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ORIGINAL PAPER

Ahmed Shafik · Olfat El-Sibai · Ali A. Shafik
Ismail Ahmed

The electrovesicogram in the overactive bladder: role in determining pathogenesis and diagnostic significance

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Abstract We investigated the hypothesis that the abnormal contractility of the smooth musculature of the overactive bladder (OAB) may be due to derangement of its electrical activity. Percutaneous electrovesicography was performed in 22 patients (mean age 46.3 years, 12 men, ten women) with OAB and 14 healthy volunteers (mean age 45.6 years, eight men, six women). Recording was performed with the bladder full and empty. Three electrodes were applied suprapubically and one reference electrode was applied to a lower limb. Reproducible regular triphasic slow waves (SWs) were recorded in the volunteers. The pattern of the full and empty bladder were similar except for the higher amplitude of the waves in the former ($P < 0.05$). The OAB patients showed a "dysrhythmic" pattern with irregular frequency, amplitude and conduction velocity in both the empty and full bladders. We obtained tachyrhythmic, bradyrhythmic and arrhythmic areas in the same recording. The OAB exhibited a "dysrhythmic" electrical pattern with areas of different electrical activity in the same recording. The tachyrhythmic, bradyrhythmic and arrhythmic areas are suggested to explain the abnormal vesical contractions and clinical manifestations of OAB. Further studies are required to investigate the cause of the dysrhythmic pattern and the electrovesicogram is suggested as an investigative tool in OAB diagnosis.

A. Shafik (✉) · A. A. Shafik · I. Ahmed
Department of Surgery and Experimental Research,
Faculty of Medicine, Cairo University, Cairo, Egypt
E-mail: shafik@ahmed-shafik.org
Tel.: +20-2-7498851
Fax: +20-2-7498851

O. El-Sibai
Department of Surgery, Faculty of Medicine,
Menoufia University, Shebin El-Kom, Egypt

A. Shafik
2 Talaat Harb Street,
Cairo 11121, Egypt

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Introduction

Overactive bladder (OAB) is a common disorder that affects the lives of many people worldwide. It is a symptom syndrome suggestive of lower urinary tract dysfunction and is characterized by urgency, with or without urge incontinence [1]. It is usually associated with frequency and nocturia with no proven infection or other obvious pathology [1].

The neurologic or myogenic activity of the urinary bladder is affected in OAB by a variety of conditions including neurologic illness or injury, bladder outlet obstruction and urethral weakness [2]; however, there is an idiopathic type of overactive bladder which so far has an unknown etiology. Denervation is commonly found in biopsy specimens of patients with OAB, suggesting that muscle abnormalities may be involved in the cause of the condition [3]. Investigators [4, 5] proposed that a change in the smooth muscle properties seems to be a prerequisite for OAB, however, it has been shown that arterial blood flow and tissue oxygen levels decrease during bladder muscle contraction in animal models, suggesting that denervation may be caused by ischemic damage [6, 7]. Furthermore, changes in neuroanatomic pathways in various disease and injury conditions may lead to OAB. Suprapontine areas exert an inhibitory influence on the pontine micturition center; damage of these areas by stroke, or Alzheimer's or Parkinson's diseases can reduce this inhibitory action [8].

Electrical activity in the form of slow waves (SWs) and action potentials (APs) has been recorded from the urinary bladder [9, 10, 11, 12, 13]. It is claimed that these waves in the gut are generated by the interstitial cells of Cajal (ICC) [14, 15, 16, 17, 18] and are responsible for the contractile activity of the smooth musculature

[19, 20, 21]. A disorder in the electrical activity presumably leads to a derangement of the contractility of the organ [22, 23]. Recent studies have demonstrated the presence of ICC in the urinary bladder [24].

We hypothesize that the abnormal contractility of the smooth musculature in OAB may be due to a derangement of its electrical activity. This was investigated in the current study.

Materials and methods

Subjects

The study comprised 36 subjects who gave informed consent before being enrolled. Twenty two had OAB and 14 were healthy volunteers who acted as controls. Of the 22 OAB patients, 12 were men and ten women with a mean age of 46.3 ± 11.3(SD) years (range 30–67). The causes of OAB in the patients are listed in Table 1. The diagnosis was based on urodynamic examination including uroflowmetry and pressure flow studies. The mean age of the healthy volunteers was 45.6 ± 10.8 years (range 28–64); they comprised eight men and six women who had no urinary manifestations in the past or at the time of enrollment. Urinalysis and sonography of the genitourinary organs were normal. Physical examination, including neurologic assessment, was also normal. All of the subjects were informed about the tests to be done and their role in the study. The Review Board and Ethics Committee of the Cairo University Faculty of Medicine approved the study.

Methods

Transcutaneous electrovesicography

This technique has been described previously [11, 12, 13]. In brief, the subjects were instructed to abstain from urination for at least 4 h prior to testing and to drink fluids excessively during this period. The purpose was to perform the test while the bladder was full. After EMG recording of the full bladder, the subject was requested to micturate and evacuate the bladder. The test was then repeated with the bladder empty.

The skin at the electrode site was prepared by shaving. Beckman-type silver-silver chloride electrodes (Smith Kline-Beckman, Los Angeles, Calif) were used. The subject lay supine and was uncovered from the subcostal margin to the mid thigh. Figure 1 shows the position of the electrodes; one was placed four fingers above and two fingers laterally to each pubic tubercle. To define this location we followed the adductor longus tendon to the first bony prominence, i.e. the pubic tubercle. A third electrode was applied two fingers above the symphysis pubis and a reference electrode was applied to one of the lower limbs. A strain gauge respiratory transducer was attached to the thoracic wall to remove respiration artifacts from the signal.

The electrical activity was recorded between the two electrodes situated four fingers above the pubic tubercle. The left electrode represented the positive pole and the right electrode the negative pole. The electrode above the symphysis pubis was used to generate other leads to determine the maximum deflection of the waves. The electrode placed on one of the lower limbs was for earthing.

With the subject relaxing the abdominal wall, the signals were recorded on paper (Van Gogh Eg-86) and stored on magnetic tape (Recall Store 14). High and low pass filters (6 dB/octave) were set at 0.01 and 0.5 Hz, respectively. At least two 20-min recording sessions were performed for each subject.

The results were analyzed using the Student's *t*-test, and values were given as the mean ± SD. Significance was set at $P < 0.05$.

Table 1 Causes of overactive bladder in 22 patients

Disease	No. patients
Cerebral palsy	5
Spinal canal stenosis	6
Cerebellar ataxia	3
Parkinson's disease	3
Idiopathic	5

Results

All of the subjects could be evaluated and no adverse side effects were encountered during or after testing.

Electrovesicography in normal volunteers

SWs were recorded from the full and empty urinary bladder. The waves were triphasic with a small positive, a large negative and another small positive deflection (Fig. 2). They had the same frequency, amplitude and velocity of conduction from all three test electrodes on each individual subject (Fig. 2). These variables were reproducible with no significant differences when the test was repeated in the same subject. The frequency, amplitude and conduction velocity of the full and empty bladder are listed in Table 2. There was no significant difference in the waves' frequency and conduction velocity between the empty and full bladder (> > Table 2), whereas the amplitude was significantly higher in the full than in the empty bladder ($P < 0.05$, Table 2, Fig. 3).

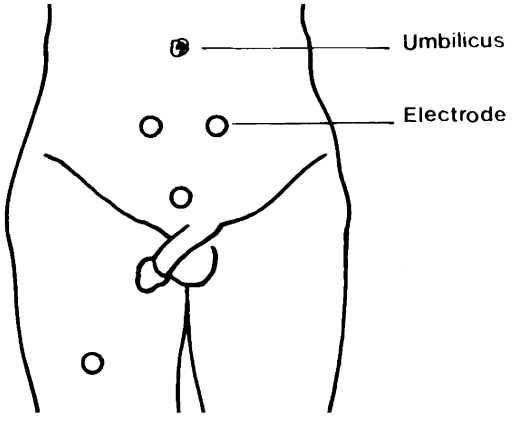

Fig. 1 The position of the electrodes

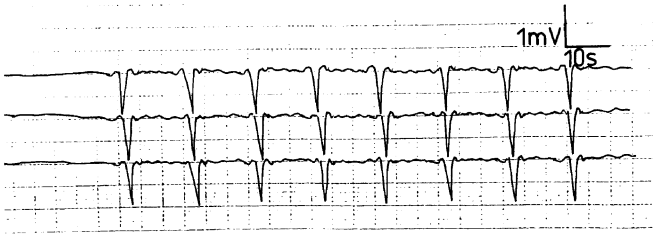

Fig. 2 Electrovesicogram of a normal subject with a full urinary bladder

Table 2 Frequency, amplitude and velocity of conduction of the slow waves recorded from the full and empty urinary bladder of 14 healthy volunteers. Values are means ± SD, $^{ns}P > 0.05$, * $P < 0.05$, P values are for a comparison of the empty with the full bladder

Slow waves						
Urinary bladder						
	Frequency (cpm)		Amplitude (mV)		Velocity (cm/s)	
	Mean	Range	Mean	Range	Mean	Range
Full	4.7 ± 1.1	3–6	1.2 ± 0.2	0.9–1.6	4.1 ± 1.1	3.6–5.4
Empty	4.2 ± 0.9^{ns}	3–6	0.71 ± 0.1*	0.6–0.9	3.9 ± 0.9^{ns}	3.2–5.1

Electromyography of the OAB

The SWs showed irregular frequency, amplitude and conduction velocity in the individual subjects (Fig. 4). These variables differed between the three test electrodes on the same subject (Fig. 4) and were not constant while being recorded. In the same registration session, we obtained areas of bradyrhythmia, tachyrhythmia and vesico-arrhythmia. This picture of "dysrhythmia" was recorded in the full and empty urinary bladder (Fig. 5). However, although the empty and full bladder both presented with the "dysrhythmic" electrovesicographic pattern, their waves' variables were different.

Discussion

The contractile activity of the smooth musculature is claimed to be initiated by electrical activity [19, 20, 21]. The source of the electric waves in the urinary bladder has not yet been conclusively determined. There have been reports that the waves in the gut are generated by the ICC [14, 15, 16, 17, 18], the presence of which could also be demonstrated in the urinary bladder [24]. In a previous study [9], we showed that the vesical electrical waves spread caudad in the bladder, postulating the possible existence of a pacemaker at the bladder vault from which the electrical waves could be generated. In the healthy volunteers, the regularity of the electric waves, presumably generated from the pacemaker, apparently leads to regular vesical contractions.

The electrical waves recorded from both the empty and full bladders are probably responsible for the basal tone of the urinary bladder at rest. The increased amplitude for the full bladder compared to the empty one seems to denote an elevated vesical tone in response to bladder filling.

It may be argued that, as we use surface electrodes for recording, simultaneous registering of the EMG activity of the abdominal striated muscles could occur. The striated muscles, however, do not evoke electrical activity while being relaxed because, unlike the smooth muscles, they have no resting electrical activity. As already mentioned, we asked the patients to relax their abdominal wall while the EMG was being recorded.

Role of the electrical activity of the bladder in the pathogenesis of the OAB

The significance of the dysrhythmic pattern of the resting electrical activity of the OAB in the pathogenesis of the OAB needs to be discussed. The electrovesicographic recordings of dysrhythmia have demonstrated different electrical activity in various areas of the bladder. They registered active areas of the urinary bladder as evidenced by the SWs with high frequency and amplitude. These

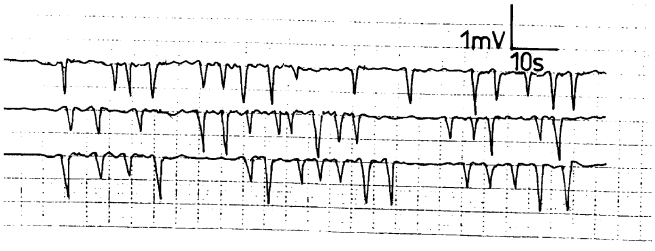

Fig. 4 Electrovesicogram of a patient with overactive bladder while the bladder was full showing a dysrhythmic pattern

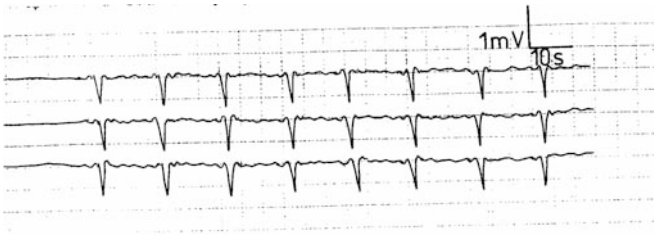

Fig. 3 Electrovesicogram of the same subject as Fig. 2 after bladder evacuation. The slow waves show a similar frequency and conduction velocity to those of the full bladder but a lower amplitude

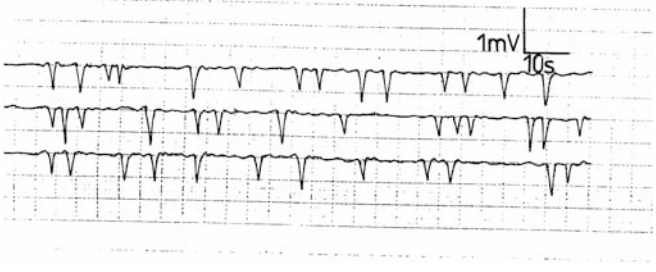

Fig. 5 Electrovesicogram of the same patient of Fig. 4 after evacuation. The slow waves show a dysrhythmic pattern which is different from that of the full urinary bladder

tachyrhythmic areas seem to represent areas in the vesical musculature with high contractile activity. Other, probably adjacent areas, exhibited less electrical activity with SWs of lower frequency, amplitude and conduction velocity; these bradyrhythmic vesical segments seem to be hypoactive with a low contractile activity. It appears that the electrically hyperactive areas of the vesical musculature initiate the urgency and the involuntary vesical contractions which may be responsible for the uncontrollable urination of the OAB. It seems that the various degrees of urinary incontinence in the OAB depend on the incidence of the electrically hyperactive bladder areas; urinary incontinence frequency is assumed to increase with the increase of these areas.

The cause of the dysrhythmic electrical activity of the OAB is unknown. The fact that the electrical waves in the urinary bladder spread caudad is likely to denote that the source of these waves is located in the vault of the bladder [9]. It appears that under normal physiologic conditions, when the bladder filling reaches a certain level, the presumed vesical pacemaker is stimulated with a resulting generation of electrical waves and vesical contraction. The waves apparently spread caudad in a regular way as has been demonstrated in the current and previous studies [9]. The waves were regular and similar all through the bladder in each subject. We assume that upon vesical filling the initiation and spread of the waves in the bladder are spontaneous and involuntary. Meanwhile, vesical contractions and urination are under voluntary control. This regular wave pattern appears to be responsible for the regular and diffuse vesical contraction. By contrast, in the OAB the variety of wave patterns we encountered in the individual bladder is believed to be induced by a disordered pacemaker which seems to generate dysrhythmic waves with variable frequency, amplitude and conduction velocity. The waves spread in the vesical musculature with this irregular pattern, leading to irregularities in vesical contractibility. The cause of the pacemaker dysfunction is not known and needs further study. Vesical dysrhythmia is suggested to be comparable with cardiac dysrhythmia which results from a disordered cardiac pacemaker and leads to irregularities in heart beat.

In conclusion, the OAB exhibited a ''dysrhythmic'' electrical pattern with irregular SWs activity. In a single recording, the electrovesicogram revealed areas of different electrical activity ranging from tachyrhythmia and bradyrhythmia to arrhythmia. This dysrhythmic electrical pattern is suggested to effect the abnormal vesical contractions and may explain the clinical manifestations characteristic of the OAB. Further studies into the cause of this dysrhythmic pattern, as well as into the possibility of using the electrical activity recordings of the bladder as an investigative tool in diagnosing OAB, are required.

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