Role of the Rectosigmoidal Junction in Fecal Continence

Concept of the Primary Continent Mechanism

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Hypothesis: At mass contraction of the descending colon, the colonic contents stop at the sigmoid colon (SC) and do not pass directly to the rectum. We investigated the hypothesis that a continent mechanism seems to exist at the rectosigmoidal junction (RSJ), preventing the direct passage of stools from the descending colon to the rectum.

Methods: The SC in 16 healthy volunteers (mean±SD age, 38.6±10.2 years; 9 men and 7 women) was distended with an isotonic sodium chloride solution–filled balloon, and the pressure response of the RSJ and the rectum was recorded at rapid and gradual filling of the balloon. The test was repeated after the SC and RSJ were anesthetized separately.

Results: Rapid SC balloon distension with a mean±SD of 52.1±3.6 mL of isotonic sodium chloride solution effected an RSJ pressure increase to a mean±SD of 67.8±18.4 cm H₂O (P<.01) with no rectal pressure response (P>.05). Slow SC filling produced a progressive increase in RSJ pressure but no rectal pressure change. At a mean±SD SC distending volume of 86.3±4.1 mL, the RSJ pressure decreased to 9.6±2.8 (P<.01), and the balloon was dispelled to the rectum; rectal pressure increased (P<.001), and the balloon was expelled to the exterior. The RSJ pressure did not respond to distension of the anesthetized SC.

Conclusions: Contraction of the RSJ at rapid SC distension with big volumes implies a reflex relationship that we call the RSJ guarding reflex. This reflex seems to prevent the descending colon contents from passing directly to the rectum. It is considered the first continent reflex and may serve as an investigative tool in the study of fecal incontinence.

Arch Surg. 2006;141:23-26

Fecal continence (FC) is an intricate mechanism that depends on voluntary and involuntary reflex actions1-4; anorectal sensation also plays an important role in FC.5-8 The pelvic floor muscles, comprising the puborectalis muscle and the external anal sphincter, are the voluntary components of the FC, and the internal anal sphincter is the involuntary one.9-11

The anorectal reflexes are important elements in the mechanism of FC. At rectal distension and the initiation of the rectoanal inhibitory reflex,12-16 other reflexes are evoked: the rectoanal reflex, which affects levator muscle contraction that opens the rectal neck (RN, or anal canal), and the rectopuborectalis reflex, which causes the puborectalis muscle contraction that closes the RN. The RN is supposedly kept closed until impulses reach the conscious level to decide whether or not to defecate. If conditions are opportune, the puborectalis and external anal sphincter (top loop) are voluntarily relaxed and the rectum is evacuated. If the circumstances are not appropriate for defecation, the puborectalis muscle remains contracted, evoking the voluntary anorectal inhibition reflex,17 which leads to reflex rectal relaxation and waning of the desire to defecate.

It is apparent from the aforementioned studies that FC depends on the top loop of the external anal sphincter, which is the conjoined external anal sphincter and the puborectalis muscle.14 This guarding muscle contracts either voluntarily or as a reflex, mediated through the aforementioned reflexes, to maintain FC. Furthermore, another reflex, the straining-puborectalis reflex, maintains FC during the transient increases in intraabdominal pressure caused by coughing or sneezing.18 The reflex causes top loop contraction at straining.

In view of the aforementioned studies, we consider the rectopuborectalis reflex the primary continent reflex. It is apparently the first reflex that reacts to rectal

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distension by closing the RN until a decision for rectal evacuation is made. If conditions are inappropriate for defecation, the voluntary anorectal inhibition reflex is evoked spontaneously, with resulting rectal relaxation. Thus, the rectopuborectalis and voluntary anorectal inhibition reflexes provide a continent mechanism at the level of the RN.

We hypothesized that there is another FC mechanism at the rectosigmoidal junction (RSJ). In normal conditions, the stools passing from the descending colon to the sigmoid colon (SC) stop at the RSJ and do not pass directly to the rectum. We assumed that, as the stools reach and distend the SC, the RSJ undergoes momentary contraction, preventing direct passage of the stools to the rectum. This hypothesis is based on the fact that the descending colon contracts in a mass action with a propelling force that may push the colonic contents not only to the SC but also directly to the rectum.

We investigated the hypothesis that a continent mechanism seems to exist at the RSJ, preventing the passage of stools from the descending colon directly to the rectum. The storage of the stools in the SC for some time appears necessary for absorptive functions.

**METHODS**

**SUBJECTS**

The study comprised 16 healthy volunteers (mean ±SD age, 38.6±10.2 years; age range, 29-54 years; 9 men and 7 women) who had no history of operations or gastrointestinal or anorectal complaints in the past or at the time of enrollment. They had normal bowel habits with a mean ±SD stool frequency of 10.3±1.1 per week (range, 9-12), which matched with the frequency of healthy subjects in our laboratory. Results of physical examination, including neurologic assessment, were normal, and laboratory test results were unremarkable. The subjects provided written consent after being informed about the details of, and their role in, the study. The Review Board and Ethics Committee of the Cairo University Faculty of Medicine approved the study.

**EXPERIMENTAL DESIGN**

The subjects fasted during the night, and the bowel was evacuated by means of defecation or isotonic sodium chloride solution enemas in the morning. The SC was distended by means of a thin polyethylene infinitely compliant balloon, 3 cm in diameter, which was attached to the end of a 10F tube (London Rubber Industries Ltd, London, England). The pressure measured within the balloon was considered representative of the SC pressure. A metallic clip was applied to the distal end of the tube for fluoroscopic control. With the subject lying in the left lateral position and with no medication, the collapsed balloon mounted on the tube was introduced through the anus into the SC by using endoscopic guidance. The tube was connected to a strain gauge pressure transducer (Statham 23BB; Oxnard, Calif). Its location in the SC during the test was controlled fluoroscopically when we noticed a change in the tube length outside the anal orifice.

The SC and rectal pressures were measured separately by means of an isotonic sodium chloride solution–perfused tube. A 10F tube, with multiple side ports at its distal closed end, was introduced through the anus by means of endoscopic control to lie in the SC. A metallic clip was attached to the distal end of the tube for fluoroscopic control. The tube was connected to a pneumohydraulic capillary infusion system (Arndorfer Medical Specialties, Greenendale, Wis), supplied with a pump delivering isotonic sodium chloride solution continuously via the capillary tube at a rate of 0.6 mL/min. The transducer outputs were registered on a rectilinear recorder (model RS-3400; Gould Inc, Cleveland, Ohio). Occlusion of the recording orifice produced a pressure elevation greater than 250 cm H2O.

The manometric tube, lying in the SC, was then gradually withdrawn by using the pull-through technique until it reached the high-pressure zone, which is the RSJ. The RSJ pressure is significantly (P=.02) higher than that of either the SC or the rectum. A second identical manometric tube was introduced through the anus for 8 to 10 cm to lie in the rectum.

Before pressure recording, the gut was allowed 20 minutes to adapt to the balloon in the SC and the two manometric tubes in the RSJ and rectum; the adaptability was evidenced by the reproducibility of the recordings. Carbon dioxide was then infused into the balloon at 2 rates: slowly, at 10 mL/min, and rapidly, at 200 mL/min. The carbon dioxide source was a carbon dioxide cystometer (Heyer-Schulte Corp, Goleta, Calif), which has a self-controlled carbon dioxide system in which disposable carbon dioxide cartridges are used.

**ANESTHETIZATION OF THE SC AND RSJ**

To define whether the response of the RSJ to SC distension was direct or reflex, the response to the anesthetized SC or RSJ was tested. Ten subjects consented to undergo this part of the test, and 6 did not wish to participate. The SC was anesthetized with 10 mL of 2% lidocaine hydrochloride (Xylocaine; Astra, Södertälje, Sweden) added to 10 mL of isotonic sodium chloride solution. Through a colonoscope, the anesthetic solution was injected into the SC wall at multiple sites around the location of the SC balloon. The RSJ and rectal responses to rapid and slow gradual SC balloon distension were recorded 20 minutes after lidocaine injection and 3 hours later when the effect of anesthesia had waned. On another day, the test was repeated with isotonic sodium chloride solution instead of lidocaine.

The response of the anesthetized RSJ to SC balloon distension was tested after 2 or 3 days after isotonic sodium chloride solution testing. The RSJ was anesthetized with 5 mL of 2% lidocaine added to 5 mL of isotonic sodium chloride solution; the solution was injected with a needle through a colonoscope into the RSJ wall at multiple points. The test was repeated on another day with isotonic sodium chloride solution instead of lidocaine.

To ensure reproducibility of the results, these recordings were repeated at least twice, and the mean value was calculated. The results were analyzed statistically by means of the t test, and the values were given as the mean±SD. Differences assumed significance at P<.05.

**RESULTS**

All subjects completed the study and were evaluated with no complication. The mean resting pressure in the SC was 9.8±2.4 cm H2O (range, 8-12 cm H2O), at the RSJ was 27.7±8.6 cm H2O (range, 18-34 cm H2O), and in the rectum was 8.7±1.3 cm H2O (range, 7-10 cm H2O).

At rapid SC distension with a mean of 52.1±3.6 mL (range, 45-55 mL) of isotonic sodium chloride solution, RSJ pressure increased significantly to a mean of
chloride solution–injected SC effected an RSJ pressure maintained after 3 hours. Distension of the isotonic sodium changes similar to those before anesthetization were obtained. When the anesthetic effect had waned, resulted in pressure changes comparable to those before anesthetization. The response of the RSJ to rapid SC distension was reproducible, provided at least 1 minute had passed between 2 distensions; no response was obtained if the distension was repeated at intervals less than 1 minute.

Slow SC balloon filling effected a progressive increase in RSJ pressure, whereas the rectum showed no significant pressure change. Figure 2 shows the pressures in the RSJ and rectum at different volumes of slow SC distension. When the distending volume of the SC reached a mean of 86.3±4.1 mL (range, 80-100 mL), RSJ pressure decreased significantly to a mean of 9.6±2.8 cm H2O (range, 7-12 cm H2O; P<.01), and the balloon was dispelled to the rectum. Rectal pressure was a mean of 55.6±5.8 cm H2O (range, 44-62 cm H2O; P<.001), and the balloon was expelled to the exterior.

Twenty minutes after the SC had been anesthetized, its rapid or slow distension with the aforementioned volumes produced no significant changes (P>.05) in RSJ pressure (Figure 3). Repetition of the test 3 hours later, when the anesthetic effect had waned, resulted in pressure changes comparable to those before anesthetization (P>.05). Likewise, slow or rapid SC distension during RSJ anesthetization induced no significant RSJ pressure changes after 20 minutes of anesthesia (Figure 4), but changes similar to those before anesthetization were obtained after 3 hours. Distension of the isotonic sodium chloride solution–injected SC effected an RSJ pressure response similar to that recorded before injection (P>.05). Furthermore, SC distension while the RSJ was injected with isotonic sodium chloride solution induced the same response as before injection (P>.05). When these tests were repeated at least twice in a subject, the results were reproducible with no significant difference (P>.05).

The present study may shed some light on the possible role of the RSJ in the FC mechanism. Slow SC filling effected a gradual increase in RSJ pressure, which presumably denotes gradual increase in the contractile activity of the RSJ. These results conform with our findings in a previous study.17 The progressive increase in RSJ pressure at slow filling of the SC was mediated through the RSJ tightening reflex.15 This reflex seems to be a guarding mechanism against leakage of SC contents to the rectum before these contents have been treated in the SC. However, an RSJ pressure decline occurred only when SC incremental distension reached a big enough volume that would stimulate the SC mechanoreceptors and result in SC contraction and RSJ relaxation, 2 actions mediated through the rectosigmoid inhibitory reflex.18 The opening of the RSJ allows the SC contents to cross into the rectum. The passage of the balloon to the rectum with the big distending volume initiated the rectoanal inhibitory reflex, effecting balloon expulsion to the exterior.

We demonstrated in earlier studies that the RSJ response to SC distension was mediated through 2 reflexes: the RSJ tightening reflex17 and the RSJ inhibitory reflex.18 The former reflex acts during slow gradual SC filling to prevent leakage of SC contents to the rectum, whereas the latter allows passage of the SC contents to the rectum when these contents have reached a volume big enough to stimulate the SC mechanoreceptors and effect SC contraction.

The present study results may demonstrate another guarding mechanism that acts during passage of the contents of the descending colon to the SC. The descending colon delivers its contents to the SC by means of a mass contraction. This process presumably results in rapid filling of the SC with big volumes that appear to induce an increase in RSJ pressure that supposedly prevents pas-
sage of the SC contents from the descending colon to the rectum directly. It seems necessary for the colonic contents to be stored in the SC for some time to be treated physiologically before being expelled to the exterior.

THE RSJ SPHINCTER

Results of the present and previous studies suggest that the RSJ offers the mechanism that controls the passage of the contents of the descending colon directly to the rectum. The earlier study showed that the RSJ acts not only as a physiological sphincter (ie, the RSJ contracts without the presence of an anatomical sphincter) but also as an anatomical sphincter in the RSJ. Furthermore, it became evident that the RSJ is not just a junction but an entire zone with a mean length of 3.8±0.2 cm (range, 3.5-4.5 cm). Three reflexes act at the RSJ: the RSJ tightening, RSJ inhibitory, and RSJ excitatory reflexes. The RSJ tightening reflex gradually seals the RSJ during progressive filling of the SC. When gradual SC filling reaches the volume that stimulates the SC stretch receptors, the RSJ opens mediated through the RSJ inhibitory reflex. The RSJ excitatory reflex closes the RSJ when the rectum contracts to avoid reflux of rectal contents to the SC.

THE RSJ GUARDING REFLEX

The present findings reveal a hitherto unrecognized relationship between rapid SC distension with big volumes and the RSJ. The pressure response of the RSJ to rapid SC distension affirms our hypothesis of the possible involvement of a reflex, which we call the RSJ guarding reflex. The constancy of this reflex relationship is evidenced by reproducibility and by its absence at separate blocking of the intramural neural plexus of the SC and RSJ, the possible 2 arms of the reflex arc. We consider this reflex to be the primary continence reflex, which appears to be evoked at rapid SC distension with big volumes. The reflex seems to prevent the descending colon contents from passing directly to the rectum without being stored in the SC for further processing. This finding is in contrast to the rectopuborectalis reflex, which is initiated at rapid distension of the rectum and could be considered the second continence reflex. This second continence reflex causes the RN to close at rapid rectal distension with big volumes. A disorder of either of these 2 reflexes is assumed to jeopardize the FC mechanism. Thus, in the presence of a dysfunction of the RSJ guarding reflex, it seems that, at mass contraction, the contents of the descending colon pass directly to the rectum without being retained in the SC; this action may not only change the stool consistency but also impair the rectal reflexes that control defecation and maintain continence.

ROLE OF THE RSJ GUARDING REFLEX AS A DIAGNOSTIC TOOL

A disorder of the RSJ guarding reflex appears to disturb FC. Recording this reflex might be informative in fecal incontinence conditions, particularly of the idiopathic type. Accordingly, this reflex could serve as an investigative tool in the study of fecal control disorder. However, the role of this reflex in fecal incontinence and in constipation needs to be investigated in a further study.

In conclusion, RSJ contraction at rapid SC distension with big volumes implies a reflex relationship that we call the RSJ guarding reflex. This reflex presumably prevents passage of the descending colon contents, at mass contraction, directly to the rectum without being retained in the SC for physiological treatment. We consider the RSJ guarding reflex the primary continence reflex in contrast to the secondary continence reflex, the rectopuborectalis reflex, which contracts at rapid rectal distension closing the RN.

Accepted for Publication: March 22, 2005.
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Acknowledgment: Margot Yehia, BCh, assisted in preparing the manuscript.

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