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Original Article

Electromyographic activity of the anterolateral abdominal wall muscles during the vesical filling and evacuation

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Abstract

BACKGROUND: The role of the anterolateral abdominal wall muscles (AAWMs) during the vesical filling and evacuation has not been sufficiently addressed in the literature. We have investigated the hypothesis that the AAWMs exhibit the increased electromyographic (EMG) activity on the vesical distension and contraction which presumably assists vesical evacuation.

METHODS: The effects of the vesical balloon distension on the vesical pressure (VP), vesical neck (VNP) pressures and the AAWMs' EMG activity were studied in 28 healthy volunteers aged 40.7 ± 9.7 years (18 men, 10 women). These effects were tested after the individual anesthetization of the bladder and AAWMs and after saline infiltration.

RESULTS: The VP and the VNP showed a gradual increase upon the incremental vesical balloon distension which started at a distending volume of 120–140 ml. At a mean volume of 364.6 ± 23.8 ml, the VP increased to a mean of 36.6 ± 3.2 cmH_2O, the VNP decreased to 18.4 ± 2.4 cmH_2O, and the AAWMs EMG registered a significant increase. This effect disappeared in the individual bladder and in the AAWMs' anesthetization. However, it did not disappear in the saline administration.

CONCLUSIONS: The AAWMs appear to contract simultaneously with vesical contraction. This action presumably increases the IAP and it assists vesical contraction. The AAWMs contraction on vesical contraction seems to be mediated through a reflex which is called the 'vesico-abdominal wall reflex'. Further studies are required to investigate the role of this reflex in vesical disorders.

KEY WORDS: Oblique muscles, transversus abdominis, rectus abdominis, vesical pressure, electromyography.

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The anterolateral abdominal wall muscles (AAWMs) consist of external and internal oblique (EOM, IOM), transverses abdominis (TAM) and rectus abdominis (RAM) muscles [1]. They consist of striated muscle bundles which contract voluntarily [1]. They work together to perform multiple functions some of which involve the generation of a positive pressure within the abdominal cavity [2]. Activities such as expiration, defecation and micturition may be assisted by the generation of a positive intra-abdominal pressure (IAP) [3]. In addition, parturition, coughing, and vomiting are usually aided by this positive pressure. Under normal resting conditions, the AAWMs provide the support for the abdominal viscera and they retain the normal abdominal contour [1,2]. The congenital absence of these muscles as in prune belly syndrome [4], would lead to lack of support of the abdominal viscera. When the

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IAP is increased, AAWMs' contraction plays an important role in the maintenance of the abdominal wall tone [1,2]. The AAWMs contract voluntarily and reflexively [5] on the increase of coughing or, straining-produced IAP. Urination is initiated when urine distends the urinary bladder to a certain volume. The stretch receptors in the vesical wall are stimulated and the micturition reflex is evoked with a vesical contraction and a sphincteric relaxation [6]. The micturition reflex is normally under voluntary control and it is organized in the pontine micturition center. The pelvic floor muscles, including the levator ani and puborectalis muscles play a significant role in urination [7-9]. However, the function of the AAWMs in urination is poorly addressed in the literature. We hypothesized that the AAWMs exhibit an increased electromyographic activity on vesical distention and contraction. Thereby, presumably, they assist vesical evacuation. This hypothesis was investigated in the current study.

Methods

Subjects

Twenty eight healthy volunteers (18 men and 10 women with the mean age of 40.7 ± 9.7 years who ranged from 29 to 52 years) after having been fully informed about the nature of the study were asked to submit their written consents to participate in the study. The subject's physical examinations, including neurogenic assessment, showed normal findings. The laboratory work-up comprising blood count, renal and hepatic function tests and electrocardiography had unremarkable results. This study has been approved by the Cairo University Faculty of Medicine Review Board and Ethics Committee.

Methodology

Before the tests were started, the subjects were asked to empty their bladder. The urinary bladder was distended by a 10 F balloon-ended catheter (London Rubber Industries Ltd, London, UK). A metallic clip was applied to the tip of the catheter for fluoroscopic control. The catheter was introduced per urethram into the urinary bladder. While the vesical pressure (VP) and the vesical neck pressure (VNP) and the AAWMs' EMG activity were registered, the balloon was filled with normal saline in increments of 20 ml. The VP was measured by a 6 F manometric catheter (London Rubber Industries Ltd, London, UK) which was introduced through urethral tube into the urinary bladder. The catheter was connected to a strain gauge pressure transducer (Statham 230B, Oxnard, CA) and to a chart recorder via amplifiers (Hewlett Packard 7798A, Waltham, MA, USA). The VNP was recorded by an identical catheter and it was introduced into the urinary bladder and was connected to a pressure transducer. The pressure read a mean of 7.9 ± 1.2 cm H_2O (ranged 6-10). The catheter was then pulled backward until it reached the bladder neck where the pressure registered a mean of 66.8 ± 8.4 cmH_2O (ranged 54-78).

EMG activity of the AAWMs

The EMG activity of each of the EAS, IAS, TAM, and the RAM was recorded by means of a concentric EMG needle electrode (type 13L49, Disa, Copenhagen, Denmark) of 45 mm in length and 0.65 mm in diameter. One needle electrode was introduced into each of the EOM, IOM, TAM and RAM. With the patient lying supine and under no anesthesia, the needle electrode for the RAM was inserted into the muscle 3-4 cm above the umbilicus and 1.5-2.5 cm away from the midline. For the EOM, IOM and TAM, the needle electrode was inserted into each muscle 2-3 cm lateral to the lateral edge of the RAM (linea semilunaris), 3-4 cm above the umbilicus and 3-4 cm apart on the horizontal level. It was introduced through the abdominal wall skin and the musculature to a depth of 0.75-1 cm for the EOM, 1-1.5 cm for the IOM, and 2-2.5 cm for the TAM. As the abdominal wall muscles were striated, they produced no electric waves at rest. Only when the subjects strained or coughed, the muscles contracted and exhibited electric activities. The correct position of the needle electrode in the corresponding muscle was indicated by the different electric waves which were discharged from each muscle on each coughing or strain-

ing; in terms of the frequency and the amplitude, the electric waves differed from one muscle to another.

The recorded potentials were amplified and displayed on a standard EMG apparatus (type MES, Medelec, Woking, UK). The amplifier (type AA6 MKUM, Medelec) was set with a low-frequency filter at 18 Hz and a high-frequency filter at 3200 Hz. The films of the potentials were taken on a light sensitive paper (Linagraph, type 1895, Kodak, London, UK) from which the measurements of the motor unit action potentials (MUAPs) were made. The EMG signals were also stored on an FM tape recorder (type 7758A, Hewlett-Packard, Waltham, Ma, USA) for further analysis. To define whether the AAWMs' response to vesical distension was a direct or reflexive action, the test was repeated after the individual anesthetization of the urinary bladder and the AAWMs. Vesical anesthetization was induced by the vesical instillation of 10 ml of 2% lidocaine added to 50 ml of normal saline. The response of the AAWMs to the vesical balloon distension with the aforementioned volumes was tested after 20 minutes of vesical anesthetization and 3 hours after the anesthetic effect had waned. On another day, the AAWMs were individually anesthetized by injecting 2 ml of lidocaine into the muscle at the site of the electrode; the AAWMs' response to the vesical balloon distension was registered after 20 minutes and it was recorded again after 3 hours. The above tests were repeated using normal saline instead of lidocaine. The reproducibility of the results was ensured by repeating at least twice the aforementioned recordings in the individual subjects and the mean value was calculated. The results were analyzed statistically using the analysis of the variance and the values were given as the mean ± SD. Significance was ascribed to $P<0.05$.

Results

No adverse side effects were encountered during or after the performance of the study and the tests were completed in all the volunteers. The basal VP recorded a mean of 8.5 ± 1.2 cmH_2O and the VNP of 62.4 ± 7.6 cmH_2O (table1). The EMG of the EOM, IOM, TAM, and the RAM recorded no basal activity as these muscles are striated. Vesical balloon distension in increments of 20 ml of normal saline and up to 120-140 ml did not have significant VP or VNP changes ($P>0.05$) or the AAWMs EMG response (figure 1, table 1). Vesical distension above this level in increments of 20 ml of normal saline affected the gradual increase of the VP and the VNP (table 1). At a mean vesical balloon distension of 364.6 ± 23.8 ml (range of 340-400), the VP recorded a mean of 36.6 ± 3.2 cmH_2O and the VNP of 18.4 ± 2.4 cmH_2O and the AAWMs' EMG activity registered a significant increase. These pressures herald the onset of the voiding phase. The EOM recorded a mean amplitude of 174.6 ± 22.3 (ranged 132-216), the IOM of 153.8 ± 23.5 (ranged 126-196), the TAM of 144.2 ± 21.8 (ranged 118-184), and the RAM of 128.6 ± 19.4 (ranged 106-164) μV (figure 2).

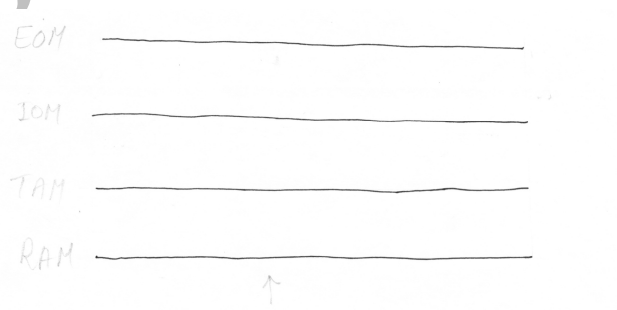

Figure 1. The anterolateral abdominal wall muscles on the vesical balloon distension with 100 ml of normal saline which indicates no EMG activity.
↑ = distension
EOM = External oblique muscle IOM = Internal oblique muscle
TAM = Transverse abdominal muscle RAM = Rectus abdominis muscle

Response of the AAWMs to anesthetization

When the urinary bladder was anesthetized and then distended with the aforementioned volumes of normal saline, the AAWMs did not exhibit EMG activity at any of these volumes after 20 minutes of anesthetization. But, it did

Table 1. Vesical and vesical neck pressures on vesical balloon distension in increments of 20 ml of normal saline+.

	Pressure (cmH_2O)			
Vesical distension (ml)	**Vesical neck**		**Vesical**	
	Mean	**Range**	**Mean**	**Range**
0 (basal)	62.4±7.6	48-72	8.5±1.2	7-10
20	62.3±7.5	48-70	8.3±1.2	7-10
60	63.2±7.6	49-72	8.5±1.2	7-10
100	62.8±7.2	48-71	8.4±1.2	7-10
120	63.2±7.4	49-73	8.5±1.2	7-10
140	62.9±7.5	50-74	8.3±1.2	7-10
160	74.3±9.1	57-84	10.4±1.6	8-12
180	88.6±10.3	71-99	14.6±2.2	11-16
220	96.8±10.8	86-108	20.3±3.3	17-24
280	98.3±9.3	88-110	26.6±3.9	21-31
340	18.4±2.4	14-21	36.6±3.2	32-39

+ The values were given as the mean ± SD.

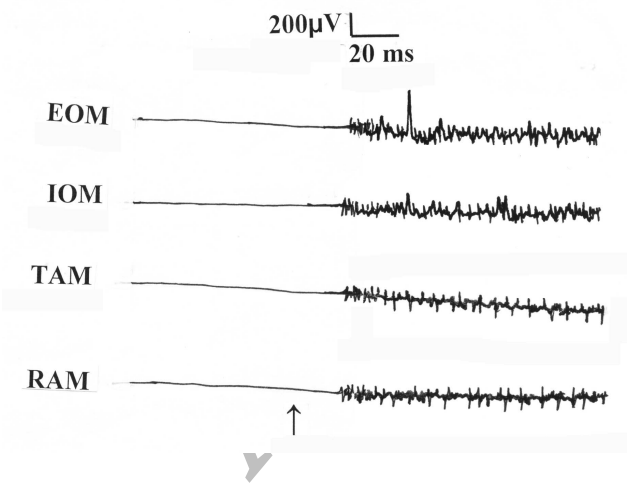

Figure 2. The anterolateral abdominal wall muscles on vesical balloon distension with 360 ml of normal saline which indicates a significant increase of the EMG activity.
↑ = distension
EOM = External oblique muscle
IOM = Internal oblique muscle
TAM = Transverse abdominal muscle
RAM = Rectus abdominis muscle

respond after 3 hours when the anesthetic effect had waned. Likewise, the anesthetized AAWMs did not respond to the vesical balloon distension with the aforementioned volumes 20 minutes after the anesthetization but, the response occurred 3 hours later. Normal saline administration instead of lidocaine in the above mentioned tests did not affect the AAWMs' response to the vesical balloon distension. When the tests were repeated in the same subjects, the aforementioned results were reproducible with no significant difference (P>0.05).

Discussion

The AAWMs which partially cover the abdominal cavity, contribute to the maintenance of IAP which is constant under normal physiologic conditions [1,2]. They respond to the increase of the IAP by reflexive contraction which is an action mediated through the 'straining-abdominal wall reflex' (SAWR) [5]. Coughing and straining evoke this reflex with a resulting AAWMs' contraction. The AAWMs contract not only reflexively but also voluntar-

ily as they are composed of striated muscle fibers. Like the other striated muscles, they have no resting electric activity; the muscle fibers exhibit no tone at rest. A recent study has shown that in awaked rats, the abdominal muscles were activated during the spontaneous voiding and during the voiding induced by saline infusion [10]. In the meanwhile another study demonstrated that bladder electrodes do not record remote muscle activities from the inferior rectus abdominis muscle [11]. The authors speculated that any increase of the detrusor electrical activity with abdominal contraction may be the result of the increase of the IAP from the abdominal contraction; however, these speculations were not proved.

The current study has demonstrated that the AAWMs' EMG did not respond to small volumes of vesical filling. At a certain volume of vesical filling and when the VP had reached its maximum with a significantly decreased VNP, the AAWMs' EMG exhibited a significant increase which presumably denotes contraction of these muscles. Thus, the AAWMs presumably contract at the time of the vesical evacuation as it is evidenced by the vesical pressure elevation, indicating vesical contraction and VNP decrease denoting VN relaxation. It seems that the increased IAP which is induced by the AAWMs' contraction assists vesical evacuation. A previous study has demonstrated that straining which is the result of the IAP increase, triggers the 'straining levator reflex' which affects the levator ani muscle contraction [12]. The latter shares the mechanism of vesical neck opening and urination. The IAP increase which results from the AAWMs' contraction, is apparently physiologic and it is ought to be differentiated from the pathologic IAP increase and straining which occur in the vesical neck obstruction. The latter comprises the failure of the vesical neck to open on vesical contraction due to the functional or mechanical causes [13]. It should be discussed whether the AAWMs' response to vesical contraction is a direct or a reflexive action. The direct action can apparently be excluded because there is not any direct connection between the AAWMs and the urinary bladder. Hence, the relation between the two actions seems to be a reflexive one.

Vesico-abdominal wall reflex

The AAWMs' contraction on vesical contraction postulates a reflexive relationship between the 2 actions. The constancy of this relationship was evidenced by its reproducibility and its reflexive nature by the absence of the AAWMs' response on the anesthetization of either of the 2 assumed arms of the reflexive arc: the urinary bladder and the AAWMs. This reflexive relationship is called the 'vesico-abdominal wall reflex' (VAWR). The reflex was not evoked with small-volume bladder distension. However, it was evoked when the vesical distension was great enough to initiate the vesical contraction. It seems that the vesical distension, upon reaching a certain volume, stimulates the vesical wall stretch receptors which send impulses along the pelvic nerve to the spinal cord and stimulate the motor neurons of the AAWMs. Motor impulses are presumably transmitted along the nerves that supply the AAWMs (T1-T6). The argument in this regard is that lidocaine which blocks the muscle activity is irrelevant in this process because lidocaine blocks only the sensory C and A α-fibers which are responsible for pain and reflexive activities [14,15]. We don't know whether the VAWR has a diagnostic significance; this point needs to be investigated.

In conclusion, the AAWMs appear to contract simultaneously with the vesical contraction. This action presumably increases the IAP and it assists the vesical evacuation. The AAWMs' contraction on vesical contraction seems to be mediated through a reflex which is called the 'vesico-abdominal wall reflex'. Further studies are required to investigate the role of this reflex in vesical disorders.

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