It's clear from the above studies that the prostatic “pressure chamber” trigger concept needs to be examined in a more critical light and certainly more laboratory studies are needed. What is certain is there are definite occasions where the ejaculatory mechanism is activated yet no seminal fluids enter the prostatic urethra.

**Candidate sites for the ejaculation “trigger”**

Because of the above findings we should look for alternative or candidate sites for the ejaculation trigger. Possible sites could be i) the penile glans, ii) the spinal cord and iii) the brain (or of course the combination of brain and spinal cord).

i  *The penile glans*
Coital friction on the penile glans and its cover the prepuce are a major sexual sensory input. The innervation of the glans is mainly of free nerve endings with sparse numbers of corpuscular end organs (a ratio of 10 free nerve endings to one of the corpuscles). This creates a protopathic innervation- its sensitivity is crude and the feelings from stimulation are poorly localised but it has some sensitivity to temperature and mechanical contact. This structure and innervation suggest that the glans is unlikely to be a major candidate for the trigger. In fact free nerve endings are usually associated with the mediation of pain but the free nerve endings in the glans (bunched and curly) do not look like those in skin (straight and branched) and they may well be polymodal and different functionally. The prepuce and the frenum do, however, have capsulated corpuscular end organs. In the prepuce these are concentrated largely in its ridged bands which are lost if the prepuce is surgically removed at circumcision. As huge numbers of men are circumcised and are able to ejaculate quite normally, these cannot be part of the neural trigger for ejaculation.

ii  *The spinal cord*
Later studies of the urethrogenital reflex in rats (McKenna *et al.*, 1991) indicated that it was mediated through a reflex centre (spinal pattern generator) in the spinal cord a site consisting of interneurons that would produce sequential rhythmical bursts of activity. It is now known to be produced by a specialised set of neurons (lumbar spinothalamic neurons, LSt cells) in the spinal cord that receives the afferent impulses from the stretched urethra. These LSt neurons are positioned to relay ejaculation-related signals from the reproductive organs to the brain, they have projections to central
autonomic nuclei, areas crucial to the emission phase (the start of ejaculation) and links with sacral parasympathetic nuclei involved in genital glandular secretion. These neurons are knocked out by a highly selective neural toxin (SSP-saporin) then there is a complete disruption of the rat’s ejaculatory behaviour but all other aspects of sexual behaviour were unaffected. The hypothesis was that these cells played a fundamental role in the ejaculation mechanism and that they constituted part of a spinal ejaculation generator (Truitt & Coolen, 2002). This generator coordinates sympathetic, parasympathetic and motor efferents that neurally mediate emission and ejaculation. Moreover, the generator integrates these outputs with summed inputs arising from the previous sexual behaviour; supraspinal sites (nucleus paragigantocellularis, hypothalamic paraventricular nucleus, medial preoptic optic area) both inhibit and excite the generator cells. The LSt cells send projections to the parvocellular subparafascicular thalamic nucleus probably contributing to the reward properties of ejaculation. All these factors suggest that the spinal ejaculation generator could well be the “trigger” for ejaculation in that when the summation of all the positive arousing stimuli becomes greater than the negative inhibitor ones, its neurons discharge to create the rhythmic contractile activity of the striated pelvic musculature (Coolen et al., 2004). This could occur without having to have an input from a distended prostatic urethra solving the problems of the previous experimental difficulties. What cautions do we have to employ before we can accept this to also be the trigger mechanism for humans? Firstly, although there is clinical evidence that the spinal cord is involved in ejaculation and orgasm (Jocheim & Wahle, 1970; Sipski, 1998) there is as yet no data that such a spinal generator area exists in the human and second, there are claimed differences between sexual arousal observed in male rat and human brains (see below for details).

### The brain

Before the advent of brain imaging it was possible to speculate that there was a single centre in the male brain (the Holy Grail of ejaculation research) that when fully activated initiated ejaculation and orgasm (see Levin, 2003). With the introduction of fMRI and PET, mapping and registering the activity in parts of the human brain during various behaviours became possible and the hunt for the site could begin. Holstege et al. (2003) were the first to use PET tomography to measure the changes in the rCBF during ejaculation and associated feelings of orgasm in human subjects induced by manual stimulation of the penis by the subject’s female partner. Primary activation (the most intense area of increased rCBF) was found in the mesodiencephalic transition zone which encompasses a number of structures (midline, ventroposterior, intralaminar thalamic nuclei, subparafascicular nucleus, zona incerta, midbrain lateral central tegmental field (LCTF) and ventral tegmental area). Because of the limited spatial resolution of the PET technique it is not possible to distinguish specific brain regions within this area. The area is involved in a wide variety of rewarding behaviours. Other areas strongly activated were the claustrum, rostral insula, striatum (ventrolateral putamen) anterior nucleus of
right thalamus. Parts of the neocortex and the cerebellum were also activated. The amygdala however was deactivated (unlike that in women). It has been suggested that this deactivation could be involved in the post-ejaculatory refractory time experienced in males (Levin, 2003) when ejaculation and orgasm cannot be immediately re-induced.

The large number of areas activated at and during ejaculation/orgasm suggest that there is not likely to be a single brain centre for ejaculation (the trigger centre) as previous authors have speculated but rather that various areas interconnect to send the final message down to the spinal cord generator cells to instigate ejaculation.

Holstege et al. (2003) interestingly point out that in rodents the medial preoptic area (MPOA), the bed of the stria terminalis and the amygdala are all activated during ejaculation but increased rCBF was not found in any of these regions in the human subjects. In fact, in male humans as described above, the amygdala actually showed a decrease in rCBF! This indicates real differences exist between human and rodent brain sexual arousal and again suggests caution in relation to assuming that because the spinal ejaculation generator is present in the rat it must have a presence in the human spinal cord. Unfortunately, we will just have to wait and see.

References


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