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Effect of External Anal Sphincter Contraction on the Ischiocavernosus Muscle and Its Suggested Role in the Sexual Act

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ABSTRACT: Whereas the bulbocavernosus muscle shares its contractile activity with the external anal sphincter (EAS), the response of the ischiocavernosus muscle (ICM) to EAS contraction could not be traced in the literature. We investigated the hypothesis that the ICM contracts reflexly upon EAS contraction. The response of the ICM to EAS squeeze and stimulation was recorded in 21 healthy volunteers (13 men, 8 women, age 36.8 ± 10.7 [SD] years). An electromyographic (EMG) needle (stimulating) electrode was introduced into the EAS and another (recording) one was inserted into the ICM. The test was repeated after individual anesthetization of the EAS and ICM and after muscle infiltration with normal saline instead of lidocaine. EAS electrostimulation (10 stimuli, 200 μs duration, 0.2 Hz frequency, 0–100 mA intensity) produced an increase of ICM EMG activity to a mean of 267.8 ± 42.7 μV, whereas anal squeeze effected an increase to a mean of 224.5 ± 45.3 μV. The ICM did not respond to stimulation of the EAS after individual anesthetization of the ICM and EAS, but it did after saline infiltration. The results were reproducible. ICM contracted upon EAS contraction. This effect seems to be mediated through a reflex that we call ''anocavernosal excitatory reflex.'' The ICM lever action is suggested to share in the erectile mechanism by elevating the penile shaft to above the horizontal level. The reflex may prove of diagnostic significance in sexual function disorders, a point that needs further study.

Key words: Electromyography, erection, impotence, corpora cavernosa, action potentials, erectile dysfunction.

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The mechanism of the sexual act is intricate and not fully understood (Burnett, 1995; Meuleman and Diemont, 1995; Anderson, 2001). The 2 cavernosus muscles, bulbo- and ischiocavernosus, are the principal muscles that act during sexual intercourse. Penile buffeting of the cervix uteri as well as vaginal distension during coitus induce reflex contraction of the cavernosus muscles; these actions are mediated through the cervicocavernosus (Shafik, 1993a) and vaginocavernosus reflexes (Shafik, 1993b). Cavernosus muscles' contraction enhances both penile and clitoral erection owing to the compression exerted on the deep dorsal vein of the penis or clitoris and on the erectile cavernosus tissue (Shafik, 1993a,b). Moreover, it helps in squeezing the semen, which fills the penile urethra, into the vagina while the penis is being withdrawn from the vagina after ejaculation. Furthermore, rhythmic contractions of the bulbo- and ischiocavernosus muscles occur at orgasm and are believed to assist in the process of ejaculation (Shafik, 1993a,b).

The sexual sensory signals are mediated through the pudendal nerve and sacral plexus to the sacral segments of the spinal cord, and are then transmitted to the cerebrum (Lue et al, 1984; Lepor et al, 1985; Steers, 1990). Also, local reflexes integrated in the sacral and lumbar spinal cord may share in the sexual reaction (Bradley and Teague, 1977; Benson et al, 1980; Tabatabai et al, 1986).

The bulbocavernosus reflex exhibits the response of the cavernosus muscles or the external anal sphincter (EAS) to stimulation of the glans penis (GP) or clitoris (Ertekin and Ree, 1976; Siroky et al, 1979; Bird and Hanno, 1998). It is used as a diagnostic tool in the diagnosis of erectile disorders.

A previous study has demonstrated that the base loop of the EAS extends uninterrupted across the perineum to the bulb of the penis where it becomes continuous with the bulbocavernosus muscle (BCM) (Shafik, 1999). While lying over the bulb, the muscle bundles were arranged in 3 groups: 1 median and 2 lateral. The median fibers form the ''retractor penis muscle,'' which is inserted into the corpora cavernosus, while the lateral fibers or the ''compressor bulbae muscle'' are inserted into the perineal membrane (Shafik, 1999). Upon glans stimulation, both the conjoined EAS and BCM contract synchronously with identical latency and action potentials (Shafik, 1999). The BCM is an integral part of the EAS and the muscle in its entirety is given the name ''anogenital muscle'' (Shafik,

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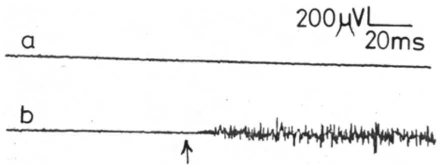

Figure 1. Electromyographic activity of the ischiocavernosus muscle **(a)** at rest (no activity); **(b)** on electrostimulation of the external anal sphincter. ↑ indicates electrostimulation.

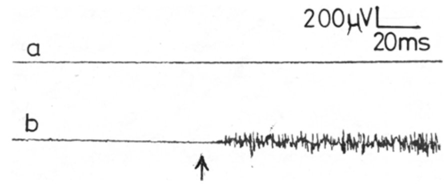

Figure 2. Electromyographic activity of the ischiocavernosus muscle **(a)** at rest (no activity); **(b)** on external anal sphincter squeeze. ↑ indicates anal sphincter squeeze.

1999) since it plays a dual and synchronous role in fecal control and sexual response.

Although the BCM shares its contractile activity with the EAS, the response of the ischiocavernosus muscle (ICM) to EAS contraction could not be traced in the literature. We hypothesized that the ICM contracts reflexly upon EAS contraction, thus assisting the BCM in the mechanism of erection. This hypothesis was investigated in the current study.

Materials and Methods

Subjects

Twenty-one healthy volunteers (13 men, 8 women, mean age 36.8 ± 10.7 [SD] years, range, 27–50) were enrolled in the study after giving an informed consent. All of the subjects were sexually active and had no anorectal or genitourinary complaint in the past or at the time of enrollment. Six of the women were multiparous with normal deliveries and 2 were nulliparous; they had normal menses and no gynecologic complaint. The physical examination including procto-, neuro-, and gynecologic assessment was normal. Examination of blood counts as well as renal and hepatic function and electrocardiography showed normal results. The study was approved by the Cairo University Faculty of Medicine Review Board and Ethics Committee.

Methods

The response of the ICM to EAS stimulation or squeeze was studied. With the subject lying supine, the knee and hip joints flexed and the thigh abducted, a concentric electromyographic (EMG) needle electrode (type 13L49 Disa, Copenhagen, Denmark) measuring 45 mm in length and 0.65 mm in diameter was introduced into each of the ICM and EAS. For the ICM, the ischial ramus with the overlying crus penis or clitoris was palpated, and the needle electrode was inserted into the muscle on the medial aspect of the ramus to a depth of 0.5 cm. An identical needle electrode was introduced 1 to 1.25 cm deep into the EAS and at a distance of 0.75 to 1 cm lateral to the anal orifice.

The EMG activity was displayed on the oscilloscope of a standard EMG apparatus (type MES, Medelec, Woking, United Kingdom). Films of the potentials were taken on light-sensitive paper (linagraph type 1895; Kodak, Rochester, NY) from which measurements of the latency of the reflex and motor unit action potentials were made. The EMG signals were, in addition, stored on an FM tape recorder (type 7758A; Hewlett-Packard, Waltham, Mass) for further analysis as required.

The correct position of the needle electrodes in the muscle was monitored by the burst of activity heard from the loudspeaker and visualized on the oscilloscopic screen as the needle entered the muscle. Fine adjustments of the needle position were made while the EMG response to needle insertion was observed on the chart recorder. Multiple recordings were done to assure reproducibility.

Before performing the experiment, we tested all subjects for the normality of the myoelectric activity of the EAS and ICM by means of stimulating them with a needle electrode that had been introduced into each of the 2 muscles separately and then recording the motor unit action potentials with the recording needle electrode. All the subjects recorded normal EMG activity of the EAS and ICM.

Anesthetization of the EAS and ICM

To test whether the response of the ICM to EAS stimulation was a direct or reflex action, the ICM was anesthetized by injecting 2 mL of 2% lidocaine into the muscle around the electrode. The response of the anesthetized ICM to EAS stimulation was recorded after 20 minutes from lidocaine injection, and 3 hours later when the anesthetic effect had dissipated. The test was repeated after EAS anesthetization and after individual infiltration of the ICM and EAS with normal saline. The aforementioned experiments were also performed on the contralateral ICM.

To ensure reproducibility of the results, the recordings were repeated at least twice in the individual subject, and the mean value was calculated. The results were analyzed statistically using the Student's t test and values were given as the mean ± SD. Significance was ascribed to $P < .05$.

Results

The study was completed in all the subjects with no adverse side effects.

The ICM did not exhibit resting electrical activity (Figure 1). EAS stimulation by a repeated series of 10 electrical stimuli of 200 μs duration at a frequency of 0.2 Hz and intensities between 0 and 100 mA produced an increase in the EMG activity of the ICM to a mean amplitude of motor unit action potentials (AMUAPs) of 267.8 ± 42.7 μV (range, 186–336; Figure 1). Upon anal squeeze, the ICM showed an increase of the AMUAPs to a mean of 224.5 ± 45.3 μV (range, 164–295; Figure 2). Repeated and successive EAS stimulation did evoke the

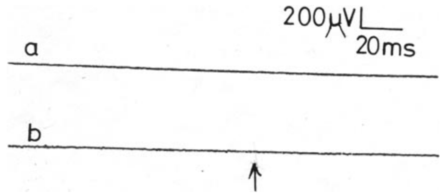

Figure 3. Electromyographic response of the ischiocavernosus muscle to electrostimulation of the anesthetized external anal sphincters (EAS) **(a)** at rest; **(b)** on EAS electrostimulation. ↑ indicates electrostimulation.

ICM response. The response was weaker in women than in men and in the multiparous than nulliparous women, although the difference was not significant ($P > .05$). The latency of the response measured from the start of EAS stimulation to the first deflection of the reflex muscle action potential complex recorded a mean of 17.4 ± 1.6 ms (range, 14–19) on EAS electrostimulation (Figure 1) and 17.7 ± 1.6 (range, 13–19) for anal squeeze (Figure 2).

Stimulation of the anesthetized EAS did not evoke the ICM response (Figure 3). Nor did EAS stimulation induce increase of the EMG activity of the anesthetized ICM. The response returned after 3 hours from lidocaine administration when the anesthetic effect had disappeared. In contrast, the ICM responded to stimulation of the saline-infiltrated EAS and the saline-infiltrated ICM responded to EAS stimulation.

The aforementioned results were reproducible with no significant difference when the test was repeated in the individual subjects.

Discussion

Although it could be demonstrated in a previous study that the BCM is an integral part of the EAS and both contract synchronously (Shafik, 1999), the current study could shed some light on the relation of the ICM to the EAS. The ICM arises from the ischial tuberosity and ramus and is directed upward and forward toward the corpora cavernosa into which it inserts (Snell, 1995) (Figure 4). The ICM exhibited increased EMG activity on EAS electrical stimulation or squeeze. This increased EMG activity of the ICM is indicative of ICM contraction.

Role of the ICM in Corporeal Congestion and Erection

During sexual intercourse, BCM contraction was found to be associated with synchronous contraction of the EAS (Shafik, 1999). EAS contraction presumably acts to prevent the leakage of flatus or fluid stools at coitus that might lead to coitus interruptus. Meanwhile, the current study has demonstrated that EAS contraction effected ICM contraction, which appears to have a role in the erectile mechanism. The ICM, as it is directed forward from its origin in the ischial tuberosity with upward inclination toward the corpora cavernosa where it is inserted, functions on contraction to pull the penis backward against the ischial tuberosity. The penile root consists of the 2 crura and the bulb. By pulling the penis backward, the ICM appears to fix the penile shaft to its root against the ischial tuberosity. This action seems to support and strengthen the penile shaft during vaginal penetration and thrusting. Meanwhile the ICM, inserting in a ''belt'' form across the proximal part of the 2 corpora cavernosa, presumably acts on contraction as a constricting band that appears to compress the deep dorsal vein of the penis. This seems to hamper the cavernosal venous return, and thus, augments penile erection. Furthermore, studies have shown that ICM contractions were associated with elevated intracavernous pressure and that ICP changes were always in phase with changes in the ICM EMG (Lavoisier et al, 1986). Results of the study suggest involvement of the ICM in the process of penile rigidity; the muscle produces suprasystolic intracavernous pressure, which would result from pudendal motoneural activity (Schmidt and Schmidt, 1993; Bernabe et al, 1999). The studies have also shown that the GP erections were associated with suprasystolic corpus spongiosum pressure (CSP) peaks concurrent with bulbospongiosus (BS) muscle bursts (Schmidt et al, 1995). Measures of CSP and BS EMG activity were found to vary significantly with glans erection intensity (Schmidt et al, 1995).

Role of the ICM in Penile Thrusting

ICM contraction seems to share in the mechanism of penile lifting to above the horizontal level during erection. While flaccid, the penis points downward (Figure 4) but on erotic stimulation, is elevated upward to a near hori-

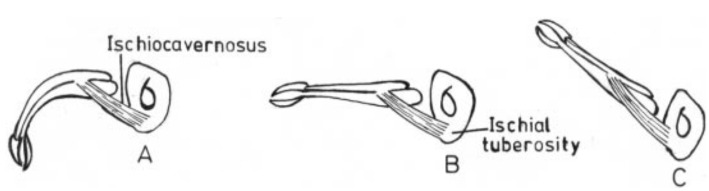

Figure 4. The mode of action of the ischiocavernosus muscle during erection. **(A)** The muscle while the penis is flaccid and pointing downward; **(B)** the muscle while the penis is tumescent and horizontally lying; **(C)** diagram illustrating the lever action of the contracting ischiocavernosus muscle on the tumescent penis. Muscle contraction effects elevation of the penile shaft to above of the horizontal level.

zontal position of varying degrees. This penile position seems to be effected jointly by the increased arterial blood flow and the inhibited venous drainage that induces cavernous tissue turgidity and tumescence. The resulting high intracorporeal pressure stretches and elevates the penis to subhorizontal or horizontal level. The elevation effect of the increased intracorporeal pressure on the flaccid penis is similar to the stretch and elevation of an empty glove's finger on inflation.

Originating from the ischial tuberosity and ramus, the ICM passes forward with an upward inclination and terminates at the sides and dorsum of the corpora cavernosa (Figure 4). It thus acts as a lever that probably comes into action after the penis is elevated horizontally by both the turgidity and increased intracorporeal pressure. While the penis is in the horizontal position, the lever action of the contracting ICM effects upward elevation of the penile shaft to above the horizontal level and acts to keep the penis in this position during penile thrusting at coitus (Figure 4).

The Anocavernosal Excitatory Reflex

The ICM contraction upon contraction of the EAS postulates a reflex relation between the 2 actions. The constancy of this relation is evidenced by its reproducibility, and its reflex nature is confirmed by its absence upon anesthetization of either the EAS or the ICM, the 2 possible arms of the reflex arc. We call this hitherto unrecognized reflex relation the anocavernosal excitatory reflex. Lidocaine blocks C and A α-fibers, which are responsible for pain and reflex activity (Yokoyami et al, 2000; Silva et al 2002). It appears that, on EAS stimulation, impulses pass to the sacral spinal cord, which eventually sends impulses to the ICM, effecting its contraction. Impulses are apparently transmitted along the pudendal nerve. Anesthetization of the EAS or ICM presumably blocks their innervation so that nerve impulses cannot be transmitted from either the EAS to the spinal cord or from the spinal cord to the ICM.

Diagnostic Role of the Anocavernosal Excitatory Reflex

The anocavernosal reflex may prove to be of diagnostic significance in erectile dysfunction. Detectable changes in the latency or the MUAPs of the reflex would indicate muscle or nerve damage from a disease of the spinal cord, spinal nerve roots or peripheral nerves, or from a central lesion. Further studies may prove that the anocavernosal excitatory reflex has the potential of an effective investigative tool in the diagnosis of sexual disorders.

On the other hand, the anocavernosal reflex seems to be more constant than the bulbocavernosus reflex, as the former is reproducible in the individual subject. The reported constancy of the bulbocavernosus reflex in normal subjects was questioned by investigators who found it inconstant and of variable latencies in the individual subject (Rattner et al, 1958; Lavoisier et al, 1989). The bulbocavernosus reflex is, however, currently used as a test to identify whether an erectile dysfunction is of neurogenic origin (Siroky et al, 1979; Ertekin et al, 1985). In this regard, the anocavernosal reflex could replace the bulbocavernosus one, a point that needs further study.

In conclusion, the ICM contracted on EAS contraction. This effect is suggested to be reflex and mediated through the anocavernosal excitatory reflex. ICM contraction seems to share in the erectile mechanism by elevating the penile shaft to above the horizontal level by means of its lever action. The reflex might prove of diagnostic significance in sexual function disorders, a point that needs further study.

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