Effect of straining on the muscles of the anterior abdominal wall. identification of the 'straining-adominal wall reflex'

Ahmed Shafik 1, Ismail A. Shafik 2, Olfat El Sibai 3, and Ali A. Shafik 4

<sup>1</sup> Department of Surgery and Experimental Research, Faculty of Medicine, Cairo University, Cairo, <sup>2</sup> Department of Surgery and Experimental Research, Faculty of Medicine, Cairo University, Cairo, <sup>3</sup> Department of Surgery, Faculty of Medicine, Menoufia University, Shebin El-Kom, <sup>4</sup> Department of Surgery and Experimental Research, Faculty of Medicine, Cairo University, Cairo Egypt

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#### 1. ABSTRACT

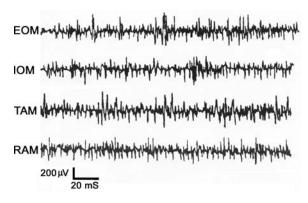
The external and internal oblique, transverse abdominis and the rectus abdominis muscles constitute the anterolateral abdominal wall muscles. They are striated and contract voluntarily. We investigated the hypothesis that contraction of these muscles by coughing or straining, can also occur as a reflex. Effect of straining on muscles was tested in 19 healthy volunteers. The intra-abdominal pressure was measured by a manometric catheter introduced into the rectum. The response of the muscles to straining was recorded by a needle electrode inserted into each of the muscles. Similar to voluntrary contractions, sudden and sustained straining produced increase in the rectal pressure and the motor unit action potentials of each of the muscles which was abolished by anesthesia. These findings suggest presence of a 'straining-abdominal wall reflex'.

#### 2. INTRODUCTION

The anterolateral abdominal wall muscles (AAWMs) consist of the external and internal oblique (EOM, IOM), transverse abdominis (TAM) and rectus abdominis (RAM) muscles (1).

They consist of striated muscle bundles which contract voluntarily (1). They act 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). Also parturition, coughing, and vomiting are usually aided by such a positive pressure.

Under normal resting conditions, the AAWMs provide support to the abdominal viscera and retain the



**Figure 1.** EMG activity of the external oblique (EOM), internal oblique (IOM), transverse abdominal (TAM) and rectus abdominis (RAM) muscles on voluntary contraction of these muscles without straining.

normal abdominal contour (1,2). Congenital absence of these muscles as in 'prune belly syndrome (4)' would lead to lack of support of the abdominal viscera. When the IAP is increased, AAWMs' contraction plays an important role in the maintenance of the abdominal wall tone (1,2)

The AAWMs contract voluntarily as well as involuntarily on the increase of the IAP as produced by coughing or straining. We hypothesized that AAWMs' contraction on increased IAP is reflex. This hypothesis was investigated in the current study.

#### 3. MATERIAL and METHODS

### 3.1. Subject

The study comprised 19 healthy volunteers (10 men, 9 women; mean age 36.6±10.7 SD years, range 21-48). After having been fully informed about the nature of the study, they gave an informed consent to participate in the tests to be done and to their role in the study.

The results of physical examination, including neurologic assessment, were normal. Laboratory work including blood count, renal and hepatic function tests as well as electromyography, had unremarkable results. The Cairo University Faculty of Medicine Review Board and Ethics Committee approved the study.

## 3.2. Methods

At first the subjects were asked to voluntarily contract the AAWMs without straining. Then the effect of straining on these muscles was tested. Straining was gauged by measuring the IAP. The intrarectal pressure was considered as representative of the IAP. The subjects fasted for 8 hours prior to the tests and the bowel was emptied by defecation or saline enema. The rectal pressure was measured by a 10F manometric catheter (London Rubber Industries Ltd, London, UK), introduced per anum 8-10 cm into the rectum. The catheter was connected to a strain gauge pressure transducert (Statham 230B, Oxnard, CA) and via amplifiers to a chart recorder (Hewlett Packard 7798A, Waltham, Ma, USA).

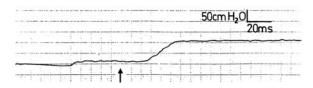
The EMG activity of each of the EOM, IOM, TAM and RAM was recorded by means of a concentric needle EMG electrode (Type 13L49, Disa, Copenhagen, Denmark), 45 mm in length and 0.65 mm in diameter. A needle electrode was introduced into each of the EOM, IOM, TAM and RAM. With the patient lying supine and 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, a needle electrode was inserted into each muscle 2-3 cm lateral to the lateral edge of the RAM (linea semilimaris), 3-4 cm above the umbilicus and 3-4 cm apart on the horizontal level. The needle electrode was introduced through the abdominal wall skin and the muscle for 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 are striated, no electric waves were recorded from them at rest. Only when the subjects strained or coughed, the muscles contracted and exhibited electric activity. The correct position of the needle electrode in the corresponding muscle was indicated by the different electric waves discharged from each muscle on coughing or straining; with regard to frequency and amplitude, each muscle discharged electric waves different from the others.

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. Films of the potentials were taken on light sensitive paper (Linagraph, type 1895, Kodak, London, UK) from which measurements of the motor unit action potentials (MUAPs) duration 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.

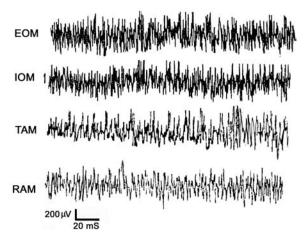
The correct position of the needle electrode was monitored by the burst of activity heard on the loudspeaker and visualized on the oscilloscopic screen of the electromyograph. Prior to performing the tests, we had verified the normality of the EMG activity of the AAWMs in all volunteers by stimulation of these muscles with a needle electrode and by recording the action potentials with the already inserted needle electrode. All the subjects had normal AAWMs EMG activity.

The subjects were asked to voluntarily contract the AAWMs but without straining. This was gauged by observing the intrarectal pressure considering that AAWMs' contraction without straining should not be associated with an increase of the intrarectal pressure.

The subjects were then asked to strain by either coughing or performing a Valsalva's maneuver, and the IAP as well as the EMG response of the EOM, IOM, TAM and RAM were recorded. Coughing represented sudden momentary straining and Valsalva's maneuver simulated slow sustained straining that may be experienced with defecation or urination. When we finished recording the IAP and AAWMs EMG response to straining on one side,



**Figure 2.** Intrarectal pressure response to suddenstraining (coughing). ↑ = coughing



**Figure 3.** EMG response of the external oblique (EOM), internal oblique (IOM), transverse abdominis (TAM) and rectus abdominis (RAM) muscles to sudden straining (coughing).



**Figure 4.** EMG response of the external oblique muscle to slow sustained straining induced by Valsava's maneuver at various rectal pressures. Rectal pressures (cmH $_2$ O): a. basal, b. 86, c. 166.



**Figure 5.** EMG response of the internal oblique muscle to slow sustained straining induced by Valsava's maneuver at various rectal pressures. Rectal pressures (cm $H_2O$ ): a.basal, b. 79, c. 172.

we transferred the needle electrode to the muscles on the contralateral side to a similar point and depth as mentioned above and the response to straining was registered.

## 3.3. Abdominal muscles anesthetization

To investigate whether the response of the EOM, IOM, TAM and RAM was direct or reflex, individual

anesthetization of these muscles as well as of the rectum was performed in all of the subjects. The individual muscle was infiltrated around the inserted electrode with 2 ml of 2% lidocaine added to 2 ml of normal saline. We kept the injected dose small enough so as not to affect the voluntary activity of the muscle during the test. The muscle response to straining was recorded after 20 minutes and 3 hours later when the anesthetic effect had waned. On another day, the test was repeated using normal saline instead of lidocaine. On a separate day, the rectum was anethetized and the aforementioned muscles' response to straining was tested after 20 minutes and after 3 hours. The anesthetic solution instilled into the rectum consisted of 20 ml of 2% lidocaine added to 20 ml of normal saline. The test was repeated using normal saline instead of lidocaine.

To ensure reproducibility of the results, the aforementioned recordings were repeated at least twice in the individual subject, and the mean value was calculated.

#### 3.4. Statistical significance

The results were analyzed statistically using the Student's t test, and the values were given as the mean  $\pm$  standard deviation (SD). Data with p<0.05 was considered significant.

#### 4. RESULTS

The tests were completed in all the subjects with no adverse side effects. The examined muscles did not exhibit electric activity at rest; no electric waves were recorded.

When the subjects were asked to contract the AAWMs without straining, the rectal pressure recorded a mean of 8.2±1.2 cm H<sub>2</sub>O (range 7-11) which did not differ significantly from the resting pressure (p>0.05). Meanwhile, the EMG activity recorded a mean of 306.4±46.7 µV for the EOM, 318.4±48.3 µV for the IOM, 258.4±40.7 µV for the TAM, and 384.7±63.7 µV for the RAM (table 1, Figure 1). On sudden straining by coughing, the rectal pressure exhibited a significant increase from the mean basal value of 7.8±1.1 cmH<sub>2</sub>O (range 6 - 10) to a mean of  $98.3 \pm 21.7$  cmH<sub>2</sub>O (range 69 -126, p<0.0001, Figure 2). Simultaneously with the rectal pressure increase, the EMG activity of the EOM, IOM, TAM, and RAM showed a significant increase to a mean of  $564.6\pm84.2$ ,  $608.4\pm79.6$ ,  $526.4\pm68.4$ , and  $664.2\pm94.8$ μV, respectively (Figure 3, table 1).

The slow sustained straining induced by Valsalva's maneuver raised the rectal pressure and EMG activity of the EOM, IOM, TAM, and RAM (figs. 4-7). The more the rectal pressure was increased by straining, the higher rose the EMG activity of the aforementioned muscles (Figures 4-7). The muscle contraction remained for a mean period of 50.6±7.6 s (range 42 – 57) and was followed by spontaneous relaxation which occurred while the straining was continuous; this is because the muscles are striated and thus easily fatigable. However, after a mean period of 18.6±2.1 s (mean 15-20), the response of the aforementioned muscles to straining was regained.

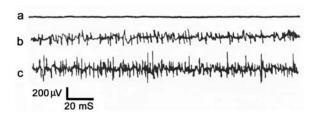
**Table 1.** EMG activity of the external oblique (EOM), internal oblique (IOM), transverses abdominis (TAM) and rectus abdominis (RAM) muscles at rest (basal) and upon sudden straining (coughing) and on voluntary contraction of these muscles without straining

Potentials (µV)								
	EOM		IOM		TAM			
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Basal	0	0	0	0	0	0	0	0
Coughing	564.6±84.2	418-674	608.4±79.6	486-712	526.4±68.4	412-628	664.2±94.8	528-821
Voluntary contraction (without coughing)	306.4±46.7	226-381	318.4±48.3	212-406	258.4±40.7	191-326	384.7±63.7	311-508

Values are mean  $\pm$  standard deviation



**Figure 6.** EMG response of the transverse abdominis muscle to slow sustained straining induced by Valsava's maneuver at various rectal pressures. Rectal pressures (cmH<sub>2</sub>O): a. basal, b. 68, c. 188.



**Figure 7.** EMG response of the rectus abdominis muscle to slow sustained straining induced by Valsava's maneuver at various rectal pressures. Rectal pressures (cmH<sub>2</sub>O): a.basal, b. 78, c. 180.

Upon cessation of straining, the rectal pressure and the EMG activity of the EOM, IOM, TAM, and RAM returned to the basal values.

# 4.1. Response of the abdominal wall muscles to anesthetization of rectum and abdominal wall muscles

When the rectum had been anesthetized and the subject was asked to strain (sudden or slow sustained), the AAWMs did not respond 20 minutes from anesthetization, but did 3 hours later after the anesthetic effect had waned. Similarly, the AAWMs did not respond to straining 20 minutes after they had been anesthetized, but responded after 3 hours. Saline administration instead of lidocaine did not affect the AAWMs' response to straining.

The aforementioned results were reproducible with no significant difference when the tests were repeated in the individual subject.

The AAWMs, consisting of striated muscle fibers, contract voluntarily (1,2). Like other striated muscles, they have no resting electric activity (5); the muscle fibers at rest exhibit no resting tone. The current study has shown that voluntary contraction without straining, as evidenced by the insignificant change of the

rectal pressure, had significantly increased the EMG activity of the AAWMs. Meanwhile, the AAWMs contracted on increase of the IAP from coughing or straining.

# 4.2. Involuntary contraction of the AAWMs

Although AAWMs' contraction could be effected voluntarily, yet their contraction on straining or coughing appears to be effected involuntarily. The current study has shown that coughing or straining were associated with an increase of the IAP, as was evident from the elevated intrarectal pressure. The increased IAP was synchronously associated with increased EMG activity of the AAWMs, supposedly denoting contraction of these muscles. Thus, the increase of the IAP is presumably associated with involuntary AAWMs' contraction. This is different from the voluntary contraction of the AAWMs which was not associated with straining or increase of the IAP. It appears that AAWMs' contraction on coughing or straining is a reflex reaction which needs to be discussed.

# 4.3. The straining-abdominal wall reflex

AAWMs' contraction on straining postulates a hitherto unrecognized reflex relationship between the 2 actions. The constancy of this relationship was assured by reproducibility. Meanwhile, the reflex nature of this relationship is evidenced by the absence of the AAWMs' response upon anesthetization of the assumed two arms of the reflex arc: the rectum and the AAWMs. We call this reflex relationship the 'straining-abdominal wall reflex' (SAWR). It appears that straining with a resulting rectal pressure elevation stimulates the rectal mechanoreceptors in the rectal wall 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 supplying the AAWMs (T1-6). The increasing contraction of the AAWMs upon increase of the rectal pressure seems to be due to augmented stimulation of the rectal mechanoreceptors by the progressively elevated rectal pressure upon incremented increase of straining. The disappearance of AAWMs contraction on prolonged straining appears to be attributable to muscle fatigue because the AAWMs are striated and easily fatigable; however, the response of the muscles to straining was regained after a short period. The AAWMs did not respond to straining after the individual lidocaine block of the 2 arms of the presumed reflex arc: rectum and AAWMs. The possible argument that lidocaine may block muscle activity can be ruled out because lidocaine does not block except the sensory fibers (C and Aα-fibers) which are responsible for pain and reflex activity (6,7).

# 4.4. Role of the straining-abdominal wall reflex in abdominal viscera support

The AAWMs act to support the abdominal viscera, particularly under stress conditions such as straining and coughing (1,2). A study on the mechanism of action of the AAWMs during stress conditions could not be found in the literature and needs to be clarified. The abdominal cavity with its visceral content is covered anterolaterally by the AAWMs. Both the abdominal cavity and the covering AAWMs are directly exposed to the effect of the IAP. Under normal physiological conditions, these muscles act to support the abdominal viscera (1,2). When however, the IAP increases, the AAWMs appear to contract reflexly in order to support the anterior abdominal wall against the high IAP. Thus on straining as occurs on coughing or during defecation or urination, the SAWR seems to be evoked leading to contraction of the AAWMs, thus lending support to the abdominal viscera and guards the abdominal wall against the increased IAP. The SAWR may have a role in the diagnosis of the neurogenic disorders of the AAWMs. A change in the amplitude of the evoked response may indicate a defective reflex pathway.

-In conclusion, AAWMs' contraction on straining postulates a reflex relaltionship between the 2 actions which we call 'straining-abdominal wall reflex'. This reflex, which effects AAWMs' contraction, supports the anterior abdominal wall against increased IAP induced by straining and the tendency of the AAWMs to subluxate. These results warrant further study of the role of a deranged reflex in the genesis of functional disorders of the AAWMs.

#### 5. ACKNOWLEDGMENT

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**Send correspondence to:** Ahmed Shafik, MD, PhD, 2 Talaat Harb Street, Cairo 11121, Egypt, Tel/Fax: +20-2-749-8851, E-mail: shafik@ahmedshafik.com

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