Investigation of Anal Motor Characteristics of the Sensori-Motor Response (SMR) using 3-D Anorectal Pressure Topography

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Short title: Sensori-Motor Response

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Abstract

INTRODUCTION: Desire to defecate is associated with a unique anal contractile response, the sensori-motor response (SMR). However, the precise muscle(s) involved is not known. **AIM:** To examine the role of external and internal anal sphincter and the puborectalis muscle in the genesis of SMR. **METHODS:** Anorectal 3-D pressure topography was performed in 10 healthy subjects during graded rectal balloon distention using a novel high definition manometry system consisting of a 6.4 cm probe with 256 pressure sensors arranged circumferentially. The anal pressure changes before, during, and after the onset of SMR were measured at every mm along the length of anal canal, and in 3-D by dividing the anal canal into 4x2.1 mm grids. Pressures were assessed in the longitudinal and anterior – posterior axis. Anal ultrasound was performed to assess puborectalis morphology. **RESULTS:** 3-D topography demonstrated that rectal distention produced a SMR coinciding with desire to defecate, and predominantly induced by contraction of puborectalis. Anal ultrasound showed that the puborectalis was located at mean distance of 3.5 cm from anal verge which corresponded with peak pressure difference between the anterior and posterior vectors observed at 3.4 cm with 3-D topography ($r=0.77$). The highest absolute and percentage increase in pressure during SMR were seen in the superior-posterior portion of anal canal, reaffirming the role of puborectalis. The SMR anal pressure profile showed a peak pressure 1.6 cm from anal verge in the anterior and posterior vectors, and distinct increase in pressure only posteriorly at 3.2 cm corresponding to puborectalis. **CONCLUSION:** Sensori-motor response is primarily induced by the activation and contraction of the puborectalis muscle in response to a sensation of desire to defecate.


Introduction

In humans, balloon distention of the rectum normally induces the recto-anal inhibitory reflex (RAIR), (Denny-Brown and Robertson 1935; Gowers 1877) as well as the recto-anal contractile reflex (RACR) (Garry 1933; Goligher and Hughes 1951; Sun et al. 1990a). RAIR is an enteric neuronal reflex that is mediated by nitric oxide, vasoactive intestinal peptide, and adenosine triphosphate and causes relaxation of internal anal sphincter (IAS) (Rattan et al. 1992). The rate and method of rectal distention (intermittent versus ramp) affects the properties of the RAIR, and this reflex response is classically absent in Hirschsprung’s disease and other conditions such as after circular rectal myotomy, and after lower anterior resection (Cortesini et al. 1983; Monteiro et al. 2007; Sun et al. 1990b).

In addition to the aforementioned reflexes, recently, we have described the sensori-motor response (SMR) (De Ocampo et al. 2007) that is also seen during balloon distention of the rectum. The SMR is a transient anal contraction that is usually seen overlying the initial relaxation phase of the RAIR (De Ocampo et al. 2007). In healthy subjects it coincides with the onset of a sensation for defecation (De Ocampo et al. 2007). A recent study showed that the SMR was abnormal in patients with rectal hyposensitivity suggesting a pathophysiologic role for this response in anorectal disorders (Remes-Troche et al. 2010). However, the precise origin of the anal contractile response, in particular the anal or pelvic floor muscle(s) that is involved in the genesis of the SMR is not known.

We hypothesized that the motor response that is seen during SMR is predominantly induced by a contraction of the puborectalis muscle. Therefore, our aims were; 1) to perform detailed characterization of the pressure profiles in the anal high pressure zone before, during,
and after the onset of SMR, and 2) to examine if the anal contractile response during the SMR originates from the puborectalis muscle, the anal sphincters, or both by performing detailed contour mapping and vector analysis of the anal canal using a novel 3-D High Definition Anorectal Manometry (HDM) system.
Materials and Methods

Subjects:

Ten healthy subjects (6 males, mean age 40 years range = 18-55) were recruited. All subjects filled out a Bowel Symptom Questionnaire that assessed their general and gastrointestinal health and anorectal function. All of them were asymptomatic and were not using laxatives or any other medications except multivitamins or oral contraceptives, and had no history of previous gastrointestinal surgery other than appendectomy. All subjects had a normal physical examination. The study was approved by the Institutional Review Board of the University of Iowa and all participants provided written informed consent.

Study Protocol:

After an overnight fast, subjects attended the motility lab. No routine bowel preparation was used. With the subject lying down in the left lateral position, a HDM probe (Sierra Scientific, Los Angeles, CA) was placed into the anorectum. This rigid probe measures 6.4 cm in length and has an outer diameter of 10.75 mm. It has 256 pressure sensors that are arranged in 16 rows, and each row has 16 circumferentially-oriented sensors. Each sensor has a 4 mm center linear spacing and 2.1 mm circumferential spacing. Although pressure is sensed at 4 mm intervals, the software linearly interpolates the space between sensors to provide measurements at 1 mm spacing with negligible error. The probe has a central lumen for inflation and a Luer lock at one end through which a balloon is attached. The probe is attached to an amplifier and recorder system and the manometric and topographic images are displayed on a computer monitor using specialized software (Motility Visualization System v.2.2, Sierra Scientific, Los Angeles, CA). The HDM system operates at a frequency response of >20Hz, a scan rate of
10Hz, and an output resolution of 0.1mmHg. The probe was calibrated immediately before the procedure by placement in a calibration chamber where it was zeroed to atmospheric pressure and set to a range of pressure up to 300 mmHg. The sensor calibration residual is +/- 2 mmHg in the 0-100 mmHg range and 2% of reading in the 100-300 mmHg range. The lubricated probe was inserted into the rectum, and placed such that the outermost panel of pressure sensors was located at the anal verge. Since the probe has circumferential pressure sensors, it was oriented such that the posterior portion corresponded to the dorsal aspect of the subject. This allowed for standardization of the vector measurements in the rectum and the anal canal.

The probe was held in place manually by one of the investigators throughout the study. After a rest period of 7 minutes, we assessed the resting and squeeze pressure profiles. Next, the rectal balloon was inflated in a stepwise, graded fashion using intermittent balloon distention technique (Rao et al. 2002; Rao et al. 1999). The subjects were given a Likert-like sensation chart and asked to describe their sensations. Rectal balloon distentions were performed by using a hand-held syringe in 10 cc increments until the subject reported a first sensation, and thereafter in 30 cc increments until maximum tolerable volume or a total volume of 320 cc was reached. Each distention was held for at least 30 seconds, and after a deflation and rest period of 2 minutes was re-inflated to the next volume. The subject was unaware of the timing or the volume of balloon distention. The threshold balloon volumes that induced a first sensation, desire to defecate, urge to defecate, and the maximum tolerable volume were recorded.

In seven subjects an anal ultrasound was performed by placing a 8 mm ultrasound probe (Hitachi, USA) into the rectum. The distance from the anal verge where the puborectalis muscle, was best visualized and its thickness at 3° clock and 9° clock positions were examined.
Data Analysis

The manometric and topographic HDM data were examined to assess the presence of SMR and to define its motor characteristics. An example of the SMR response as seen with manometry and 3-D topography is shown in Figure 1. The top line of the manometric image corresponds to the rectal balloon pressure and the lower lines represent the anal pressure changes at different distances from the anal verge. Rectal balloon distention induces a reflex relaxation of the anal canal or RAIR. Immediately after the RAIR reaches its nadir, the SMR is visualized manometrically as a transient contraction with an increase in anal canal pressure. This anal contractile response coincided with the subject signaling the onset for a desire to defecate.

The manometric anal pressure changes were measured at four time points during the course of rectal distention and are depicted in Figure 1. This figure displays simultaneous topographic and manometric images. The four time points designated as A, B, C, and D correspond to the following components of the recording; (A) the baseline anal pressure, (B) the anal relaxation pressure immediately before the onset of the SMR, (C) the highest amplitude of the anal contractile response, and (D) the residual pressure of the anal relaxation response. The topographic images shown in Figure 1 provide a 3-D pressure profile image, with the outer edges of the map showing the pressure changes in the posterior portion of the anal canal and the middle or the inner portion of the image showing the pressure changes in the anterior portion of the anal canal. The topographic view also allowed us to define the zero point or the anal verge which was the point where we observed a sharp drop in anal pressure that equaled atmospheric pressure (Figure 1B). After the anal canal length was set using the topographic pressure map, and the four time points were identified using the manometric recording, a detailed pressure analyses could be performed.
We analyzed the anal pressures at each of the four time points and at every millimeter distance along the length of the anal canal from the anal verge. From these individual pressure values, we calculated an average circumferential anal pressure at a resolution of one millimeter. The topographic pressure map was divided into 4 mm x 2.1 mm grids, and an average anal pressure per grid was calculated. This allowed us to provide a multidimensional analysis of the anal canal pressures both in the longitudinal and in the circumferential axes. Using this vector mapping method, we compared the pressures in the anterior half with the posterior half of the anal canal at a resolution of 4 millimeters.

The SMR anal contractile response was also analyzed for the changes in pressure. We measured anal pressures immediately before the onset of SMR as well as the peak SMR amplitude. The absolute SMR pressure increase was calculated at every millimeter distance along the length of the anal canal. Additionally, the anal canal was subdivided into one centimeter segments, and the percentage increase in pressure during the SMR and in each segment was calculated.

Anal ultrasound data was analyzed for the distance from the anal verge where the puborectalis muscle was best visualized in each subject.

Statistical Analysis

The anterior and posterior anal canal pressures during the baseline period were compared at every 4 mm distance along the anal canal using Student’s t test. Also, the anterior and posterior and circumferential anal pressures before, during, and after the SMR were compared at every millimeter distance using a two-tailed student’s t-test. The percent change in anal pressure for the four segments of the anal canal (A, B, C, D, Figure 1) were compared using ANOVA and
a Bonferroni's Multiple Comparison post Test analysis. Statistical significant was considered when p<0.05. The distance from the anal verge at which the puborectalis muscle was identified with anal ultrasound was compared and correlated with the distance from the anal verge where the pressure difference between the anterior and posterior half of the anal canal was the highest per subject with Pearson correlation coefficient. This most likely represented the level at which the puborectalis contributed the greatest fraction of the total anal pressure.
Results

A SMR was present in all of our subjects and was demonstrated both manometrically and topographically (Figure 1). The SMR coincided with the onset of a desire to defecate in all of our subjects. The average duration of the SMR was $6.4 \pm 0.5$ seconds and the mean rectal distention volume to induce SMR was $107 \pm 6$ cc. The rectal distention volume that induced a first sensation was $17 \pm 3.4$ cc, desire to defecate was $100.0 \pm 14.8$ cc, and urge to defecate was $160.0 \pm 15.1$ cc. All subjects tolerated the HDM and ultrasound recording satisfactorily.

SMR Pressures Profiles and Contribution of Puborectalis Muscle:

The topographic pressure image during each of the four time points that is shown in Figure 1 facilitates a better spatio-temporal visualization of the anal pressure changes. Here, one can visualize the activation or relaxation of the individual muscle components that surround the anal canal. For example, the puborectalis muscle can be identified in the superior-posterior (upper sides) section of the image. Through these sequential topographic pressure images, it is also possible to visualize the origin and progression of the anal contractile response that occurs during SMR and how the activation and contraction of the puborectalis muscle appears to be the predominant muscle contributing to this response.

This role of the puborectalis muscle was further confirmed by the measurements of the mean circumferential pressures immediately before and during the SMR and is shown in Figure 2. We found a statistically significant ($p \leq 0.05$) increase in anal canal pressure during the SMR from a distance of 2.4 cm to 3.2 cm from the anal verge. This location is represented with arrows in Figure 2 and corresponds to the puborectalis muscle. (Figure 4)

Anal Pressure Changes during SMR
The peak increase in anal pressure during SMR was seen at about 2.8 cm from the anal verge (Figure 2) and spanned a distance of 0.8 cm from 2.4 cm to 3.2 cm. Because this region corresponds to the locale of the puborectalis muscle within the anal canal, it further suggests that the anal contractile response during SMR is most likely induced by the puborectalis. The percent increase in anal pressure at each centimeter distance from the anal verge during the SMR is shown in Figure 3. The superior portion of the anal canal shows the greatest percent increase, with the superior 2 centimeters of the anal canal having a significant percentage pressure increase compared to the most distal section of the anal canal. This again reflects the contraction of the puborectalis muscle.

Vector and Longitudinal Changes in Basal Anal Sphincter Pressure

When the baseline anal pressure was analyzed along the length of the anal canal, the peak pressure was seen at 1.6 cm from the anal verge, both in the anterior and posterior vectors (Figure 4). A second smaller peak or hump was also seen consistently in all subjects at approximately 3.2 cm. However, this hump was only seen in the posterior region and was distinctively absent in the anterior pressure profile (Figure 4). The posterior specific hump spanned a distance of 1.6 cm and was seen between 2.4 cm to 4 cm from the anal verge. When we compared the mean anterior versus mean posterior basal sphincter pressures at 4 mm increments from the anal verge, there was no difference up to a distance of 2.4 cm, but a significant pressure difference between 2.4 cm and 4 cm (Table 1). This pressure difference and its location most likely correspond to the posterior sling fibers of the puborectalis muscle. Thus the puborectalis offers a distinct and important contribution to the resting anal sphincter profile, over and above the peak pressure offered by the internal anal sphincter in both the anterior and posterior vectors that begins at 1.6 cm.
Correlation between anal ultrasound and HDM pressure profile:

Anal ultrasound data was obtained in 7/10 subjects. We found that the distance from the anal verge where the puborectalis was best identified using anal ultrasound, was located at mean distance of 3.5cm. This corresponded nicely with the location from the anal verge where the pressure difference was highest between the anterior and posterior half of anal canal, mean 3.4cm, which most likely corresponded to the puborectalis (Table 2) p=0.6. The Pearson correlation coefficient was 0.77. Owing to technical reasons the ultrasound study could not be performed in 3 subjects.
Discussion

Our main objective was to examine the anal sphincter spatio-temporal changes during SMR and to delineate the predominant anal muscle component for this response using 3-D High Definition anorectal pressure topography. The SMR was consistently seen in all of our subjects overlying the RAIR. Its onset coincided with a desire to defecate in all of our subjects, as previously described (De Ocampo et al. 2007). Detailed topographic and vector characterization of the anal contractile response during the SMR revealed that the rise in anal pressure was largely due to the contraction of the puborectalis muscle. The absolute pressure change showed that the highest increase the anal region corresponded to the location of the puborectalis muscle. Additionally, the greatest percentage increase in pressure during the SMR was also seen at the superior portion of the anal canal, further supporting our hypothesis that the anal contractile response seen during the SMR is primarily due to the contraction of the puborectalis muscle. Furthermore, anal ultrasound studies provided independent validation that the pressure profiles observed in the superior-posterior portion of the anal canal with 3-D topography corresponded to the location where the puborectalis muscle provided the greatest fraction of the total anal pressure, reaffirming the role of this muscle in the genesis of this response.

The neurohumoral mediators for this anal excitatory muscle response are not known and remain to be explored. It is unclear if this is a locally mediated response similar to RAIR (Sangwan and Solla 1998) or whether it is modulated through a spinal or even central process. Because the response is invariably triggered with the onset of a need to defecate we suspect that this must be centrally modulated. Additionally, much like the graded changes in RAIR, (Monteiro et al. 2007; Sun et al. 1990b) the SMR also exhibits a larger response at higher volumes of rectal distention (De Ocampo et al. 2007). Supra spinal and parasympathetic
Pathways are implicated in the modulation of RAIR. We suspect that the sacral parasympathetics may be involved in the SMR as evidenced by modulation of puborectalis through stimulation of sacral plexus (Bharucha 2006; Percy et al. 1981). Future studies of the SMR in patients with neurological conditions and lower bowel dysfunction may demonstrate pathophysiological changes, much like the alterations in RAIR seen in patients’ with Hirschsprung’s disease (Sangwan and Solla 1998) and spinal cord injury (Beuret-Blanquart et al. 1990).

The functional significance of the SMR is also unknown. One possible explanation is that the SMR acts as an involuntary mechanism to signals of imminent defecation or help to maintain continence during the abrupt changes in rectal pressure from distention or movements of gas or stool in the distal rectum. This hypothesis may explain why the SMR is associated with the onset of a desire to defecate and larger rectal distention volumes produced a stronger SMR response (De Ocampo et al. 2007). A second possibility is that a transient contraction of the puborectalis would allow the individual to sense whether the contents are solid, which would have a weight and mass to press against during the SMR, rather than gas. The consistency of rectal contents has previously been shown to influence defecation (Rao et al. 2006). This concept also explains why the SMR is associated with the desire to defecate. It therefore follows the arousal of a desire to defecate induces an anal canal contraction that is mediated by voluntary, striated muscle, such as the puborectalis.

In this study, we also performed detailed analysis of the spatiotemporal pressure changes along the anal canal. We found that in the resting stage there are two peaks in the anal canal profile; the first peak occurred at 1.6 cm, and this largely corresponds to the IAS (Liu et al. 2006; Raizada and Mittal 2008). The second less prominent inflection of the pressure profile, spanning a distance of approximately 1.6 – 1.8 cm occurred at a distance of 2.4 cm to 4.0 cm from the anal
verge. This most likely corresponded to the location of the posterior sling fibers of the puborectalis muscle. Furthermore, we found that there was a significant difference in the pressure profile between the anterior and posterior region of the anal canal at rest for the same 1.6 cm distance, spanning between 2.4 cm to 4 cm from the verge. Liu et al found the puborectalis to be located 2.5 cm to 4.0 cm from the anal verge using luminal ultrasound pull though technique, which matches exactly with this study’s observation that the posterior vector pressure from 2.4 cm to 4 cm is largely due to the puborectalis muscle (Liu et al. 2006).

The detailed pressure and vector analysis was largely possible because of the superior and 3-D resolution of the HDM system. This imaging modality has the advantage of simultaneously measuring pressure recordings over the entire length of the anal canal and also circumferentially. This degree of fine detail allowed for concurrent assessment and correlation of anatomy with pressure profiles, rather than performing correlations using two different techniques such as manometry and ultrasound (Gantke et al. 1993) or MRI (Cornella et al. 2003; Fenner et al. 1998). Additionally, the closely spaced sensors oriented circumferentially and longitudinally along the length of the anal canal provided a continuous display of pressure without loss of signals, unlike a water perfused, air perfused, or solid state pressure transducers that can only provide pressure measurements at predetermined points or levels. HDM not only allowed for a higher resolution and more clear understanding of the anal canal pressures, but also facilitated the ability to distinguish each component of the high pressure zone. Thus, in this regard our measurements and anatomical delineation corresponds to those described by Mittal and colleagues (Raizada and Mittal 2008) and confirm and extend their findings. HDM has implications for improving our understanding and diagnosis of anorectal disorders.
In conclusion, this study reaffirms the presence of SMR in healthy subjects. Furthermore, through detailed topographic and manometric characterization using HDM, our study demonstrates that the anal contractile response seen during the SMR is primarily induced by the activation and contraction of the puborectalis muscle.
Table 1 – This shows changes in the baseline anal pressure at each 4 mm level from the anal verge and at the anterior and posterior vectors.

<table>
<thead>
<tr>
<th>Length from anal verge (cm)</th>
<th>Anterior (mm Hg)</th>
<th>Posterior (mm Hg)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>12.77</td>
<td>8.8</td>
<td>0.165</td>
</tr>
<tr>
<td>0.4</td>
<td>26.92</td>
<td>21.88</td>
<td>0.381</td>
</tr>
<tr>
<td>0.8</td>
<td>38.98</td>
<td>38.24</td>
<td>0.946</td>
</tr>
<tr>
<td>1.2</td>
<td>60.15</td>
<td>61.01</td>
<td>0.947</td>
</tr>
<tr>
<td>1.6</td>
<td>70.86</td>
<td>79.69</td>
<td>0.482</td>
</tr>
<tr>
<td>2</td>
<td>67.5</td>
<td>81.16</td>
<td>0.221</td>
</tr>
<tr>
<td>2.4</td>
<td>56.02</td>
<td>73.75</td>
<td>0.067</td>
</tr>
<tr>
<td>2.8</td>
<td>46.42</td>
<td>71.77</td>
<td>0.014</td>
</tr>
<tr>
<td>3.2</td>
<td>40.02</td>
<td>68.96</td>
<td>0.01</td>
</tr>
<tr>
<td>3.6</td>
<td>32.84</td>
<td>59.31</td>
<td>0.007</td>
</tr>
<tr>
<td>4</td>
<td>27.59</td>
<td>46.44</td>
<td>0.016</td>
</tr>
<tr>
<td>4.4</td>
<td>17.92</td>
<td>24.19</td>
<td>0.319</td>
</tr>
<tr>
<td>4.8</td>
<td>13.86</td>
<td>14.53</td>
<td>0.904</td>
</tr>
</tbody>
</table>
Table 2 – This shows the individual values for the distance from the anal verge where the puborectalis was best identified with anal ultrasound, and with high definition 3-D topography (HDM).

<table>
<thead>
<tr>
<th>HDM (cm)</th>
<th>Ultrasound (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.2</td>
<td>3.75</td>
</tr>
<tr>
<td>3.2</td>
<td>3.5</td>
</tr>
<tr>
<td>3.2</td>
<td>3</td>
</tr>
<tr>
<td>3.2</td>
<td>3.5</td>
</tr>
<tr>
<td>4.0</td>
<td>4.75</td>
</tr>
<tr>
<td>3.2</td>
<td>2.5</td>
</tr>
<tr>
<td>3.6</td>
<td>3.5</td>
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</tbody>
</table>

Mean = 3.37  Mean = 3.5
**Figure Legends**

**Figure 1** – The top panel shows a continuous recording of the rectal and anal manometric pressure sequences and the lower panel shows 4 separate snapshots or zones of the high resolution topographic pressure profiles taken before, during, and after a SMR response in a healthy subject. Zone A represents the anal and rectal resting pressure profile before rectal balloon distention. Following this, the rectal balloon distention can be seen as a rise in the pressure in the rectal pressure channel. The rectal balloon distention induces a RAIR that is seen as a relaxation of the anal resting pressure throughout all anal channels. Zone B represents the pressure changes just before the onset of the SMR and predominately consists of anal relaxation. In zone C, the subject signals a sensation of desire to defecate and simultaneously, there is a unique anal contractile response that is seen throughout the anal high pressure zone which represents the SMR. The topographic image further shows a transient contraction of the puborectalis muscle. (Arrow) Following the SMR, the RAIR continues to inhibit anal pressures throughout the anal canal and this residual pressure is represented by zone D. All measurements were taken at a specific time point within the A,B,C,D ranges.

**Figure 2** – This shows the circumferential pressure changes along the length of the anal canal before and during the SMR, zone B and C respectively. The arrows outline the location where there was a significant ($p \leq 0.05$) rise in pressure during the SMR. This location (2.4 cm – 3.2 cm from the anal verge) is the same location at which the puborectalis muscle was identified to generate anal canal pressure in Figure 3.

**Figure 3** – Mean percent increase in anal pressure during the SMR from all subjects at every centimeter along the anal canal. The superior portion shows the largest percentage increase in pressure, with the superior 2 centimeters of the anal canal showing a more significant percentage
pressure increase when compared to the more distal sections of the anal canal. The SEM is shown per segment. Data is the mean from all subjects.

**Figure 4** – Mean pressure changes along the length of the anal canal from all subjects in the anterior and posterior vectors during baseline, i.e. resting. The locations of the three anal canal muscular structures that contribute to the anal high pressure zone as described by Mittal and colleagues (Liu et al. 2006; Raizada and Mittal 2008).
References


Figure 3

Distance from Anal Verge (cm)

Percent Pressure Increase during SMR

0 to 1 1 to 2 2 to 3 3 to 4
Figure 4

[Graph showing pressure (mm Hg) versus length (cm) for anterior and posterior baseline conditions.]