Effects of pelvic floor muscle contraction on anal canal pressure

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Padda BS, Jung SA, Pretorius D, Nager CW, Den-Boer D, Mittal RK. Effects of pelvic floor muscle contraction on anal canal pressure. Am J Physiol Gastrointest Liver Physiol 292: G565–G571, 2007. First published October 5, 2006; doi:10.1152/ajpgi.00250.2006.—The role of pelvic floor muscle contraction in the genesis of anal canal pressure is not clear. Recent studies have suggested that vaginal distension increases pelvic floor muscle contraction. We studied the effects of vaginal distension on anal canal pressure in 15 nullipara asymptomatic women. Anal pressure, rest, and squeeze were measured using station pull-through manometry techniques with no vaginal probe, a 10-mm vaginal probe, and a 25-mm vaginal probe in place. Rest and squeeze vaginal pressures were significantly higher when measured with the 25-mm probe compared with the 10-mm probe, suggesting that vaginal distension enhances pelvic floor contraction. In the presence of the 25-mm vaginal probe, rest and squeeze anal pressures in the proximal part of the anal canal were significantly higher compared with no vaginal probe or the 10-mm vaginal probe. On the other hand, distal anal pressures were not affected by any of the vaginal probes. Ultrasound imaging of the pelvic floor revealed that vaginal distension increased the anterior-posterior length of the puborectalis muscle. Atropine at 15 μg/kg had no influence on the rest and squeeze anal pressures with or without vaginal distension. Our data suggest that pelvic floor contractions increase pressures in the proximal part of the anal canal, which is anatomically surrounded by the puborectalis muscle. We propose that pelvic floor contraction plays an important role in the fecal continence mechanism by increasing anal canal pressure.

REST AND SQUEEZE Pressures in the anal canal are mostly the result of internal anal sphincter (IAS) and external anal sphincter (EAS) contraction, respectively (6, 15, 27). The contraction of the puborectalis muscle (PRM), a part of the pelvic floor muscles, is thought to enhance the fecal continence mechanism by decreasing the anorectal angle (3, 21). Using a novel instrument, the “perineal dynamometer,” Fernandez-Fraga et al. (8) demonstrated that the levator ani or pelvic floor traction force is impaired in patients with fecal incontinence. This force improves with pelvic floor training, suggesting an important role of pelvic floor muscles in the fecal continence mechanism. The mechanism by which pelvic floor traction enhances fecal continence is not entirely clear. A recent study (14) from our laboratory showed that PRM contraction increases pressure in the proximal part of the anal canal and that EAS contraction increases pressure in the distal part of the anal canal. Our conclusions were based on the observations made by three-dimensional ultrasound images (anatomy) and manometry (function) of the anal canal.

There is a high-pressure zone (HPZ) in the vaginal canal, which is thought to be due to the contraction of pelvic floor muscles (2, 12). Distension of the vaginal canal using a speculum increases vaginal closure force (17, 18). We as well as Bo et al. (1, 19) found that the magnitude of pressure in the vaginal HPZ is directly related to the size of the vaginal probe or, in other words, vaginal distension. It is likely that the increase in vaginal closure force (measured with the vaginal speculum) and the increase in pressure of the vaginal HPZ with the increase in the vaginal probe reflects the length-tension properties of the pelvic floor muscle, whereby an increase in the length of pelvic floor muscles with vaginal distension increases the force of pelvic floor muscle contraction. We took advantage of the above observations to determine the effects of pelvic floor muscle contraction on anal canal pressure. We studied the effects of vaginal distension on anal canal pressure. Our hypothesis is that enhanced pelvic floor contraction induced by vaginal distension enhances anal canal pressure.

MATERIALS AND METHODS

The University of California-San Diego Institutional Review Board approved the study protocol, and each subject signed an informed consent form prior to participation in the study. A total of 15 nulliparous women (mean age: 35 yr, range: 20–55 yr) were studied for this protocol. Each subject completed a medical history and urinary and fecal incontinence scoring questionnaire to confirm the absence of incontinence symptoms (25, 26).

Pressures were recorded in the anal and vaginal canals using two different types of water-perfused manometry catheters of the same diameter (4.5 mm). Anal manometry was performed using a four side-hole catheter; all side-holes were placed circumferentially, 90° apart, and at the same axial level. The manometry catheter was placed in the rectum, and rest and squeeze anal pressures were measured using the station pull-through technique. The catheter was withdrawn every 5 mm during the station pull through. Vaginal manometry was performed with a separate 4.5-mm-diameter catheter that was equipped with a sleeve sensor (5). The catheter was placed in custom-designed catheter holders of 10- and 25-mm diameters. These probe holders were made of noncompliant material (propylene) and had a groove for the insertion of a sleeve-sensor manometry catheter into the holder (Figs. 1 and 2). The catheter was placed in the holder with the pressure-sensing surface of the sleeve facing outward. The holder along with the catheter was placed in the vagina with the sleeve sensor facing in the posterior midline direction (Fig. 2). All pressures...
were recorded using a Polygraf HR (Medtronic, Minneapolis, MN) and a personal computer.

Study design. Prior to the start of each study, subjects were trained to contract pelvic floor muscles by the following prompt: “squeeze as if you were trying to prevent a bowel movement or hold a stream of urine.” Anal pressures were recorded using the station pull-through technique for a total of three times: 1) with no vaginal probe, 2) with a 10-mm vaginal probe in place, and 3) with a 25-mm vaginal probe in place. At each station, pressures were recorded at rest and during the pelvic floor muscle contraction. Subjects were instructed to squeeze for 10 s; each squeeze was followed by a 30-s relaxation period to minimize fatigue. In 8 of 15 subjects, the above protocol was repeated after the administration of atropine (15 μg/kg). The latter was administered intravenously, and pressures were measured 10–15 min after the injection.

The effect of vaginal distension on the length of the PRM was measured using the pelvic floor ultrasound imaging technique that we described previously (11). For these experiments, a polyvinyl bag with a maximal diameter of 35 mm was placed partly in the vagina and partly outside. The bag was inflated with water volumes of 40 and 70 ml, and, at each bag volume, the three-dimensional ultrasound volume of the pelvic floor muscle was obtained using the 3D Philips HD11 US system (Phillips Medical Systems, Bothell, WA). The ultrasound transducer was placed on the perineum and was directed in the cranial direction.

Data analysis. All pressures were measured in reference to the atmosphere as the zero pressure. At each station, pressures were recorded at rest and during squeeze in all four quadrants of the anal canal. The vaginal probe recorded pressure in the posterior direction of the vaginal HPZ during rest and squeeze. Resting anal and vaginal pressures were determined during the 5-s period before the subject was asked to squeeze. The squeeze pressure was the peak pressure during the 10-s squeeze period. Based on our previous study (14), in which we performed three-dimensional ultrasound images of the anal sphincter complex, proximal and distal parts of the anal canals were surrounded by the PRM and EAS, respectively. The craniocaudal lengths of the PRM and EAS were ~2–3 cm each. We hypothesized that the pressure in the proximal part of the anal canal was related to the IAS and PRM. On the other hand, pressure in the distal part of the anal canal was due to the IAS and EAS. Therefore, we analyzed the highest pressure in the anal canal at rest and with voluntary squeeze in the proximal (3 cm above the anal verge) and distal (within the distal 3 cm of the anal verge) segments as related to the PRM and EAS, respectively. To determine whether pressures in each of these zones were affected by vaginal distension, a paired t-test was performed. Asymmetry of the anal canal pressure in the PRM and EAS zones was determined by examining the highest pressure recorded in each quadrant of the PRM and EAS zones. To determine whether the pressures in the anal canal were asymmetric, ANOVA was performed on the measurements in each of the four quadrants. If the ANOVA proved that the pressures were asymmetric (P < 0.05), then a Holm-Sidak test, a pairwise comparison of pressures measured in each

Fig. 1. Manometry catheters and the vaginal probe holder. Vaginal pressure was recorded using a 4.5-mm manometry catheter. The manometry catheter was placed inside probe holders with diameters of 10 and 25 mm. The sleeve sensor recorded the maximum pressure along the length of the vaginal high-pressure zone (HPZ). There were two side-hole sensors that were placed above and below the sleeve sensor, and they recorded vaginal proximal (abdominal) and atmospheric pressures, respectively. The anal catheter was also 4.5 mm in diameter and had four side-hole sensors that were located on the same axial plane and were 90° apart from each other.

Fig. 2. Schematic of puborectalis muscle (PRM) anatomy. The PRM is a sling-like muscle that wraps around the urethra, vagina, and anal canal. The schematic shows the placement of the vaginal probe (facing posterior in the vaginal HPZ) and anal catheter. The two zones of the anal canal, the PRM zone and the external anal sphincter (EAS) zone, are displayed with their appropriate depths shown in parentheses.
of the four quadrants, was performed to determine whether a particular quadrant recorded higher pressure than the others.

Three-dimensional ultrasound volumes were viewed with the Q-lab 4.2 software program (Phillips Medical Systems). A two-dimensional ultrasound image of the pelvic floor was extracted from the three-dimensional ultrasound volume in the transverse plane of the pelvic floor (a line connecting the lower end of the pubic bone with the anorectal angle). The anterior-posterior length of the PRM was determined from the two-dimensional ultrasound image and represented the distance between the pubic bone to the inner surface of the PRM.

All data are presented as means \( \pm \) SE.

**RESULTS**

**Effect of vaginal distension on vaginal pressure.** The sleeve sensor measured the peak pressure in the vaginal HPZ, and the side holes distal and proximal to the sleeve reflected abdominal and atmospheric pressure, respectively. Rest and squeeze pressures in the vaginal HPZ before and after atropine are shown in Fig. 3. Both 10- and 25-mm probes showed a significant increase in vaginal pressure with squeeze \( (P < 0.001) \). Rest and squeeze pressures were significantly higher when the vaginal HPZ was recorded with the 25-mm vaginal probe compared with the 10-mm vaginal probe \( (P < 0.001) \). There were no increases in the distal vaginal pressure (abdominal pressure) with increases in probe size. Atropine had no effect on pressure in the vaginal HPZ.

**Anal canal pressure profile: rest and squeeze pressures with and without the vaginal probe.** The anal canal was \( \sim 5.0 \) cm long. Rest pressures in the anal canal increased from the proximal (cranial) to the distal (caudal) end, and the highest or peak pressure was located at \( \sim 2 \) cm from the anal verge. With each squeeze, there was an increase in anal canal pressure at each station, with the biggest pressure increase occurring at \( \sim 1–2 \) cm from the anal verge. There were no differences in rest and squeeze pressures in either of the two anal canal zones between no vaginal probe and the 10-mm vaginal probe (Fig. 4). The 25-mm vaginal probe, on the other hand, resulted in increases in rest and squeeze anal canal pressures in the proximal or PRM zone but not in the distal or EAS zone. Rest pressure increased in the PRM zone from 35 to 45 mmHg \( (P = 0.05) \), and squeeze pressure increased in the PRM zone from 83 to 134 mmHg \( (P < 0.001) \). Difference between rest and squeeze anal pressures with no probe (48 mmHg) and with the 25-mm probe in place (88 mmHg) were also highly significant in the PRM zone \( (P < 0.001) \). On the other hand, the EAS zone did not show a different pressure in the absence or presence of the 25-mm vaginal probe at either rest (77 and 70 mmHg, \( P = 0.08 \)) or squeeze (173 and 172 mmHg, \( P = 0.71 \); Fig. 5).

**Circumferential pressure asymmetry of the anal canal.** Anal canal pressures in the four quadrants of the PRM and EAS zones are shown in Fig. 6. Rest and squeeze pressures in the PRM zone of the anal canal revealed circumferential pressure asymmetry in the four quadrants \( (P < 0.001) \). Posterior rest and squeeze pressures in the PRM zone were significantly higher than in the other three quadrants \( (P < 0.001) \). On the other hand, rest and squeeze pressures in the EAS zone were symmetrical, i.e., pressures did not differ significantly among

![Fig. 3. Effects of vaginal distension and atropine on vaginal pressure. Vaginal pressure is shown with two probe sizes, 10 and 25 mm, during rest and squeeze. There were significant increases in vaginal rest and squeeze pressures with the larger probe \( (P < 0.05) \). Atropine had no significant effect on vaginal pressure. Error bars represent SEs.](http://ajpgi.physiology.org/)

![Fig. 4. Individual effect of vaginal distension on rest and squeeze anal pressures. The anal pressure profiles for an individual subject are displayed with no vaginal probe and with 10- and 25-mm vaginal probes in place. The anal pressures in the proximal (PRM) and distal (EAS) zones are labeled; note the increases in pressures in the PRM but not EAS zone with the 25-mm vaginal probe. The 10-mm vaginal probe had no effect on anal pressures.](http://ajpgi.physiology.org/)
the four quadrants ($P = 0.07$ and $P = 0.27$, respectively). Vaginal distension with the 25-mm probe resulted in increases in rest and squeeze pressures in the PRM zone. Increase in PRM zone pressures with vaginal distension were observed in all four quadrants ($P < 0.001$), and the circumferential pressure asymmetry was preserved. On the other hand, vaginal distension had no effect on pressures of the EAS zone of the anal canal at rest and squeeze ($P = 0.932$).

**Effect of smooth muscle relaxant atropine on anal canal pressure.** Atropine (15 μg·mg/kg) did not affect rest or squeeze pressures in the anal canal. Vaginal rest and squeeze pressures were also unaffected by atropine (Figs. 3 and 7).

**Effect of vaginal distension on PRM length.** The anterior-posterior PRM length increased with vaginal distension (Fig. 8). At the 40-ml bag volume, the mean anterior-posterior length in eight subjects was $5.0 \pm 0.3$ cm, and with 70-ml distension, the PRM length increased to $5.7 \pm 0.5$ cm.

**DISCUSSION**

Our data show the following: 1) an increase in vaginal probe size increases pressure in the vaginal HPZ, 2) a 25-mm probe in the vagina increases pressure in the proximal (PRM zone) but not distal (EAS zone) part of the anal canal, 3) anal pressure is circumferentially asymmetric in the proximal (PRM zone) but not in the distal (EAS zone) part of the anal canal, 4) anticholinergic agent atropine neither affects rest nor squeeze pressures in the anal canal irrespective of the vaginal probe, and 5) vaginal distension increases the length of the PRM.

The major goal of our study was to determine the role of pelvic floor muscles in the genesis of anal canal pressure. Our previous study (14) showed that with voluntary squeeze, there was an increase in anal canal pressure throughout the length of the anal canal. Ultrasound images of the anal canal showed that the proximal part of the anal canal was composed of the IAS.
and PRM and the distal part of the anal canal was composed of the IAS and EAS. Based on the observation that there was an increase in anal canal pressure with voluntary squeeze in proximal and distal anal zones, we proposed that these pressure increases were related to contractions of the PRM and EAS, respectively (14). How does the PRM, a “U”-shaped muscle,
increase anal canal pressure? Our hypothesis is that the contraction of the two arms of the PRM lifts the anal canal in the anterior direction and compresses it against the vagina, urethra, and back of the pubic bone. In accordance with our hypothesis, anal canal pressure was circumferentially asymmetric in the proximal zone with higher pressure in the posterior direction, an observation similar to that of Taylor et al. (24). Furthermore, we found circumferentially asymmetric pressure in the vaginal HPZ the anterior and posterior pressures were higher than right and left pressures (12, 19). Finally, ultrasound and MRI studies (11, 13) have shown that during pelvic floor contraction, there is indeed a shortening of the anterior-posterior length of the pelvic floor hiatus, which is formed by the PRM.

Recently, we (19) found that pressures in the vaginal HPZ increase with increases in vaginal probe size. Others (17, 18) measured vaginal closure force using a vaginal speculum found that with an increase in anterior posterior length of the vaginal speculum, there is an increase in vaginal closure force. An increase in the vagina probe size or the vaginal speculum increases the length of the PRM. We speculate that the increase in vaginal pressure with the increase in the vaginal probe and the increase in vaginal closure force with the increase in the anterior-posterior length of the vaginal speculum are related to the length-tension properties of the PRM (one of the pelvic floor muscles). We took advantage of the above observations to provide further evidence for the role of the PRM in the genesis of anal canal pressure. Both rest and squeeze vaginal pressures were higher with the 25-mm probe compared with the 10-mm probe, suggesting that vaginal distension did indeed enhance pelvic floor contraction. Our anal canal pressure data show that the increase in the force of contraction of the pelvic floor muscle increases pressure in the proximal part but not in the distal part of the anal canal. Since the PRM surrounds the proximal but not distal parts of the anal canal, one would only expect pressure to increase in the proximal part of the anal canal, which is consistent with our hypothesis. We did not find differences in anal canal pressures between no probe and with the 10-mm vaginal probe; the reason may be that the difference in pelvic floor contraction between no vaginal probe and the 10-mm probe is relatively small.

Is the increase in anal canal pressure with vaginal distension related to active muscle contractions or passive stretches of the PRM? We can’t be sure if the rest anal pressure with the 25-mm vaginal probe in place is active or passive; however, the higher change between rest and squeeze pressures in the PRM zone supports an active PRM contraction. The definite proof for the active versus passive contribution to pressure requires one to study the effect of a skeletal muscle relaxant, i.e., general anesthesia, on anal canal pressure.

We tested the effects of the anticholinergic agent atropine on anal canal pressures with and without the vaginal probe in place. An in vitro study (20) has shown that atropine relaxes muscle strips of the IAS. An in vivo study (22) similar to ours, however, failed to find any effect of atropine on anal canal pressure. The lack of effect of atropine may be because of the influence of the PRM and EAS (skeletal muscles) on anal pressures. It is widely believed that 70% or more of the rest anal pressure is related to the IAS, a smooth muscle (7, 10). If the dose of atropine (15 μg/kg) we used has major effects on smooth muscles of the other regions, i.e., lower esophageal sphincter and esophagus (16, 23), then why was the resting anal canal pressure not altered by atropine? It is generally felt that the anal canal pressure reflects the sum of the contractions of the IAS and EAS; however, we suspect that may not the case. Transmission of EAS and PRM contractions across the IAS to the lumen of the anal canal (as measured by intraluminal manometry) is likely to be related to the wall properties of the IAS. If the latter is rigid or noncompliant, the EAS contraction will not be transmitted to the anal canal lumen. On the other hand, a strong EAS contraction is likely to be transmitted to the anal canal lumen; however, the luminal pressure may not be the sum of EAS and IAS contractions. We suspect that the EAS and PRM are in a state of fairly strong tonic contractions and rest anal pressure is mostly reflective of the EAS and PRM. However, if these muscles are paralyzed, as can be done under experimental conditions in animals (4) and humans (9), then the rest anal pressure is entirely due to the IAS. We cannot exclude the possibility that adrenergic (28) or other (other than cholinergic) influence is more important in the IAS and accounts for the lack of effect of atropine on resting anal canal pressure.

In summary, our study shows that PRM contraction contributes to rest and squeeze pressures in the proximal part of the anal canal. Rest and squeeze pressures in the vaginal canal most likely reflect tonic activity of the PRM. The PRM, a part of the pelvic floor, is a powerful muscle that “most likely provides” a strong sphincter mechanism to the anal canal and possibly the urethra. The PRM is likely to be a common link between the several subspecialties of medicine, i.e., gastroenterology, urology, urogynecology, and colorectal surgery.

GRANTS

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REFERENCES


