Effect of Micturition on the External Anal Sphincter: Identification of the Urethro–Anal Reflex

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Abstract

Background/Objective: A study on the response of the external anal sphincter (EAS) to the passage of urine through the urethra during micturition could not be found in the literature. We investigated the hypothesis that urine passage through the urethra effects EAS contraction to guard against possible flatus or stool leakage during micturition.

Methods: The study was performed in 23 healthy volunteers (age, 38.6 ± 10.8 [SD] years; 14 men and 9 women). The EAS electromyogram (EMG) was performed during micturition by surface electrodes applied to the EAS. Also, the EAS EMG response to urethral stimulation by a catheter-mounted electrode was registered. The test was repeated after individual anesthetization of the EAS and urethra.

Results: The EAS EMG recorded a significant increase ($P < 0.01$) during micturition and on urethral stimulation at the bladder neck. Stimulation of the prostatic, membranous, or penile urethra produced no significant change in the EAS EMG. Urethral stimulation after individual EAS and urethral anesthetization did not cause any changes in the EAS EMG.

Conclusions: Urine passing through the urethra or urethral stimulation at the vesical neck produced an increase in the EAS EMG, which presumably denotes EAS contraction, which seems to guard against flatus or fecal leakage during micturition. EAS contraction on urethral stimulation is suggested to be mediated through a urethro–anal reflex. Further studies on this issue may potentially prove the diagnostic significance of this reflex in micturition and defecation disorders.

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Key Words: Electromyography; Sphincter reflex; Flatus; Stools; Urethra; Defecation; Micturition

INTRODUCTION

The mechanisms of defecation and urination are intricate and are under the control of voluntary and reflex actions (1–6). As the rectum and urinary bladder become full, stimulation of the mechanoreceptors in their walls trigger the evacuation reflexes with a resulting rectal or vesical contraction, internal sphincter relaxation, and evacuation (1,2). The bladder and rectum receive their sympathetic supply from L1, L2, and L3, whereas the parasympathetic supply derives from S2, S3, and S4 (7,8). When sympathetic fibers enter the pelvic plexuses, they intermingle with the sacral parasympathetic pathway and postganglionic axons from the sympathetic chain.

While urination and defecation are initiated by reflex actions, they can be inhibited voluntarily by external sphincter (urethral or anal) contraction. Contraction of the external sphincter prevents internal sphincter relaxation, which is initiated by vesical or rectal contractions that are mediated through the evacuation reflexes (1,2). Failure of internal sphincter relaxation leads to reflex vesical or rectal relaxation and waning of the desire to urinate or defecate; this reflex action is mediated through the voluntary inhibition reflex (6,9).

The relation of micturition to the anorectal function was studied by some investigators (10–13). It was reported that anal distension is associated with an increase in vesical pressure (12), whereas bladder distension increased the internal anal sphincter tone (13). However, a study on the response of the external anal sphincter (EAS) to the passage of urine through the

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urethra during micturition could not be found in the literature. We hypothesized that urine passing through the urethra effects EAS contraction to guard against possible flatus or stool leakage during micturition. This hypothesis was investigated in this study.

MATERIALS AND METHODS

Subjects

Twenty-three healthy volunteers (14 men and 9 women; mean age, 38.6 ± 10.8 [SD] years; range, 27–50 years) were enrolled in the study after giving informed consent. They had no anorectal or urinary complaint in the past or at the time of enrollment. Laboratory tests comprised of urinalysis, blood count, and electrocardiography, as well as hepatic and renal function tests, were unremarkable. Abdominal sonography was normal. The study was approved by the Cairo University Faculty of Medicine Review Board and Ethics Committee.

Electromyographic Study

The subjects were instructed to fast for 12 hours before the tests. They were asked to drink water during the preparation for the test. Two monopolar silver–silver chloride surface electrodes (Smith Kline Beckman, Los Angeles, CA) were applied to the EAS. The electrode had a diameter of 0.8 mm and was covered by an insulating vinyl sheath except for the tip. Both electrodes were fixed by electrode gel to the EAS at the anal verge, one electrode at 3 o'clock and the second at 9 o'clock, and were fastened to a metal cannula containing a 2-pin socket. The insulated wire leads were attached to the sockets in the cannula and connected to a Brush Mark 200 rectilinear pen recorder (SmithKline Beckman, Los Angeles, CA). The electric activity of the EAS was recorded from the 2 electrodes.

When the bladder became full, and the desire to micturate was initiated, the subjects were asked to micturate. The electromyogram (EMG) activity of the EAS was recorded during micturition. The subjects were asked to interrupt micturition several times during the EAS EMG recording.

Localization of the Urethral Site That Produces the EAS Response During the Passage of Urine

To determine the part of the urethra that was stimulated by the passage of urine and caused the EAS response, we stimulated different parts of the urethra with a catheter-mounted electrode and recorded the EAS response. A 16F catheter with a stainless steel ring electrode fixed to its distal part was introduced into the urethra so that the ring electrode lay in the urethra at the bladder neck. The ring electrode was bonded to wires that were conducted to the exterior and connected to the input of an amplifier. After the urethra at the bladder neck had been stimulated and the EAS response was recorded, the catheter was moved so that the electrode lay successively in the prostatic, membra-

nous, and penile urethra. At each of these sites, the urethra was stimulated through the ring electrode, and the EMG response was recorded. The electrical stimulation was effected by a train of 5 square wave pulses of 1-ms duration and separated by 1 ms, and the threshold varied from 44 to 62 mA (mean, 51.4 ± 8.4 mA). The latency of the EAS EMG response to urethral stimulation was measured from the start of stimulation to the first deflection of the muscle action potentials complex recorded in the sphincter.

Urethral and EAS Anesthetization

To examine whether the EAS response to urethral stimulation was a direct or reflex action, we performed individual anesthetization of the EAS and the urethra, and the EAS response to urethral stimulation was tested. The urethra at the bladder neck and the prostatic, membranous, and penile urethra were separately anesthetized by cystoscopic injection of 5 mL of 2% lidocaine at each of the aforementioned sites. The response of the EAS to stimulation of the anesthetized urethra using the aforementioned parameters was recorded after 20 minutes of anesthetization and after 3 hours when the anesthetic effect had waned. On another day, the EAS was anesthetized by injecting 3 mL of 2% lidocaine into the sphincter, and the EAS responses to individual stimulation of the urethra at the bladder neck and the prostatic, membranous, and penile urethra were recorded after 20 minutes and after 3 hours of anesthetization. The procedure was repeated on a different day using normal saline instead of lidocaine.

Reproducibility of the results was ensured by repeating the recordings at least twice in the individual subject, and the mean value was calculated. The results were analyzed statistically using analysis of variance (ANOVA) with a *t* test. Significance of difference was set at $P < 0.05$ and was expressed as mean $\pm$ SD.

RESULTS

No adverse side effects were encountered during or after the test performance, and all subjects were evaluated.

The basal EAS EMG activity (motor unit action potentials [MUAPs]) recorded a mean of 84.6 ± 9.4 microvolt (μ V) (range, 77–106 μ V; Figure 1a). During micturition, a significant increase of the EAS EMG activity was registered. The MUAPs recorded a mean of 482.6 ± 48.3 μ V (range, 408–538 μ V; $P < 0.01$; Figure 1b), which was maintained during micturition. On interruption of micturition, the EAS EMG activity returned to basal activity with no significant difference ($P > 0.05$). When micturition was resumed after interruption, the EAS EMG activity showed an increase that was similar to before micturition was interrupted ($P > 0.05$). At the end of micturition, the EAS EMG returned to basal activity ($P > 0.05$). The aforementioned results showed no significant difference between

Figure 1. EMG activity of the external anal sphincter: (a) basal activity and (b) during micturition. Micturition (↑).

men and women or between young and elderly subjects.

Response of the EAS to Urethral Stimulation With Catheter-mounted Electrodes

Urethral stimulation at the bladder neck using the aforementioned parameters caused a significant increase in the EAS MUAPs to a mean of $518.4 \pm 50.6 \mu\text{V}$ (range, 432–553 μV ; $P < 0.01$; Figure 2). This effect did not differ significantly from that induced by urine flow during micturition ($P > 0.05$). The EAS response to urethral stimulation occurred after a mean of $16.3 \pm 1.2 \text{ ms}$ (range, 15–18 ms). This latency was reproducible when the test was repeated in the individual subject. When the electrode was moved to lie in the prostatic, membranous, or penile urethra, electric stimulation of the urethra at these sites with the aforementioned parameters produced no significant effect on the EAS (Figure 3).

Effect of Urethral Stimulation on the Anesthetized EAS and Urethra

The anesthetized EAS did not respond to stimulation of the different sectors of the urethra 20 minutes after anesthetization (Figure 4), but did respond after 3 hours when the anesthetic effect had waned. Similarly, stimulation of the anesthetized urethra did not cause a significant change in the EAS EMG activity 20 minutes after anesthetization. Three hours later, when the anesthetic effect had disappeared, the EAS responded to urethral stimulation like the response before anesthetization with no significant difference ($P > 0.05$). The EAS response to urethral stimulation using normal saline instead of lidocaine was similar, with no significant difference against that without saline.

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

DISCUSSION

Our study may shed some light on EAS status during micturition. Both the EAS and the external urethral sphincter (EUS) have the same nerve supply from the

Figure 2. EMG activity of the external anal sphincter: (a) basal activity and (b) on urethral stimulation at the bladder neck. Stimulation (↑).

pudendal nerve, albeit through different nerve branches: the inferior rectal nerve for the EAS and the perineal nerve for the EUS. For this reason, there may be concomitant EAS relaxation and risk of flatus and fecal leakage when the EUS relaxes during micturition.

This study showed an increase in EAS EMG activity during micturition, indicating contraction of the EAS muscle that was maintained throughout micturition. This action seemingly acts to guard against the leak of flatus or fluid and formed stools during micturition. To define whether the EAS response to micturition was a direct or reflex action, the aforementioned anesthetic test was performed.

Urethro–Anal Reflex

The EAS contraction on urethral stimulation by passage of urine in the urethra or by means of a catheter-mounted electrode postulates a reflex relationship between the 2 actions. This relationship is evidenced by reproducibility and its reflex nature by its disappearance on anesthetization of either the urethra or the EAS, the 2 possible arms of the reflex arc. We call this hitherto unrecognized reflex relationship the urethro–anal reflex. It seems that, on stimulation of the urethral mechanoreceptors, impulses pass along the autonomic nerve fibers from the urethra to the sacral spinal cord, which eventually sends impulses along the pudendal nerve (S2–S4) to the EAS, causing its contraction. Anesthetization of the urethra or the EAS by lidocaine seems to block their innervation such that nerve impulses cannot be transmitted from the urethra to the spinal cord or from the spinal cord to the EAS. Lidocaine blocks the sensory fibers (C and A α -fibers) responsible for pain and reflex activity (14,15).

The urethro–anal reflex is presumably disordered in patients with spinal cord injury when 1 arm or the 2 arms of the reflex are injured. It may be argued that the significant increase of the EAS EMG on urethral stimulation could be a physical stimulation by the passage of urine and that anal EMG and periurethral EMG seem to go together (16). However, such physical stimulation seems to be negated by the above mentioned EAS and urethral anesthetization test, which seems to denote that

Figure 3. EMG activity of the external anal sphincter: (a) basal activity, (b) on stimulation of the prostatic urethra, (c) on stimulation of the membranous urethra, and (d) on stimulation of the penile urethra. Stimulation ($\uparrow$).

the EAS response to urethral stimulation was a reflex rather than a direct action.

Urethral Site That Produces EAS Response to Passage of Urine

The results of this study revealed that EAS stimulation was caused through stimulation of the urethra at the bladder neck, whereas stimulation of the rest of the urethra did not produce an EAS contraction. The cause of the proximal urethra close to the bladder neck being the only part of the urethra that was stimulated by the passage of urine is not known. Does this area contain stretch receptors that respond to urethral stretch by the column of urine? This concept does not seem to be plausible because the urinary stream apparently stretches the whole of the urethra when passing through it. Does the proximal urethra contain chemoreceptors that respond to the chemical nature of the urine? The assumed presence of these receptors in the urethra at the bladder neck seems to allow for EAS contraction simultaneously with EUS relaxation at the start of micturition. The synchronicity of the 2 sphincteric actions presumably guards against any anal leak during micturition, in particular because micturition may normally be associated with mild straining (17) that may jeopardize the EAS competence. Investigators have reported that the bladder neck area is the main sensitive area in the bladder (18). Bladder contractions could be elicited after stimulation of the bladder neck region; the response could not be elicited from other parts of the urethra or bladder wall. The EAS contraction that is maintained throughout the act of micturition would secure the anus against leakage. It may be argued that the EAS is a striated muscle that cannot keep contracting for long periods.

Figure 4. EMG activity of the external anal sphincter 20 minutes after sphincter anesthetization: (a) basal activity, (b) on urethral stimulation at the bladder neck, (c) on urethral stimulation of the prostatic urethra, (d) on stimulation of the membranous urethra, and (e) on stimulation of the penile urethra. Stimulation ($\uparrow$).

However, the EAS can contract continuously for about 50 seconds (19), during which time micturition would have been terminated. Meanwhile, periods of micturition interruption, which commonly occur during micturition, seem to give the EAS periods of relaxation.

Diagnostic Role of the Urethro–Anal Reflex

The urethro–anal reflex may prove to be of diagnostic significance in micturition and/or defecation disorders, in particular those of neurologic origin as in spinal cord injury. Detectable changes in MUAP frequency, amplitude, or conduction velocity or in the latency of the reflex response might indicate a defect in the reflex pathway such as damage to the EUS or EAS or nerve damage from a disease of the spinal cord, spinal nerve roots, or peripheral nerves, or from a central lesion. The reflex may be included in the diagnosis of patients with spinal cord injury. However, further studies are needed before the urethro–anal reflex can be incorporated as an investigative tool in the diagnosis of such conditions.

CONCLUSION

In conclusion, passage of urine through the urethra or urethral stimulation at the vesical neck produced an increase in the EAS EMG, which presumably indicates EAS contraction. This EAS contraction seems to guard against flatus or fecal leakage during micturition. EAS contraction

on urethral stimulation is suggested to be mediated through a urethro–anal reflex. This reflex may prove to be of diagnostic significance in micturition and defecation disorders, provided further studies are carried out.

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