ORIGINAL ARTICLE

The anorectal defaecation reflex: a prospective intervention study

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Abstract

Aim: Our hypothesis is that there may be a neural pathway with sensory afferent neurons in the anal canal that leads to rectal contraction to assist defaecation. We aimed to compare rectal motility between healthy participants with or without anal anaesthesia.

Method: This prospective intervention study consisted of two test sessions: a baseline session followed by an identical second session. During each session we performed the anal electrosensitivity test, the rectoanal inhibitory reflex test and rapid phasic barostat distensions. Prior to the second session, participants were randomly assigned to receive either a local anal anaesthetic or a placebo.

Results: We included 23 healthy participants aged 21.1 ± 0.5 years, 13 of whom received an anal anaesthetic and 10 a placebo. All participants showed a transient rectal contraction during the first test session, which decreased significantly after anal anaesthesia (18.6 ml vs. 4.9 ml, $p = 0.019$). The maximum rectal contraction was comparable to the baseline results in the placebo group. Furthermore, the electrosensitivity at the highest centimetre of the anal canal correlated with the maximum rectal contraction ($r = -0.452$, $p = 0.045$).

Conclusion: All healthy study participants display an involuntary, reproducible rectal reflex contraction that appears to be innervated by afferent nerves in the proximal anal canal. The rectal reflex contraction appears to play a role in defaecation and we therefore refer to this phenomenon as the anorectal defaecation reflex. Knowledge of the anorectal defaecation reflex may have consequences for the diagnostics and treatment of constipation.

KEYWORDS
anal canal, constipation, defecation, physiology, rectum

INTRODUCTION

Defaecation requires coordinated contraction and relaxation of muscle layers in the anorectal wall and is controlled by complex neural networks. The process is initiated by an increasing amount of stool entering the rectum [1]. The stool is stored in the rectum until sufficient distension of the rectal wall is created [1,2]. The rectoanal inhibitory reflex (RAIR) is initiated, leading to a transient relaxation of the internal anal sphincter for sampling of the rectal contents [1–4]. Subsequently, intramural mechanoreceptors in the rectal wall are thought to induce rectal contractions, with defaecation being the eventual result [1,5,6].

Recent advances highlight the diversity of sensory afferent neurons in the human colon and proximal rectum [7], while the innervation of the distal rectum and anal canal remains unclear. Remarkably, many highly sensitive nerve endings are found in human anal mucosa that are lacking in the distal rectal mucosa [8]. This finding is in contradiction to the classical theory of defaecation, in which rectal contractions are initiated by receptors located in the rectal wall. Furthermore, the fact that in half of patients constipation cannot be treated effectively [9] also demonstrates the current incomplete understanding of defaecation [1].

Based on these observations and previous studies that showed intramural reflexes upon anal stimulation [10], we hypothesize that there may be a neural pathway with sensory afferent neurons in the anal canal that upon activation leads to rectal contraction to assist defaecation. Our aim was therefore to compare rectal motility between healthy participants with or without anal anaesthesia.

METHOD

Study design

This prospective intervention study was carried out at the University Medical Center Groningen between 2018 and 2019. Healthy individuals aged 18 years or older were invited to undergo two identical sessions of anorectal function tests. To assess their bowel function and medical history, participants were first requested to complete the validated Groningen Defecation and Fecal Continence (DeFeC) questionnaire [11]. Any signs of constipation, faecal incontinence, congenital anorectal abnormalities, neurological dysfunction, trauma or surgery of the gastrointestinal tract or pelvic floor were reasons for exclusion. The eligible participants used sodium phosphate enemas on the evenings before both test sessions.

Test protocol

During the entire test protocol the participants lay in left lateral position with hips and knees flexed.

Rectoanal inhibitory reflex test

The RAIR test was performed to measure the sudden relaxation of the internal anal sphincter upon stimulation of the rectum, known as the RAIR [3,12]. A catheter with multiple distal pressure sensors and a small latex balloon on top was inserted into the anal canal. By means of a hand-held syringe the rectal balloon was rapidly inflated and deflated after 1 s with 10 ml stepwise increasing volumes of air [3,12,13]. After each inflation and deflation, we waited until the anal pressure completely returned to basal anal pressure before we started with a new inflation. The first inflation was always with a volume of 5 ml. The maximum distension was with 100 ml or after the

What does this paper add to the literature?

In all healthy participants we found a reproducible rectal reflex contraction that appears to be innervated by afferent nerves in the proximal anal canal; we refer to this as the anorectal defaecation reflex. The anorectal defaecation reflex probably has consequences for the future diagnostics and treatment of constipation.

maximum RAIR had been recorded. Maximum RAIR was defined as the maximum decrease of anal canal pressure relative to basal anal pressure [3,12,13]. In addition, we recorded the inflation volume at which the first RAIR occurred. We defined the functional length of the anal canal as the distance between the highest pressure sensor that detected a RAIR-related relaxation and the first pressure sensor outside the anal canal, which detected atmospheric pressure.

Anal electrosensitivity test

The anal electrosensitivity test was started by inserting a small catheter with two electrodes to measure the minimally perceived electrosensitivity of every centimetre of the anal canal [14,15]. A current of between 1 and 20 mA was passed through the electrodes until a minimal signal was perceived [15,16]. Subsequently, the smallest perceived electrosensitivity at the highest and lowest centimetre of the individual anal canal was calculated.

Barostat tests

The barostat assembly consists of a catheter with a noncompliant balloon on top which is inserted into the rectum and is subsequently connected to a computer-driven barostat device [14,17]. This device produces stable, predetermined pressures in the rectal balloon while the volume of air that is needed to create and maintain these pressures is recorded [14,17].

First, the rectal balloon was slowly ramp inflated (<1 ml/s) until a rectal pressure of 15 mmHg was reached and maintained for 2 min. This step was performed as a conditioning distension and to ensure complete unfolding of the balloon [18]. After a 2 min recovery time, we started a series of seven rapid, phasic balloon distensions. During each phasic distension, the rectal balloon was inflated rapidly (45 ml/s) up to a preprogrammed pressure. This pressure was kept constant for 90 s, after which the balloon was deflated at the same rate. We allowed a 60 s recovery time between the phasic distensions. The subsequent preprogrammed pressures of the different balloon inflations were 10–15–20–25–30–35–40 mmHg. Each time we asked the participants to rate their level of sensation. This ranged from first sensation, constant sensation, urge sensation to maximum

tolerable volume [12,19]. If a participant indicated maximum tolerable volume or pain the barostat protocol was stopped immediately.

For illustrative purposes, the barostat protocol was carried out once with the barostat balloon placed within an elastic, rubber balloon instead of the rectum.

Definitions

Because the barostat assembly is a closed, noncompliant system, a sudden decrease in rectal volume was considered to be a rectal contraction. Rectal capacity was defined as the maximum intrabag volume during the highest rectal pressure achieved [20]. Additionally, we calculated the relative amplitude as a percentage of individual rectal capacity [20].

Randomization

All participants underwent two test sessions: a baseline test session followed by an identical test session. Prior to the second test session, the participants were randomized to receive either a local anal anaesthetic or a placebo. Lidocaine Vaseline cream 30 mg/g or xylocaine ointment 50 mg/g were used as the anaesthetic substance and Vaseline cream was used as a placebo. Separate sets of randomly labelled containers holding 20 g of cream, stratified by sex, were prepared by an external researcher. Both the investigators who carried out the tests and the participants were blinded to the contents of the containers. We applied the 20 g of cream on a coiled surgical gauze and inserted it into the anal canal. To prevent the rectum from being anaesthetized, we asked the participants to sit up immediately after we had placed the gauze. The surgical gauze was removed after 20 min when maximum effect of both the lidocaine Vaseline cream and the xylocaine ointment was expected.

Measuring equipment

We recorded and analysed all the tests with solar gastrointestinal high-resolution manometry equipment, version 9.3 (Laborie/ Medical Measurement Systems, Enschede, the Netherlands). We performed the RAIR test with a solid-state Laborie/Unisensor K12981-12F catheter (Boston type). For the anal electrosensitivity test, we used a Laborie/Unisensor 8F catheter with two circular electrodes at the distal end. We performed the barostat tests with a 600 ml polyethylene balloon (CT-BP600R, MUI Scientific, Mississauga, Canada) and a double-lumen catheter (CB-CR-001, MUI Scientific, Mississauga, Canada), which were connected to a barostat device (Barostat Distender Series II, G&J Electronics Inc, Toronto, Canada).

Outcome measures

The primary outcome was rectal volume after sudden isobaric distension, as measured by the barostat. Secondary outcomes were the differences in rectal volumes after sudden isobaric distension with versus without anal anaesthesia, and the reproducibility of the measurements.

Statistical analysis

All data were analysed with IBM SPSS Statistics for Windows, version 23.0 (Armonk, NY, USA, IBM Corp.). Categorical variables are shown as numbers (percentages). Continuous variables are presented as mean $\pm$ standard error of the mean (SEM) or median with interquartile range (IQR). For comparisons we used either the t-test for paired data or the Wilcoxon signed rank test. To determine whether there was an association between rectal motility and participant characteristics we performed univariable linear regression analyses, while we used univariable binary regression analyses to study the association between the use of anal anaesthesia and rectal sensation. We used Spearman correlations to assess the relation between rectal motility versus anal electrosensitivity and the RAIR. We defined a p-value of <0.05 as statistically significant.

Ethical approval

Approval was obtained from the Medical Ethical Review Board of the University Medical Center Groningen (approval code METc 2017/245). Each participant gave informed consent prior to the study.

RESULTS

Participant characteristics

We included 23 healthy participants, all of whom completed both test sessions. The mean age of the participants was 21.1 $\pm$ 0.5 years and the median time between the two test sessions was 21.0 days (IQR 14.0–33.0 days). Immediately before the second test session, 13 participants received anal anaesthesia and 10 participants received the placebo (Table 1).

Rectal contraction during the barostat measurements

All participants showed a transient rectal contraction during the first test session seconds after we started inflating the rectal balloon (Figure 1A). The rectal contraction was less pronounced after pretreatment with an anal anaesthetic (Figure 1B) and entirely absent

when we carried out the identical barostat measurements in a rubber balloon (Figure 1C). We assessed rectal volumes at the beginning and at maximum rectal contraction, as well as the maximum volume amplitude of the contraction (Figure 1A).

The amplitude of the maximum rectal contraction gradually decreased along with balloon inflations with increasing rectal pressure (Figure 2A). We observed a similar pattern in participants who we tested with a placebo (Figure 2B). After anal anaesthesia, however, we observed no, or only a small, rectal contraction (Figure 2C). The amplitude of the maximum rectal contraction during the first test session

was 15.3 ± 2.9 ml and occurred at an isobaric inflation with 15.0 mmHg (IQR 15.0–20.0 mmHg) (Table 2). The rectal capacity of all participants as measured during the first test session was 382.9 ± 12.1 ml. The amplitude of the maximum rectal contraction relative to the individual rectal capacity was 3.9% ± 0.8%. Neither the absolute nor the relative amplitude of the maximum rectal contraction were significantly associated with sex, age or body mass index (Table S1 in the Supporting Information)

After anal anaesthesia, the amplitude of the maximum rectal contraction decreased significantly compared with the first test

TABLE 1 Participant characteristics

	Anal anaesthetic (n = 13)	Placebo (n = 10)
Sex, n (%)		
Female	9 (69.2)	9 (90.0)
Male	4 (30.8)	1 (10.0)
Age at test sessions (years), mean ± SEM	22.1 ± 0.7	22.2 ± 0.8
Body mass index (kg/m^2), mean ± SEM	21.8 ± 0.6	21.7 ± 0.6
Time between test sessions (days), median (IQR)	14.0 (10.0–14.0)	33.0 (28.0–85.0)

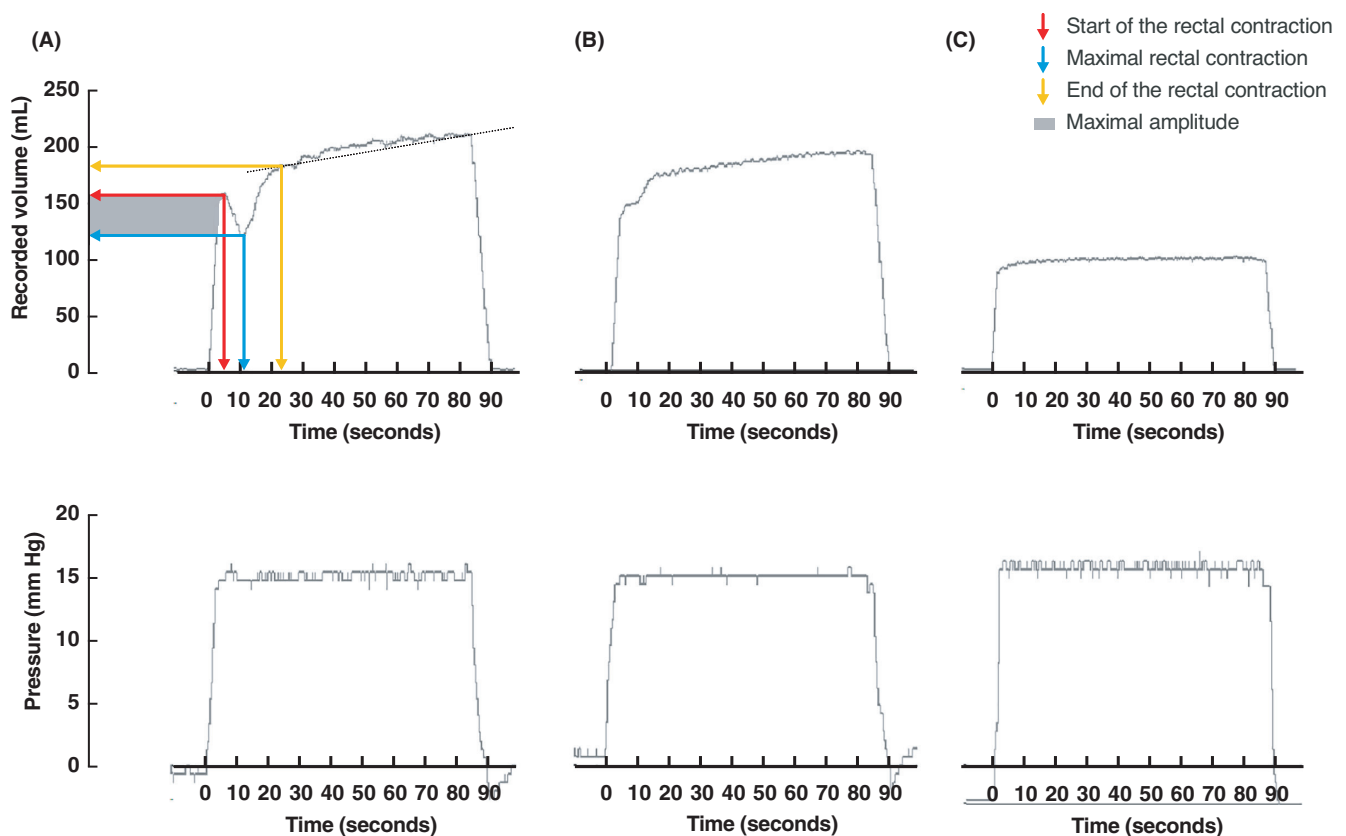*Abbreviations:* SEM, standard error of the mean; IQR, interquartile range.

FIGURE 1 Representative recordings of barostat measurements. (A) Rectal volume and pressure for one of the participants during the first baseline test session. (B) Rectal volume and pressure of this participant after applying an anal anaesthetic during the second test session. (C) The same measurement in a rubber balloon, as a model of passive compliance
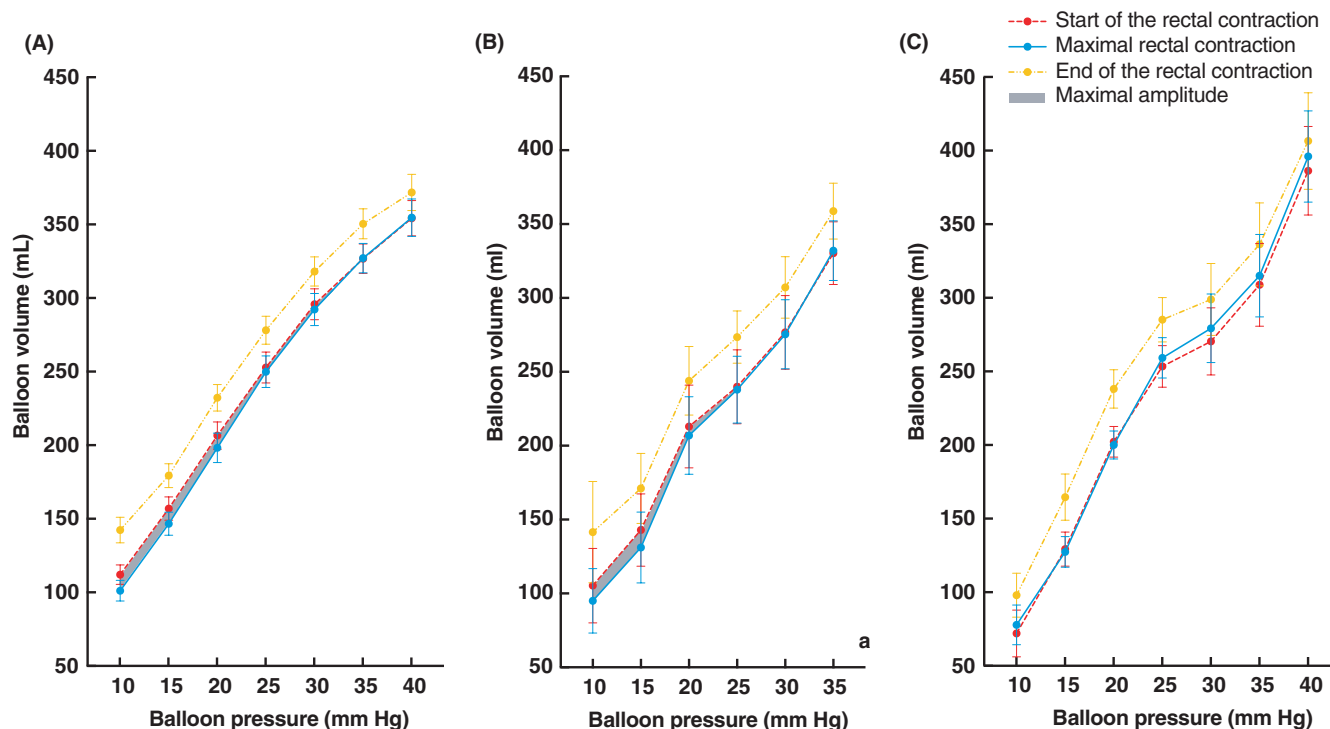
FIGURE 2 Rectal volumes and pressures of the barostat measurements. (A) First baseline test sessions. (B) Second test sessions of participants with a placebo. (C) Second test sessions of participants with anal anaesthesia. Values are reported as mean ± standard error of the mean. [a]The values for the 40 mmHg balloon distensions are missing, because too few participants in this group reached this distension pressure

session without anaesthesia (4.9 ml vs. 18.6 ml, $p = 0.019$; Table 2). The same applied to the relative amplitude of the maximum rectal contraction (1.1% vs. 4.7%, $p = 0.018$). The pressure at which the maximum rectal contraction occurred was significantly higher after anal anaesthesia compared with the first test session without anaesthesia (20.0 mmHg vs. 15.0 mmHg, $p = 0.020$). After the placebo, neither the absolute and relative amplitudes of maximum rectal contraction nor the pressures at which the maximum contraction occurred were significantly different from the first test session.

The onset of the maximum rectal contraction was 5.2 s (IQR 4.4–6.1 s) after balloon distension started. Maximum amplitude was reached after another 4.3 s (IQR 3.9–5.2 s). The total duration of the rectal contraction was 12.1 s (IQR 10.2–13.6 s). There were no significant differences between the timing of the maximum rectal contraction with or without anal anaesthesia (Table 2).

Rectal sensation levels versus rectal contraction

The maximum rectal contraction during the first test session mostly occurred when participants experienced a constant sensation (72.7%) or urge sensation (18.2%). Both the absolute and relative rectal contraction that occurred at constant sensation had a significantly larger amplitude compared with the contraction at urge sensation (both $p < 0.001$, Table 3). We found no significant association

between the use of anal anaesthesia and the minimum pressure required to elicit constant sensation or urge sensation during the second test session (Table S2).

Anal electrosensitivity versus rectal contraction

The minimum perceived anal electrosensitivity was 3.0 mA (IQR 3.0–4.0 mA) for all participants during the first test session. Electrosensitivity at the highest centimetre of the anal canal increased significantly after anal anaesthesia (12.0 vs. 4.0 mA, $p = 0.003$; Table 2).

We found no significant correlation between the electrosensitivity at the highest centimetre of the anal canal and the maximum amplitude of the rectal contraction during the barostat measurement of the first test session (Figure 3A). For the second test session, however, there was a moderate correlation that was significant ($r = -0.452$, $p = 0.045$; Figure 3B). We found no significant correlations for the electrosensitivity at the lowest centimetre of the anal canal (Figure 3A,B).

Rectoanal inhibitory reflex versus rectal contraction

All participants exhibited the RAIR. The first RAIR was elicited with a median volume of 5.0 ml (IQR 5.0–5.0 ml) and relative anal canal

TABLE 2 Comparison of the results of the barostat test, the anal electrosensitivity test and the RAIR test

	Total group (N = 23)	Anal anaesthetic (n = 13)			Placebo (n = 10)		
	Test session 1	Test session 1	Test session 2	p-value	Test session 1	Test session 2	p-value
Barostat measurements							
Amplitude of the maximal rectal contraction (ml), mean ± SEM	15.3 ± 2.9	18.6 ± 4.8	4.9 ± 2.9	0.019*	13.1 ± 4.4	11.9 ± 4.0	0.693
Relative amplitude of the maximal rectal contraction (%), mean ± SEM	3.9 ± 0.8	4.7 ± 1.3	1.1 ± 0.7	0.018*	3.4 ± 1.3	3.2 ± 1.1	0.828
Pressure when maximal rectal contraction occurred (mm Hg), median (IQR)	15.0 (15.0–20.0)	15.0 (15.0–17.5)	20.0 (20.0–22.5)	0.020*	15.0 (15.0–20.0)	15.0 (15.0–25.0)	0.892
Time to start of the rectal contraction (s), median (IQR)	5.2 (4.4–6.1)	4.8 (4.4–5.5)	6.3 (5.1–8.2)	0.050	5.4 (4.9–8.3)	5.9 (3.7–7.3)	0.515
Time from start to maximum of the rectal contraction (s), median (IQR)	4.3 (3.9–5.2)	4.5 (4.1–5.6)	4.0 (2.5–4.3)	0.286	4.2 (3.7–4.6)	4.4 (3.7–4.5)	0.859
Total duration of the rectal contraction (s), median (IQR)	12.1 (10.2–13.6)	13.3 (10.7–16.2)	15.3 (9.3–20.4)	0.722	11.4 (9.8–13.1)	11.5 (10.6–13.5)	0.173
Anal electrosensitivity test							
Electrosensitivity at highest cm of the individual anal canal (mA), median (IQR)	4.0 (3.0–5.5)	4.0 (3.0–5.0)	12.0 (10.0–20.0)	0.003**	4.5 (3.0–6.0)	4.5 (3.0–6.0)	0.394
Electrosensitivity at lowest cm of the individual anal canal (mA), median (IQR)	4.0 (3.0–4.0)	4.0 (3.0–4.0)	4.0 (3.0–5.0)	0.334	4.0 (3.0–4.0)	3.0 (3.0–4.0)	0.046*
RAIR test							
Functional length anal canal (cm), mean ± SEM	3.8 ± 0.1	3.6 ± 0.2	3.8 ± 0.2	0.303	4.0 ± 0.1	3.9 ± 0.1	0.799
Volume when first RAIR occurred (ml), median (IQR)	5.0 (5.0–5.0)	5.0 (5.0–5.0)	5.0 (5.0–5.0)	0.655	5.0 (5.0–5.0)	5.0 (5.0–5.0)	0.414
Relative anal canal relaxation at maximum RAIR (%), mean ± SEM	66.7 ± 3.0	71.2 ± 4.3	63.9 ± 2.7	0.076	59.4 ± 3.6	58.9 ± 4.2	0.799

Abbreviations: IQR, interquartile range; RAIR, rectoanal inhibitory reflex; SEM, standard error of the mean.

* Statistical significance of p < 0.05 (Italic)

** Statistical significance of p < 0.005 (Italic)

TABLE 3 Maximum rectal contractions at constant sensation versus urge sensation

	Constant sensation	Urge sensation	p-value
Maximum amplitude of the rectal contraction (ml), mean $\pm$ SEM	14.9 $\pm$ 2.9	5.8 $\pm$ 2.6	<0.001**
Relative maximum amplitude of the rectal contraction (%), mean $\pm$ SEM	3.8 $\pm$ 0.8	1.5 $\pm$ 0.8	<0.001**

Abbreviation: SEM, standard error of the mean.

** Statistical significance of p < 0.005 (Italic)
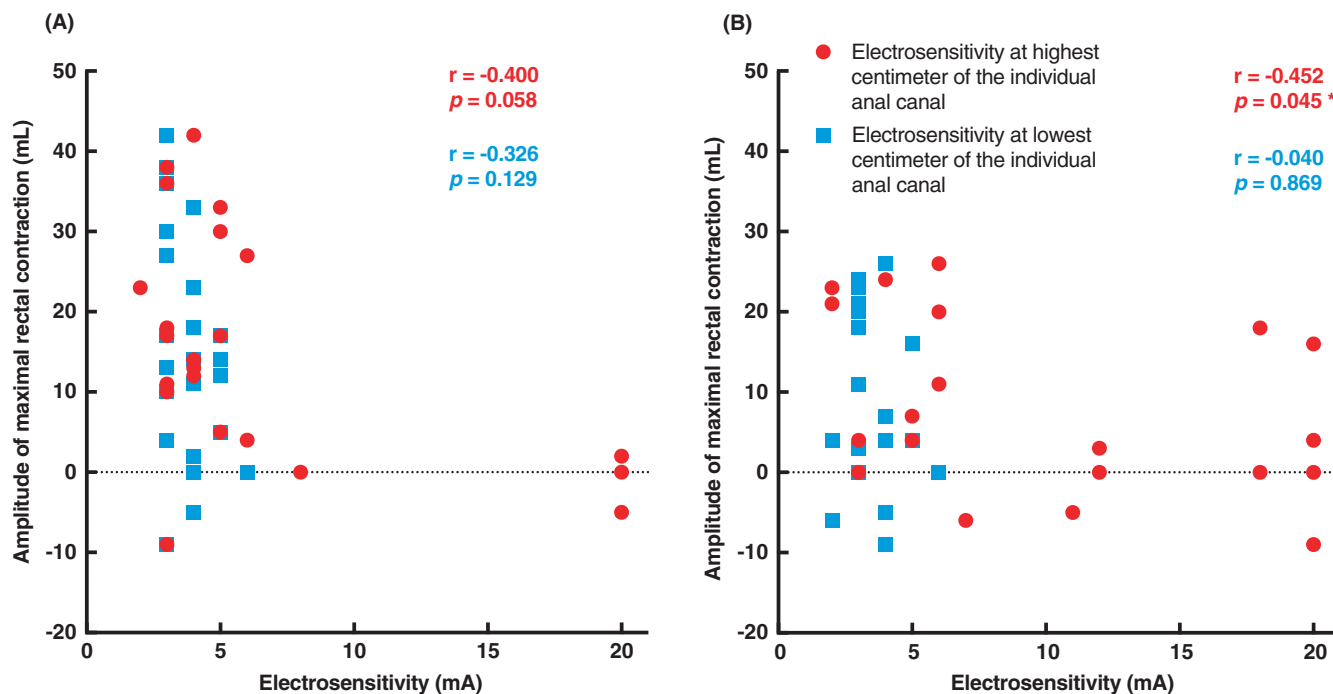
FIGURE 3 Correlation between the maximal rectal contraction and anal electrosensitivity during the first (A) and second test sessions (B)

relaxation at maximum RAIR was 66.7% $\pm$ 3.0% for all participants during the first test session (Table 2). Neither variable was significantly different between the first and second test session for either the participants with a placebo or those with an anal anaesthetic (Table 2). Additionally, we found no correlation between relative anal canal relaxation at maximal RAIR and the maximum amplitude of the rectal contraction during the barostat measurement (Figure S1).

DISCUSSION

The current study showed an involuntary and reproducible rectal contraction in all healthy participants that decreased or even disappeared after anal anaesthesia. This finding points to the presence of a communicating pathway between the anal canal and the rectum.

The rectum is mainly considered as the organ that either transports or stores stool depending on the degree of rectal filling [1,2,19]. Central to this theory is that the rectal wall initiates rectal contractions upon reaching a certain viscoelastic limit, which eventually leads to defaecation [1,2,19]. Many studies have therefore focused

on the viscoelasticity of the rectal wall, usually measured as rectal compliance [5,14,17,20]. However, the sudden rectal contraction described in the current study was not caused by passive compliance of the rectal wall, that is, by elasticity. This is illustrated by the fact the phenomenon was absent when the measurements were carried out in an elastic rubber balloon (Figure 1C), which showed the standard passive compliance curve [5,20].

Because of their involuntary and transient nature, we interpreted the rectal contractions that we observed as reflex movements. Our results demonstrated that neither sex nor age affected the rectal reflex contractions. Furthermore, the amplitude, the eliciting pressure and the onset as well as the duration of the rectal reflex contractions were comparable between the first and second test sessions of the respondents who received the placebo. In other words, the rectal reflex contractions had an excellent reproducibility in healthy people. This strengthens the concept that the rectal reflex contraction is a physiological phenomenon and not an iatrogenic artefact.

Although a rectal reflex contraction upon rapid isobaric stimulation has already been observed [21], its reproducibility and its regulating neural pathway have never been explored. Our finding

of decreased anal electrosensitivity following anal anaesthesia led us to conclude that the sensory input from the anal mucosa and/or submucosa was inhibited successfully. It is important to mention that the minimally perceived anal electrosensitivity of all healthy participants was within the normal range when measured under normal conditions [15,16]. Subsequently, we found that the rectal reflex contraction decreased or disappeared following local anal anaesthesia. This observation supports the presence of afferent nerve endings in the anal mucosa or submucosa. It is also in keeping with previous histochemical studies that reported an as yet unexplained large number of free and organized nerve endings in the anal mucosa with intramural nerves running to the rectum [8].

What remains unclear is the exact location of the afferent nerves within the anal canal. To account for the interindividual variability of the length of the anal canal, we used the highest centimetre of the individual functional anal canal instead of a fixed height. The correlation between anal electrosensitivity measured at the highest centimetre of the individual anal canal and the amplitude of the rectal reflex contraction may indicate that afferent nerves are located primarily in the proximal anal canal. This finding needs to be investigated in detail.

Another reflex mechanism that involves both the rectum and the anal canal is the RAIR. The RAIR is intramurally controlled by the myenteric plexus and additionally mediated by spinal nerves [1,3,13]. The minimum eliciting volume and maximum RAIR remained unaltered following anal anaesthesia. This confirms a previous finding [22] and is suggestive of rectal afferent nerves of the RAIR or deep-lying anal receptors. During the process of defaecation there may be interplay between the RAIR and rectal reflex contraction. The RAIR may increase the exposure of the afferent nerves located in the proximal anal canal to stool, thereby stimulating the rectal reflex contractions. This hypothesis corroborates the previously unexplained observation of massive rectal contractions that accompanied enhanced internal anal sphincter relaxation in patients with a spinal transection [23].

Remarkably, maximal rectal reflex contractions were especially seen at constant sensation levels instead of higher rectal sensation levels. This coincides with the physiological situation in which the daily stool volume is smaller than the total volume of the rectal reservoir [24].

In summary, we propose a new model of the defaecation process: stool enters the rectum, the RAIR leads to a drop in proximal anal canal pressure and a small amount of stool descends into the proximal anal canal [4] stimulating the afferent nerves located there. This leads to a rectal reflex contraction, simultaneously increasing the rectal filling sensation. This eventually results in defaecation by voluntary relaxation of the external anal sphincter and pelvic floor muscles. On account of the anal receptors and the subsequent rectal reflex contraction that assists defaecation, we termed this reflex the anorectal defaecation reflex.

One can imagine that individuals who have a weak or absent anorectal defaecation reflex would present with chronic constipation. Moreover, surgical resection may destroy the neural pathways of the anorectal defaecation reflex and have a serious impact on the postoperative ability to defaecate. This is strengthened by the fact that constipation-associated complaints are common after very low anterior resections [25], but future research is required. On the other hand, an overactive anorectal defaecation reflex may cause faecal incontinence by extremely strong and frequent rectal contractions.

The first limitation of this study is that we used two different anaesthetic substances: lidocaine Vaseline cream and xylocaine ointment. Both substances contain the same active anaesthetic component with a half-life of 1.5–2 h. Differences in anaesthetic effect between the two anaesthetics are therefore highly unlikely. Second, we measured the participants in the left lateral position, while the usual position during defaecation is sitting upright or squatting [26]. We do, however, expect an even stronger anorectal defaecation reflex in the physical upright position, when gravity presses the stool into the proximal anal canal. Third, it could be suggested that the absolute differences in rectal volume at the maximum anorectal defaecation reflex are small. It is, however, likely that the anorectal defaecation reflex is augmented by many other factors, such as voluntary abdominal straining and relaxation of the anal sphincters [1].

CONCLUSION

All healthy study participants display an involuntary, reproducible rectal reflex contraction that appears to be innervated by afferent nerves located in the proximal anal canal. The rectal reflex contraction appears to play a role in defaecation and we therefore refer to this phenomenon as the anorectal defaecation reflex. Knowledge of the anorectal defaecation reflex may have consequences for the diagnostics and treatment of constipation and requires future research.

CONFLICT OF INTEREST
There are no conflicts of interest to disclose.

AUTHOR CONTRIBUTIONS
Substantial contributions to the conception or design of the work, or the acquisition, analysis or interpretation of data for the work: SV, MT and PB. Drafting the work or revising it critically for important intellectual content: SV, MT and PB. Final approval of the version to be published: SV, MT and PB. SV, MT, and PB are accountable for all aspects of the work in ensuring that questions related to the

accuracy or integrity of any part of the work are appropriately investigated and resolved.

ETHICAL APPROVAL

Ethical approval was granted by the Medical Ethical Review Board of the University Medical Center Groningen (approval code METc 2017/245). Each participant gave informed consent prior to the study.

DATA AVAILABILITY STATEMENT

Data are available on application to the corresponding author.

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