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THE EFFECT OF PUDENDAL NERVE BLOCKADE ON LEVATOR ANI FUNCTION

Hypothesis / aims of study

The aim of this study was to investigate the possible pudendal nerve innervation of the levator ani muscle by measuring vaginal pressures, anal pressures, and urogenital hiatus measurements before and after pudendal nerve blockade.

Study design, materials and methods

Eleven nulliparous women without symptoms of anal or urinary incontinence were studied after informed consent was obtained. All subjects were instructed to contract the pelvic floor during a simultaneous digital vaginal examination. Throughout the study, electromyography (EMG) was recorded with concentric needle electrodes placed in the external anal sphincter (EAS) and puborectalis muscle (PRM). Vaginal manometry was performed with a 6 cm long, reverse-perfused sleeve sensor. The sleeve sensor measures the highest pressure along its length. Vaginal manometry was performed at rest and during squeeze (contraction of the pelvic floor muscles). Anal manometry was performed with a 4.5 mm diameter catheter with four side-holes placed circumferentially (90 degrees apart) at the same axial level. The catheter was placed in the anal canal and was withdrawn at a constant speed of 8 mm/sec using a motorized puller. Anal manometry was performed at rest and during squeeze. Threedimensional ultrasound of the pelvic floor was performed with a 5-9 MHz transducer placed on the perineal body. These images were collected at rest and during squeeze. The threedimensional image was analyzed with a software program that allows for manipulation of the 3D volume. The volume was rotated to view the urogenital hiatus in the plane of the levator ani muscles. In this plane, the distance between the lower end of the pubic bone and the apex of the anorectal angle was measured and defined as the anterior-posterior length of the urogenital hiatus (Figure 1). Two independent investigators measured this distance and the inter-observer correlations were determined. PNB was performed transvaginally with injection of 10 ml of 1% lidocaine bilaterally, approximately 8 mm medial to the tip of the ischial spine and to a depth of approximately 1 cm using a standard pudendal nerve block needle without the 1/4 inch spacer. All manometric recordings and ultrasound measurements were repeated after the PNB. All pressure measurements were made relative to atmospheric pressure. Data were examined for normality and student's paired t-test was used to compare means where the data was normally distributed. The Wilcoxon signed rank test was used for nonnormally distributed data. A p value < 0.05 was considered significant. Data are shown as mean ± standard deviation unless otherwise noted.

Figure 1



Results

The effect of the PNB was assessed by sensory and motor testing. Based on the sensory exam, six subjects were felt to have complete bilateral block. The PNB reduced the EMG activity of both the EAS and the PRM in all subjects. The mean reduction in the EMG during voluntary squeeze was 73% (range: 41% to 94%) for the EAS and 55% (range: 14% to 92%)

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for the PRM (Figure 2). Therefore, the PNB technique that we used was effective in paralyzing both EAS and PRM, albeit not completely. After PNB, mean vaginal resting pressures decreased from 19 ±10 to 15 ±10 mmHg (p<0.05) and mean vaginal squeeze pressures decreased from 61 ±29 to 37 ±24 mmHg (p< 0.05) (Figure 3). The PNB reduced maximum resting pressure in the anal canal from 98 ±24 to 51 ±17 mmHg and squeeze pressure from 144 ±43 to 70 ±38 mmHg (p<0.05). After PNB, the anterior-posterior length of the urogenital hiatus increased from 51 ±4 to 55 ±5 mm at rest (p<0.05) and increased from 47 ±3 to 52 ±5 mm during squeeze (p<0.05) (Figure 4). The inter-observer correlation coefficient was 0.79, p<0.001).

Figure 3





Figure 4



Interpretation of results

The most important findings of our study is that pudendal nerve blockade significantly reduced vaginal pressures, increased resting length of the urogenital hiatus, reduced shortening of the urogenital hiatus during contraction, and reduced EMG activity of the PRM. Our hypothesis that pudendal nerve does not innervate pelvic floor muscles was not proven. We expected that if the levator ani was innervated by direct sacral branches and not the pudendal nerve, pudendal nerve blockade would have no effect on vaginal pressures or urogenital hiatus measurements. The implication of our study is that pudendal nerve injection affects both, the levator ani and the EAS. Therefore, our data provides strong evidence that the pudendal nerve innervates the levator ani muscle.

Concluding message

Pudendal nerve blockade decreases vaginal and anal canal pressures, increases length of urogenital hiatus and decrease EMG activity of the PRM, all of which suggests the pudendal nerve does innervate the levator ani muscles.