THE RELATIONSHIP BETWEEN PELVIC FLOOR AND ABDOMINAL MUSCLE ACTIVATION AND THE GENERATION OF INTRAVAGINAL PRESSURE IN HEALTHY CONTINENT WOMEN

Stéphanie J.Madill and Linda McLean
School of Rehabilitation Therapy, Queens University, Kingston, Ontario, Canada

INTRODUCTION

Stress urinary incontinence (SUI) is "the complaint of involuntary leakage [of urine] on effort or exertion, or on sneezing or coughing"[1]. It affects 26% of women aged 30 to 59, peaking in the 40 to 49 year age group[2]. It is caused by the failure of the pelvic floor muscles (PFM) and the urethral sphincters to resist increases in intra-abdominal pressure[3], however, the mechanism by which the PFM and abdominal muscles work together to maintain continence is not well understood.

METHODS

PFM electromyography (EMG) data were acquired using a Femiscan™ probe seated in the vagina[4]. The probe had two pairs of bipolar bar electrodes mounted laterally, and was modified by mounting a pressure transducer in a hole cut through its posterior surface. Surface EMG data were recorded from rectus abdominis (RA), transversus abdominis (TA), internal obliques (IO) and external obliques (EO) using Meditrace™ 133 surface Ag-AgCl adhesive electrodes. All EMG data were amplified using Bortec AMT-8 amplifiers, and both EMG and pressure data were acquired at 1kHz using a 16-bit Analog to Digital Converter and Labview v. 6.1.

Resting data were recorded first, with each subject positioned in supine and asked to relax their PFM and abdominal muscles. After a period of instruction to familiarize subjects with the proper performance of a PFM contraction, volunteers performed three repetitions of a maximum voluntary contraction of their PFM while EMG and pressure data were recorded simultaneously from all sensors.

All pressure and EMG data were smoothed by computing the root mean square (RMS) value using a moving window of 20ms across the contraction time, less the resting RMS value The data were then normalized based on the maximum smoothed pressure or EMG amplitude achieved during each contraction. The normalized pressure vs. EMG curves were ensemble averaged, and the equations of these curves were computed and tested ($p<0.05$). Significant curves were used to model the EMG vs. pressure relationship.

RESULTS AND DISCUSSSION

Thirteen urinary continent women, mean age 36.3 $\pm$9.9 years (10 nulliparous, three parous) participated in the study. Since the EMG amplitudes from the two sides of the PFM were highly correlated in all cases (cross-correlation coefficient 0.90, 95% confidence interval 0.89-0.92), the side with the larger EMG amplitude was used in the analysis for each subject and each contraction.

The ensemble average EMG vs pressure curves for three of the abdominal muscles (RA, TA and IO) were "S" shaped (See Figure 1), whereas that for the PFM showed a steep initial rise followed by a leveling off. (Figure 2) The EO versus pressure curve did not produce any predictable pattern ($p>0.09$). All of the curves, except that for RA, were best defined by second order polynomial equations ($p<0.05$). RA was best defined by a third order polynomial equation.

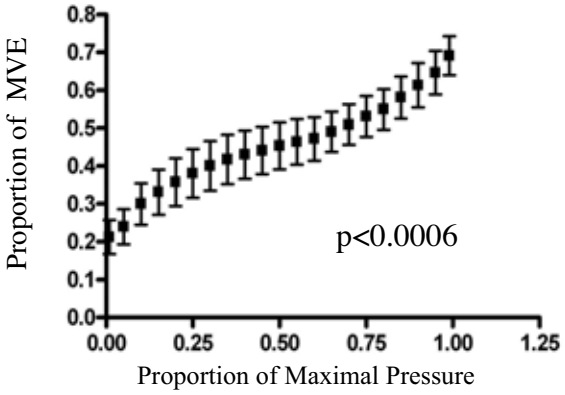

Figure 1. Ensemble average curve (n=13) for normalized IO EMG versus lower vaginal pressure. Squares indicate the mean proportion of MVE for each pressure increment while the whiskers indicate one standard deviation.

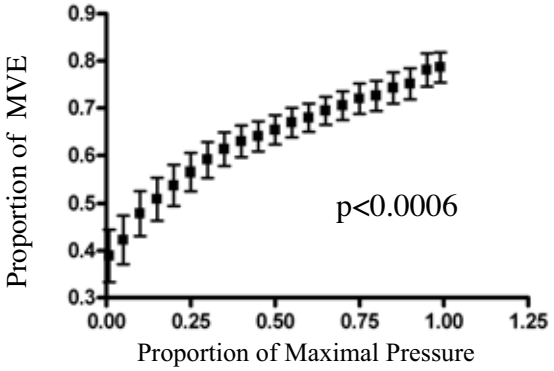

Figure 2. Ensemble average curve (n=13) for normalized PFM EMG versus normalized vaginal pressure. Squares indicate the mean percent MVE for each pressure increment while the whiskers indicate one standard deviation.

In voluntary PFM contractions, lower intravaginal pressure in urinary continent women is not solely the product of PFM activation; it receives significant contributions from TA, RA and IO. The EO muscles did not appear to have a predictable pattern of activation in response to a voluntary PFM contraction and are therefore thought to contribute minimally to the generation of lower intravaginal pressure.

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