Electromyographic study of ejaculatory mechanism
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Introduction
During sexual intercourse, penile thrusting continues until ejaculation occurs. Penile thrusting rhythmically stimulates the glans penis (GP) and eventually results in orgasm and ejaculation. These responses are under the control of numerous central and peripheral neural systems. The central supraspinal systems are mainly localized in the limbic system, in the hypothalamus and its nuclei (Argiolas & Melis, 2003; Motofei & Rowland, 2005). Neural information travels through the brain stem, spinal cord and autonomous nervous system to the genital apparatus. The role of neuropeptides, brain monoamines and nitric oxide has been emphasized in the control of sexual desire, erection, copulation, ejaculation, and sexual satiety (Stahl, 2001). The post-ejaculatory refractory period is accompanied by a greater penile sensory threshold but with no change in the values of the sacrally evoked response and cortical somatosensorily evoked potential (Yilmaz & Aksuy, 2000).

At orgasm, spasm occurs in various muscle groups including the rectus abdominis muscles, sternomastoid and facial musculature (Masters & Johnson, 1966; Bancroft, 1989). The muscle tension declines rapidly once orgasm has passed. Masters & Johnson (1966, 1970) differentiated two stages of ejaculation. In stage I, or the emission stage, smooth muscle contraction occurs in the vas deferens of the testis, epididymis, vas deferens, together with the seminal vesicles (SVs), prostate and vasal ampulla (VA). These structures are sympathetically controlled by pre-sacral and hypogastric nerves (McLeod & Reynolds, 1973). Stage II, or the ejaculation stage, is marked by relaxation of the external urethral sphincter as well as rhythmic contractions of the bulbocavernous muscle (CM), seminal vesicle (SV) and vasal ampullary (VA) contractions at ejaculation are said to be reflex mechanisms (ejaculatory reflex), which have been scarcely dealt with in the literature. We investigated the hypothesis that contraction of the CMs, SVs and VA at ejaculation is a reflex action. The electromyographic (EMG) activity of CM, SV and VA during ejaculation was recorded in 28 healthy men. The test was repeated after separate anaesthetization of the glans penis (GP), CMs, SVs, and VA in the pre-ejaculatory period. Latent ejaculatory time (LET) was calculated. CMs showed no EMG activity until rigid erection phase was reached. SVs and VA exhibited resting EMG activity which increased gradually with different stages of erection. At ejaculation, CMs, SVs and VA showed two to four intermittent contractions. The mean LET was 1.3 ± 0.2 sec. GP anaesthetization led to the disappearance of CM, SV and VA EMG activity at ejaculation, while bland gel did not affect EMG activity. CMs, SVs and VA when anaesthetized in the pre-ejaculatory period exhibited no EMG activity at ejaculation, while saline did not affect EMG activity. Increased EMG activity of CM, SV and VA apparently denotes increase in their contractile activity. CM, SV and VA contraction on GP stimulation and ejaculation are assumed to be reflex actions and are mediated through the ‘glans-cavernosovesicular reflex’ (GCVR) which presumably represents the ejaculatory reflex. Changes in LET or evoked response would indicate a defect in the reflex pathway. The GCVR might act as an investigative tool in diagnosing erectile dysfunction, provided further studies are performed in this respect.
Ejaculation mechanism and reflex

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ischiocavernous muscles (BCM, ICM), the sphincter urethrae and the urethral bulb. This results in the forceful expulsion of seminal fluid through and out of the urethra (Peterson & Stener, 1970; Seftel & Althof, 1997). These muscles are under somatic control and are innervated by the pudendal nerve (Biester & Howards, 1988).

Contractions of BCM and ICM, SVs and VA during ejaculation are claimed to occur through a reflex action, designated the ‘ejaculatory reflex’ (Anderson & Wagner, 1995; Lue, 1998). The identification of the reflex mechanism of the above-mentioned actions at ejaculation was however incompletely addressed in the literature. We hypothesized that contractions of the BCM, ICM, SVs and VA at ejaculation are mediated through a reflex action. This hypothesis was investigated in the current study.

Materials and methods

Subjects

Twenty-eight healthy men (mean age 38.6 ± 7.3 SD years, range 30–48) volunteered for the study and were paid. They were married, sexually active and had fathered children. They had normal erection and did not complain of erectile dysfunction in the past or at the time of enrolment. Physical examination including neurological examination showed normal results. The laboratory workup comprising blood picture, hepatic and renal function tests and electrocardiogram was unremarkable. The subjects gave an informed consent, and the study was approved by the Cairo University Faculty of Medicine Review Board and Ethics Committee.

Methods

The electromyographic (EMG) response of the BCM, ICM, SVs and VA to ejaculation achieved by GP stimulation was recorded. Erection and ejaculation were induced by GP electrovibration (Schellan, 1968). The EMG activity of the ICM and BCM was recorded by means of a concentric EMG needle electrode (type 13L49; DISA, Copenhagen, Denmark) measuring 45 mm in length and 0.65 mm in diameter. The ischiopubic ramus of the ICM with the overlying crus penis was palpated and the needle inserted into the ICM lying on its medial aspect. A second identical needle was placed in the BCM; the penile bulb was palpated and the needle electrode introduced into the muscle overriding it. A ground electrode was applied to the thigh.

Likewise, EMG needle electrodes were introduced into the SVs and VA. With the patient lying in the left lateral position, and under no anaesthesia, a fenestrated rectoscope was introduced into the anal canal. While the SVs and VAs were being exposed, an electrode was inserted into either of them. The correct position of the needle electrode in the SVs or VA is known by the burst of activity heard from the loudspeaker and visualized on the oscilloscopic screen as the needle entered the muscle fibres of the organ. Furthermore, the recorded EMG activity of the SVs was different from that of the VA as regards the amplitude of the motor unit action potentials (MUAPs).

The EMG activity was displayed on the oscilloscope of a standard EMG apparatus (Type MES; Medelec, Woking, UK). Films of the potentials were taken on light-sensitive paper (Linagraph type 1895; Kodak, London, UK) from which measurements of the latency of the reflex and amplitude of MUAPs were made. The EMG signals were also stored on an FM tape recorder (Type 7758A; Hewlett-Packard, Waltham, MA, USA) for further analysis as required.

The correct position of each needle in the corresponding structure was monitored by the burst of activity heard from the loudspeaker and visualized on the oscilloscopic screen as the needle entered the muscle fibres.

Latent ejaculatory time (LET)

The time lapse between the onset of intermittent contractions of the CMs, VA and SVs, as shown in the oscilloscope of the EMG apparatus, and the start of ejaculation was calculated. This period represented the LET. In the pre-ejaculatory period, two types of contraction were identified: sustained and intermittent. It needs to be stressed that the recording was performed a short period before the start of intermittent VA, SV and CM contractions. The intermittent BCM, ICM, VA and SV contractions were followed by ejaculation.

GP, CM, VA and SV anaesthetization

To test whether BCM, ICM, SV and VA contraction at ejaculation is a reflex or a direct action, the GP was anaesthetized. It was rubbed with lidocaine gel while in the rigid erection phase (pre-ejaculatory period) and while both CMs, SVs and the VA were recording EMG activity. The time elapsed between lidocaine topical application and GP stimulation was 10 minutes. GP electrovibration was performed up to the instant where ejaculation occurred and the EMG of BCM, ICM, SV and VA during ejaculation was recorded. After a few days, the test was repeated with bland gel instead of lidocaine gel.

Two to three days later, the BCM, ICM, SVs and VA were separately anaesthetized while the penis was in the rigid erection phase. Two millilitres of 2% lidocaine was injected into each of the BCM, ICM, SVs and VA; the injection was performed into the area surrounding the
needle electrode that had already been applied to each of them. Ten minutes after lidocaine injection, the response of the anaesthetized SVs, VA and CMs to GP electrovibration at ejaculation was recorded. The test was repeated 2–3 days later using normal saline instead of lidocaine.

To ensure reproducibility of the results, the tests were repeated at least twice in the individual subject, and the mean value was calculated. The parameters for which the mean value was calculated comprised the EMG activity of the CMs, SVs and VA during ejaculation and before and after anaesthesia of these structures and GP. The results were analysed statistically using analysis of variance (ANOVA) and values were given as mean ± SD. Differences assumed significance at \( p < 0.05 \).

**Results**

Adverse side effects were not encountered during or after performance of the tests and all the subjects were evaluated. Basal electric activity was not recorded from the two CMs, but for the SVs a mean of 76.2 ± 7.2 \( \mu V \) and for the VA a mean of 68.4 ± 6.9 \( \mu V \) (Fig. 1, Table 1) was recorded. Upon GP electrovibration, the penis became tumescent, then fully erect. During the periods of tumescence and full erection, the CMs did not display EMG activity while the SVs and VA exhibited increased EMG activity (Table 1). At full erection, the SVs recorded mean MUAPs of 142 ± 11.3 \( \mu V \), and the VA of 164.8 ± 10.7 \( \mu V \) (Fig. 2).

At rigid erection, both CMs exhibited EMG activity, while the SVs and VA exceeded the EMG activity they had recorded at full erection (Fig. 3, Table 1). The EMG recorded mean MUAPs of 218.6 ± 21.7 \( \mu V \) for the BCM, 206.3 ± 18.4 \( \mu V \) for the ICM, 186.6 ± 18.4 \( \mu V \) for the SVs and 216.4 ± 23.7 \( \mu V \) for the VA (Fig. 3, Table 1). The electric activity (MUAPs) of both CMs, the SVs and the VA remained constant during the period of rigid erection. During the last few seconds preceding ejaculation, i.e. the pre-ejaculatory stage, the MUAPs of both CMs, the SVs and VA exhibited a progressive increase until, at ejaculation, the BCM recorded mean MUAPs of 642.7 ± 42.8 \( \mu V \), the ICM of 688.6 ± 46.8 \( \mu V \), the SVs of 386.4 ± 27.6 \( \mu V \) and the VA of 493.7 ± 33.9 \( \mu V \) (Table 1). The increase in the MUAPs of both CMs, the SVs and the VA occurred intermittently at intervals of 0.6–1.1 sec. The MUAPs reached the aforementioned levels during the periods of increase, and then dropped to basal levels. The number of intermittent increases in EMG activity of BCM, ICM, SVs and VA ranged from 2 to 4 (mean 2.8 ± 0.6). The EMG activity then returned to basal levels. The LET recorded a mean of 1.3 ± 0.2 sec (range 1–2).

**Effect of CM, SV and VA anaesthetization on the response of BCM, ICM, SVs and VA**

When the GP was rubbed with lidocaine gel while the penis was in the pre-ejaculatory phase and the CMs, SVs and VA were exhibiting increased EMG activity, the EMG activity of the CMs disappeared, while the SVs and VA recorded the basal EMG activity. Repetition of the above-mentioned test using bland gel instead of lidocaine gel did not affect the EMG activity of the CMs, SVs or VA.

Glans penis stimulation while the BCM, ICM, SVs and VA were being individually anaesthetized did not affect the EMG activity of the BCM or ICM while the SVs and VA retained their resting basal EMG activity. Using normal saline instead of lidocaine in the aforementioned test did not affect the results. All the aforementioned results were reproducible with no significant difference \( (p > 0.05) \) when the test was repeated in the individual subject.

**Discussion**

Erectile response is a vascular event initiated by neuronal action and maintained by a complex interplay between vascular and neurological and perhaps humoral phenomena (Anderson & Wagner, 1995; Lue, 1998). The phases of the erectile process consist of tumescence, full erection

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**Table 1** Motor unit action potentials of the bulbocavernosus (BCM) and ischiocavernosus (ICM) muscles as well as seminal vesicles (SV) and vassal ampulla (VA)

<table>
<thead>
<tr>
<th></th>
<th>BCM</th>
<th>ICM</th>
<th>SV</th>
<th>VA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Range</td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td>Basal</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Full erection</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Rigid erection</td>
<td>218.6</td>
<td>21.7</td>
<td>164–253</td>
<td>206.3</td>
</tr>
<tr>
<td>Ejaculation</td>
<td>642.7</td>
<td>42.8</td>
<td>428–812</td>
<td>688.6</td>
</tr>
</tbody>
</table>

Values given as the mean ± SD.
and rigid erection which terminates in orgasm and ejaculation. It is claimed that ejaculation is a reflexogenic process, i.e. it is manipulated through a reflex action (Anderson & Wagner, 1995; Lue, 1998). However, the mechanism of action has hitherto not been completely explored and needs to be discussed.

The orgasmic phase ends with ejaculation. The penile ejaculatory reaction is manifested by regularly recurring involuntary but coordinated contractions of various groups of muscles which include sphincter urethrae, bulbo- or ischio-cavernosus muscles, puborectalis muscle, the transverse superficial and the deep perineal muscles (Peterson & Stener, 1970).

The current study has shown that the SVs and VA exhibited resting electric activity. This EMG activity increased during tumescence and full erection, probably indicating increased contractile activity of the SV and VA musculature; it reached its maximum during ejaculation. Meanwhile, the CMs exhibited EMG activity only at the stage of rigid erection and reached their maximal EMG activity at ejaculation. These muscles showed no resting electric activity. The BCM and ICM, the SVs and the VA all exhibited maximal EMG activity during the ejaculatory phase, and basal activity between the semen jets.

The LET determines the time lapse between BCM, ICM, SV and VA contraction and ejaculation. It may be abnormally prolonged which would point to a disorder in the GP, the CMs, SVs and the VA or in the nerve pathway between these structures. Therefore, the LET may have the potential to serve as an investigative tool in the diagnosis of such conditions.

The CMs exhibited involuntary contractions at ejaculation. These contractions seem to be reflexogenic, a phenomenon that needs to be discussed.

The glans-cavernosovesicular reflex (GCVR)

The contraction of CMs, SVs and VA upon GP electrovibration postulates a reflex relationship between the two
actions. The constancy of this relationship is assured by reproducibility. Meanwhile, the reflex nature of this relationship is evidenced by the absence of the CM/SV/VA response upon individual anasthetization of the assumed two arms of the reflex arc: the GP as one arm and the CMs, SV or VA as the other arm. We call this reflex relationship the GCVR. It seems that GP electrovibration stimulates mechanoreceptors within it and the impulses pass along the pudendal nerve to the spinal cord which eventually sends impulses to the CMs, SVs and VA affecting their contraction. It may be useful to denote that lidocaine does not block the muscle activity but rather the sensory fibres (C and A α-fibres) which are responsible for pain and reflex activity (Yokoyami et al., 2000; Silva et al., 2002). It appears that penile thrusting during coitus stimulates the GP sensory receptors continuously until upon reaching a certain level of stimulation, the GCVR seems to be evoked with a resulting CMs-SVs-VA contraction and ejaculation. During the ejaculatory phase, the intermittent contractions of the two CMs assist not only in ejaculating the semen but also in keeping the penis erect by compressing the dorsal penile veins. The GCVR seems to represent the ejaculatory reflex. Clinical studies investigating the ejaculatory reflex process were performed by other investigators (Wieder et al., 2000; Yang & Bradley, 2000). The somatic reflex innervation of the BCM was determined. Three distinct somatic bulbocavernous reflexes were detected which are components of normal ejaculation (Yang & Bradley, 2000). Wieder et al. (2000) investigating nerve pathways which are necessary to achieve ejaculation in men with spinal cord injury showed that the ejaculatory response to penile vibratory stimulation requires the presence of intact dorsal penile nerves.

It may be argued that ejaculation is not only evoked by the GCVR. It can occur during sleep with nocturnal erection, or by psychogenic stimulation like smell, light, hearing or touch, and also by stimulation of other body parts, but without GP stimulation. However, the current study focused on demonstrating the mechanism of ejaculation that occurs with sexual intercourse. In sexual intercourse, continuous GP stimulation during penile thrusting would evoke the GCVR with a resulting ejaculation.

Diagnostic significance of the GCVR

The GCVR apparently examines the response of the CMs, SVs and VA to GP stimulation and in particular at ejaculation. The reflex may prove to be of diagnostic significance in erectile function disorders. The test could be performed by inducing erection and examining the EMG response of CMs, SVs and VA at ejaculation. Detectable changes in the MUAPs of the evoked response or in LET would indicate a defect in the reflex pathway that could be a nerve or muscle damage. For this reason, measurements of the reflex-evoked potentials could be added to the current andrological investigations when patients with ejaculatory dysfunction need to be monitored.

In conclusion, CMs, SVs and VA contract upon GP stimulation and ejaculation. This contraction did not happen upon anasthetization of the GP or the CMs, SVs and the VA. The reaction of the CMs, SVs and the VA to gut stimulation seems to be reflex and mediated through the GCVR which apparently represents the ejaculation reflex. Changes in the LET or evoked response would indicate a defect in the reflex pathway. The GCVR might thus act as an investigative tool in the diagnosis of erectile dysfunction, provided further studies are performed in this respect.

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References


