

Electrophysiologic Activity of the Tunica Albuginea and Corpora Cavernosa: Possible Role of Tunica Albuginea in the Erectile Mechanism

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ABSTRACT

Introduction. It is claimed that the tunica albuginea (TA) shares in the erectile mechanism by compressing the emissary veins passing through it. However, the TA does not contain smooth muscle fibers.

Aim. We investigated the hypothesis that TA lacks a contractile activity on the emissary veins passing through it.

Methods. Fourteen healthy male volunteers (mean age 35.2 ± 4.3 years) were studied. The electromyographic (EMG) activity of the TA and corpora cavernosa (CC) was individually recorded in the flaccid and erectile phases by EMG needle electrodes. Recording was performed in the upper, middle, and lower third of the TA and CC on one and then on the contralateral side.

Main Outcome Measures. The TA lacks a contractile activity on the emissary veins passing through it.

Results. The EMG of the CC in the flaccid phase recorded regular slow waves and random action potentials. The wave variables in the erectile phase exhibited a significant decrease ($P < 0.01$) compared with the variables in the flaccid phase of the same subject. The TA EMG showed no electric waves in the flaccid or erectile phases. These recordings were similar from the upper-, middle-, and lower-third of the penis, and were reproducible from the contralateral CC.

Conclusions. Electric waves were recorded from the CC in the flaccid phase; wave variables decreased at erection. In contrast, the TA showed no electric waves in the flaccid or erectile phases. It appears that the TA acts as a CC covering sheet which expands passively at erection, and shares in compressing the subtunica venular plexus between it and the tumescent CC. **Shafik A, Shafik AI, El Sibai O, and Shafik AA. Electrophysiologic activity of the tunica albuginea and corpora cavernosa: Possible role of tunica albuginea in the erectile mechanism. J Sex Med 2007;4:675–679.**

Key Words. Erection; Impotence; Smooth Muscle; Corpora Cavernosa; Electromyography; Slow Waves; Action Potentials

Introduction

The tunica albuginea (TA) of the penis covers the penile corporal tissue. It consists of collagen fibers impregnated with elastic fibers [1,2]. It has various histologic patterns, the commonest being the two-layered TA (with an incidence of 71.4%), which consists of an inner circular and an outer longitudinal layer [2,3]. The second commonest (with an incidence of 21.4%) is the three-layered pattern with an additional outer circular

layer. The single-layered TA is formed of circular fibers and occurs in only 7.2% [3]. Intracavernosal pillars arise from the inner layer and radiate into the corpora cavernosa (CC). Together with the intercavernosal septum, they provide support to the erectile tissue [4,5]. The TA of the corpus spongiosum consists of only one layer of circular fibers [5]. The TA does not contain smooth muscle fibers [1–5].

It is claimed that the TA shares in the erectile mechanism by compressing the emissary veins

passing through it [1,4,6,7]. However, this claim appears to be theoretical, as to our knowledge, no experimental studies could be traced in the literature proving this concept.

As the penile TA does not contain smooth muscle fibers, we hypothesized that it lacks a contractile activity on the emissary veins passing through it. This hypothesis was investigated in the current study.

Materials and Methods

Subjects

Fourteen healthy volunteers (mean age 35.2 ± 4.3 years, range 28–42) were enrolled in the study. They signed an informed consent form after being informed about the nature of the study and their role in it. The subjects were married, led a normal sexual life, and had fathered children. They had normal erection as gauged by the International Index of Erectile Function. The results of physical examination, including neurologic assessment, were unremarkable. The laboratory work, which comprised blood count, renal and hepatic function tests, and electrocardiogram, recorded normal findings. The study was approved by the Review Board and Ethics Committee of the Cairo University Faculty of Medicine.

Methods

The electromyographic (EMG) activity of the TA and CC was individually recorded in the flaccid and erectile phases by means of concentric EMG needle electrodes (Type 13L49, Disa, Copenhagen, Denmark) measuring 40 mm in length and 0.65 mm in diameter. A needle electrode was introduced into the TA and another one into the CC in the middle third of the penis. The needle electrode for the TA was introduced through the penile skin and into the TA for 0.75 mm; a gritty sensation was felt while the needle electrode was inserted into the TA. The needle electrode for the CC was introduced for 0.5–0.75 cm from the penile skin. The needle electrodes were applied after full erection had occurred, so as to avoid needle movement during the phases of erection. A ground electrode was applied to the thigh, and a strain-gauge respiratory transducer to the thoracic wall. The proper location of the needle electrode into the TA was controlled by feeling the gritty sensation on needle insertion, absence of erectile waves recording, and on deeper penetration, the CC waves were recorded. We used to insert the needle electrode into the penis until the CC waves

appear, and then the needle was withdrawn until the waves had disappeared, while rocking the needle electrode to feel the gritty sensation of the TA.

When the electric activity of the TA and CC in the middle third of the penile shaft had been recorded, we transferred the electrodes consecutively to the upper, then lower third of the penis, and registered the electric activity. This was followed by recording the EMG activity from the upper, middle, and lower third of the contralateral TA and CC.

The aforementioned recordings of the EMG activity of the TA and CC in the flaccid phase were repeated while the penis was erected. Penile erection was induced by injecting the CC with papaverine in a dose of 60 mg. The erection lasted 2–3 hours (mean 2.4 ± 0.07).

A standard EMG apparatus (Type MES, Medelec, Woking, UK) was used to display and amplify the recorded potentials. Films of these potentials were taken on a light-sensitive paper (Linagraph type 1895, Kodak, London, UK), from which measurements of the motor-unit action potentials (APs) were obtained. The EMG signals were, in addition, stored on an FM tape recorder (type 7758A; Hewlett-Packard, Waltham, MA, USA) for further signals as required. All filtered signals were collected and recorded using an online computer with data acquisition and analysis software (Chart V 4.2; AD Instruments, Castle Hill, Sydney, Australia). The acquisition rate was 10 Hz, and the EMG nominal band width was 0.1–5.0 Hz.

To ensure reproducibility of the results, the EMG 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 mean \pm SD. Significance was ascribed to $P < 0.05$.

Results

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

EMG Activity of the CC

Slow waves (SWs) were recorded in the flaccid phase from the electrode introduced into CC. The wave was negatively deflected (Figure 1A) and its shape was constant when the recordings were repeated from the same site. The SWs exhibited similar frequency, amplitude, and conduction

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