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REVIEW

Translational Physiology

Novel insights into physiological mechanisms underlying fecal continence

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Abstract

The machinery maintaining fecal continence prevents involuntary loss of stool and is based on the synchronized interplay of multiple voluntary and involuntary mechanisms, dependent on cooperation between motor responses of the musculature of the colon, pelvic floor, and anorectum, and sensory and motor neural pathways. Knowledge of the physiology of fecal continence is key toward understanding the pathophysiology of fecal incontinence. The idea that involuntary contraction of the internal anal sphincter is the primary mechanism of continence and that the external anal sphincter supports continence only by voluntary contraction is outdated. Other mechanisms have come to the forefront, and they have significantly changed viewpoints on the mechanisms of continence and incontinence. For instance, involuntary contractions of the external anal sphincter, the puborectal muscle, and the sphincter of O'Beirne have been proven to play a role in fecal continence. Also, retrograde propagating cyclic motor patterns in the sigmoid and rectum promote retrograde transit to prevent the continuous flow of content into the anal canal. With this review, we aim to give an overview of primary and secondary mechanisms controlling fecal continence and evaluate the strength of evidence.

fecal continence; mechanism; review

INTRODUCTION

Fecal continence is the physiological condition that prevents involuntary loss of bowel contents, including solid feces, liquid stool, and flatus. It prevents the devastating condition of fecal incontinence. Fecal continence is maintained by synchronized actions of the musculatures within the anorectum and pelvic floor (1, 2). The puborectal muscle and the anal sphincters are the most significant contributors to the closing of the anorectal canal (3–5). Other mechanisms, such as the colonic haustral boundaries and the rectosigmoid junction, play an indirect, supportive function. Furthermore, also anal cushions and rectal filling sensations play a role. Indirectly, colonic and rectal contraction patterns, characterized by different frequencies, amplitudes, and propagation directions, may also contribute to fecal continence. Finally, the enteric nervous system controls all the musculatures involved in fecal continence, the extrinsic autonomic nervous system, as well as the sensory and motor nerves from higher centers, which enable both voluntary and involuntary actions that all together, and not as a single mechanism, maintain fecal continence (6). Too often, the focus is only on a single mechanism regulating fecal continence, in particular the closing of the external anal sphincter. Here, we review the broad spectrum of mechanisms that maintain fecal continence on the premise that fecal continence is maintained by a multitude of mechanisms that cooperate and complement each other (Table 1, Fig. 1). We present and discuss primary and secondary (supporting) mechanisms for which there is evidence and discuss other proposed mechanisms for which more research is needed to evaluate their clinical significance.

Primary Mechanisms Involved in Fecal Continence

Feces enter the rectum after being formed in the colon, and then, depending on circumstances, the fecal content can enter the anal canal. The anal canal strongly contributes to fecal continence at different levels along its length through cooperation between pelvic floor muscles and the internal and external anal sphincters. This cooperation is possible due to a variety of specialized sensory nerve endings and receptors located in the anal mucosa and submucosa (46). These nerve endings and receptors forward the stimuli to ganglia and nuclei associated with the spinal cord and central nervous system from where, via efferent

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Table 1. *Summary of all proposed mechanisms involved in fecal continence*

Anatomic Location	Name of the Mechanism	Voluntary/Involuntary	Direct Function[a]	Proved[b]	Function of the Mechanism
Puborectal muscle	Voluntary contraction	Voluntary	Direct	Sufficient	Voluntary contraction decreases the anorectal angle (4, 7).
	Straining-puborectalis reflex	Involuntary	Direct	Sufficient	Maintains the anorectal angle via involuntary contraction of the puborectal muscle at straining (8).
	Puborectal continence reflex	Involuntary	Direct	Sufficient	Maintains the anorectal angle via involuntary contraction of the puborectal muscle in the presence of solid feces (4, 9).
Internal anal sphincter	Tonus	Involuntary	Support	Sufficient	Contributes in 55%–85% to the anal basal pressure during rest (1, 10).
	Rectal anal inhibitory reflex	Involuntary	Support	Sufficient	The temporary relaxation of IAS opens the anal canal briefly and allows its contents to be sampled and to discriminate between the different types of fecal matter in the anal canal (11–13).
	Vagino-anorectal reflex	Involuntary		Borderline	Dilatation of the vaginal wall increases tone of the IAS (14).
External anal sphincter	Resting pressure	Involuntary	Support	Sufficient	Contributes in 35% to the basal pressure during rest (5).
	Squeezing	Voluntary	Direct	Sufficient	Increases the distal anal canal pressure and closes anal canal voluntarily (15).
	Anal-external sphincter continence reflex	Involuntary	Direct	Sufficient	Contracts the EAS by reflex when solid or liquid feces come into contact with anal canal mucosa (3).
	Coughing reflex	Involuntary	Direct	Sufficient	Contracts EAS when coughing or sneezing (16, 17).
	Cutaneoanal reflex	Involuntary	Direct	Sufficient	Contracts the EAS when feces touches the perianal skin (18, 19).
	Cavernoso-anal reflex	Involuntary		Borderline	Coitus first leads to contraction of the cavernosus muscle (20, 21) and subsequently causes the EAS to contract (22).
	Functional anal canal length	Involuntary		Rejected	Measurement of the length in continent and incontinent women has not confirmed that the functional length of the anal canal has a predictive value for fecal (in)continence (23).
	Rectoanal pressure gradient	Involuntary		Rejected	Clinical investigations indicated that the rectoanal pressure gradient during simulated evacuation is not associated with defecatory problems, which undermines the value of RAPG for diagnosing fecal incontinence (24).
Colon	Retrograde cyclic motor pattern	Involuntary	Support	Sufficient	Retrograde propagation of the cyclic motor pattern in the distal colon prevents feces from entering the rectum (25, 26).
	Haustral boundaries	Involuntary	Support	Sufficient	Retain the solid contents while simultaneous pressure waves expel gas and liquid (27).
	Water extraction and increased viscosity	Involuntary	Support	Sufficient	Prevents formation of diarrhea as a risk factor of fecal incontinence (28, 29) and regulates stool viscosity (30).
Rectosigmoid junction	Sphincter of O'Beirne	Involuntary		Borderline	Higher pressure zones in the rectosigmoid junction slow down the movement of contents into the rectum (31).
	Retrograde cyclic motor pattern	Involuntary	Support	Sufficient	Cyclic motor patterns move the contents in the retrograde direction (31).
	Rectosigmoid junction tightening reflex	Involuntary	Support	Sufficient	Progressive distention of sigmoid colon slows down movement of colonic contents into the rectum by gradually increasing pressure in rectosigmoid junction (32).
	Rectosigmoid junction guarding reflex	Involuntary		Borderline	Rapidly increasing pressure in rectosigmoid junction during rapid distention of sigmoid colon (33).
Rectum	Storage	Involuntary	Support	Sufficient	Temporary storage site for feces (34).
	Retrograde propagating periodic rectal motor activity	Involuntary	Direct	Sufficient	A retrograde propagating periodic rectal motor activity serves to keep the rectum empty (35).
	Rectal compliance	Involuntary	Support	Sufficient	Slows down rectal pressure increases (34, 36–40).
	Rectal filling sensation	Involuntary		Borderline	Influences the rectal capacity (34) and senses the presence of feces in the rectum (41, 42).

Continued

Table 1.— *Continued*

Anatomic Location	Name of the Mechanism	Voluntary/Involuntary	Direct Function[a]	Proved[b]	Function of the Mechanism
Anal cushion	Resting pressure	Involuntary	Direct	Sufficient	Contributes to approximately 15% of the anal resting pressure (5, 43).
	Vascular cushions expand at rest	Involuntary	Direct	Sufficient	Expands as a cushion by impeding the drainage of the venous and enlarge their size, aiding fecal continence (44).
Gluteal maximus muscle	Straining-gluteal reflex	Involuntary		Borderline	Contraction of gluteal maximus muscle on increase of intra-abdominal pressure helps to extend and laterally rotate femur, which aids to fecal continence (45).

[a]Direct function: Direct: Prevents fecal incontinence directly. Support: Only functions as part of support; does not help fecal continence directly. [b]Proved: Level of evidence of the mechanism. Sufficient: Well proven. Borderline: proven that this mechanism can aid fecal continence, but the details of how this mechanism works require further investigation. Rejected: not proven or contradictory evidence. EAS, external anal sphincter; IAS, internal anal sphincter; RAPG, rectoanal pressure gradient.

nerve pathways, muscles respond adequately via contraction or relaxation when the continence mechanism is functional. In short, the puborectal muscle and the internal and external anal sphincter provide fecal continence through their contractions. The contractions result in closing of the anal canal, and this process, in healthy subjects, holds feces safely in the rectum until the proper moment for defecation.

Puborectal muscle.

As part of the levator ani muscle, the puborectal muscle prevents the involuntary passage of stool by maintaining angulation of the rectoanal axis by contraction (7). The loop it forms embraces the upper part of the anal canal. In this way, contraction or relaxation of the puborectal muscle influences the rectal pressure and changes the anorectal angulation to prevent involuntary loss of stool (4, 7).

Because the puborectal muscle is a skeletal muscle innervated by somatic nerves, including the pudendal nerve and sacral nerve roots (47), this muscle was thought to contract only voluntarily. However, one is not able to continuously squeeze skeletal muscles or strain for many minutes. Also, during sleep, we are not able to consciously contract these muscles, but still, if healthy, we remain continent. This assumption that the puborectal muscle contracts only voluntarily was first contradicted by Shafik et al. (8), who

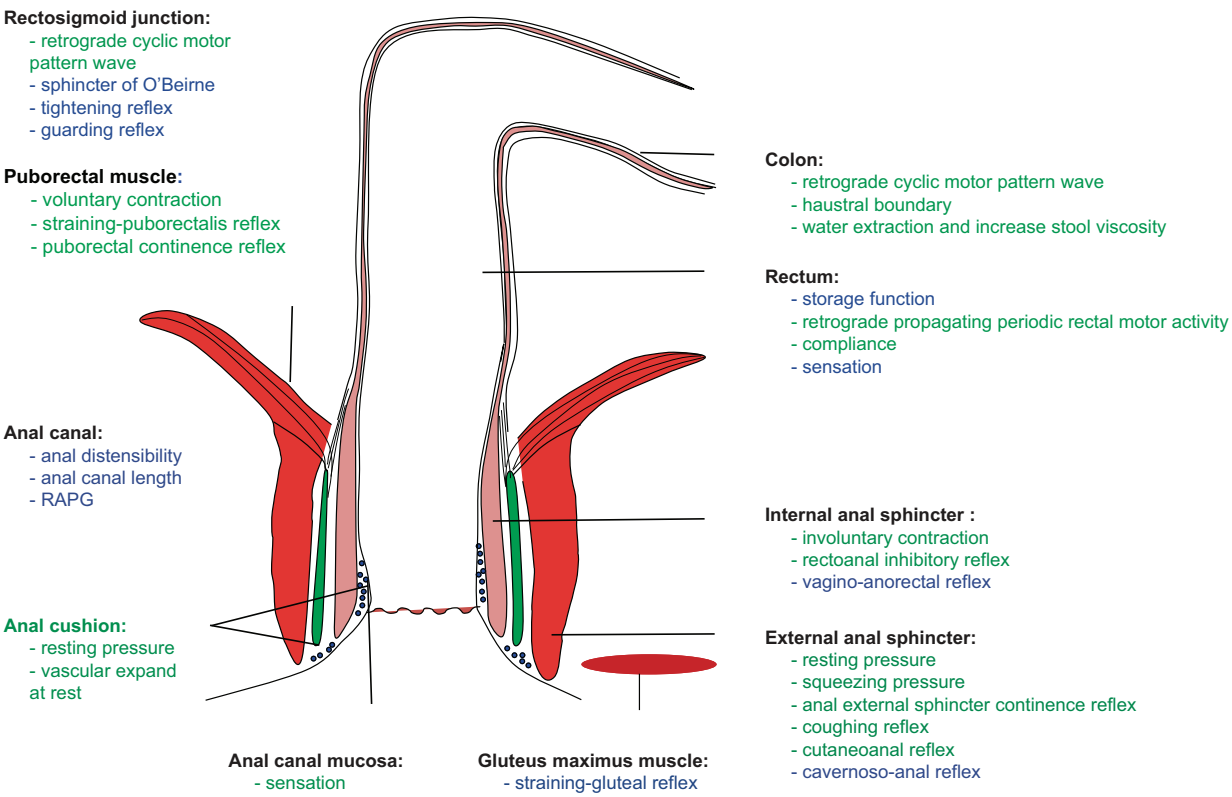

Figure 1. Overview figure of fecal continence mechanisms described in current literature, including the level of evidence provided. Sufficient (green): well proven. Borderline (blue): proven that this mechanism can help fecal continence, but the details of how this mechanism works still require investigation.

found that the puborectal muscle can involuntarily contract via the so-called straining-puborectalis reflex. This reflex can be induced by a sudden increase in intra-abdominal pressure, for instance, during coughing or the Valsalva maneuver.

We further clarified this by showing that the puborectal muscle can contract involuntarily via the puborectal continence reflex on dilatation of the rectum during the balloon retention test (4). We observed that during gradual dilatation of the rectum, the angulation degree of the anal canal axis was gradually decreasing: the puborectal muscle loop was increasingly tightening the rectoanal canal when the pressure in the rectum was increasing. In this way, the puborectal continence reflex prevented involuntary loss of the rectal balloon, even though the subjects had no rapidly increased abdominal pressure. The puborectal continence reflex was found to be activated in the presence of solid stool only, which might explain why it is more difficult to maintain continence for liquid compared with solid stool (4, 9, 48). Of note, the puborectal continence reflex can still function when voluntary contractions are absent, for instance, in patients with pudendal neuropathy, which indicates that the puborectal continence reflex is mediated by nerves different from those involved in the voluntary contractions (49) (Fig. 1).

Internal anal sphincter.

The internal anal sphincter (IAS) maintains basal tone and prevents involuntary loss of stool via its continuous involuntary contraction, sometimes reported as the spasm of the IAS (1, 10). Involvement of the IAS has been estimated to contribute ~55%–85% to the anal basal pressure, and this pressure is often reported as the main determinant of fecal continence (1).

Importantly, the IAS is able to temporary relax, and this action, mediated by the rectoanal inhibitory reflex (RAIR), also contributes to fecal continence; the IAS transiently opens the anal canal and allows rectal content to be sampled by receptors in the upper anal canal (11). The wall of the anal canal is exquisitely sensitive to pain, temperature, and distention or touch (13). The sampling allows for the recognition of the content of the anal canal to discriminate between gas, solid, and liquid stool (11), and this information is forwarded to the cortex (12). Hence, gas can pass with the maintenance of fecal continence.

Finally, the increasing pressure of the IAS on dilatation of the vaginal wall might play a role in fecal continence during sexual intercourse (14). However, no studies are available to prove that the absence of the reflex leads to impaired continence during intercourse (Fig. 1).

External anal sphincter.

The external anal sphincter (EAS) is a striated muscle forming the external and most distal part of the anal sphincter. It has been estimated that the EAS contributes 35% to the basal pressure because of the continuing activity of motor neurons within the pudendal nerve generating tone that keeps the anus closed during rest (5). Because the EAS consists of striated muscle, it has been assumed that this muscle can contract only voluntarily to increase the anal canal pressure (15) and in this way provide fecal continence when a subject feels the urgency to defecate (Fig. 2). But this is not the only mechanism by which the EAS maintains continence (48). Two studies reported that EAS contraction can also be evoked involuntarily. Haynes and Read, using a saline continence test, showed that injection of saline into the rectum causes a synchronized contraction of the rectum, a relaxation of the IAS, and a contraction of the EAS (48). We confirmed this finding for both liquid and solid stool by performing a rectal infusion test, i.e., injection of water into the rectum, and by the balloon retention test, i.e., distention of the rectum using a rectal balloon (3). Because this reflex is activated on

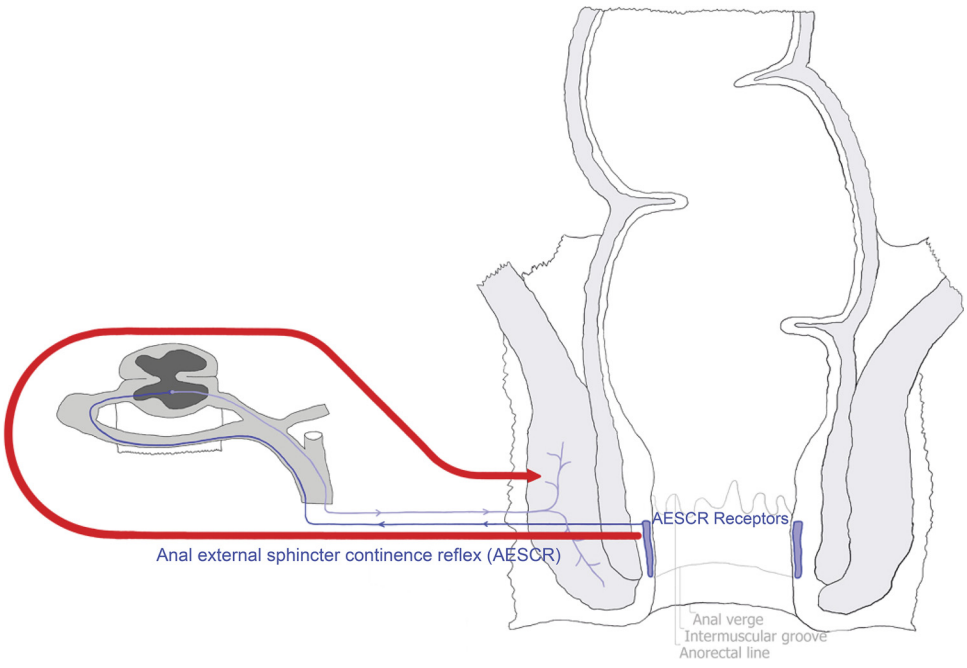

Figure 2. Schematic representation of the autonomous fecal continence mechanism in which the external anal sphincter is involved. Filling the rectal balloon, which mimics stool, makes it descend into the anal canal. There, the balloon activates the receptor of the anal external continence reflex and a signal is given to the external sphincter to contract. This signal is connected directly to the external sphincter by a spinal reflex, without involving the brain. Fecal continence is, therefore, independent of awareness of rectal filling sensation and depends directly on the anal-external sphincter continence reflex. From the study by Broens et al. (3) with permission.

contact of liquid or solid stool with the anal mucosa (Fig. 2), this involuntary contraction of the EAS was called "the anal-external sphincter continence reflex'' (AESCR)(3). Presumably, the receptors triggering the afferent branch of this reflex are contact receptors located at the luminal surface of the distal anal canal. This AESCR reflex does not depend on stimuli from the cortex and is also available when patients have a complete backbone injury, so it behaves as a spinal reflex (3). This mechanism provides fecal continence without the presence of urge to defecate. The exact sensory and motor neuronal circuitries for the AESCR are yet to be elucidated.

When the intra-abdominal pressure rises during coughing or sneezing, the EAS contracts via the so-called cough response of the anal sphincter (16). The anal pressure during a vigorous cough is greater than the voluntary squeeze pressure. The response to coughing can be detected after complete spinal transection, which indicates that the EAS contraction is regulated by a reflex, possibly initiated by stretch receptors located in the EAS and surrounding pelvic floor muscles (17). The response of the anal sphincter to coughing was thought of as a simple spinal reflex because of the increase in abdominal pressure. However, according to Xavier et al. (50), the coughing reflex is an intricate reflex involving complex integrative centers, because EAS activity already increases before external intercostal muscle activity and the rise of the intra-abdominal pressure.

Contraction of the EAS can also be mediated via the cutaneoanal reflex, which is initiated by scratching or pricking the perianal skin (18, 19). This reflex evokes contraction of the EAS when feces touch the anal verge. It is not known whether this reflex contributes to fecal continence because when stool is contacting the perianal skin, it is too late to stay continent. The afferent pathways for this reflex travel in the pudendal nerves, which synapse in the spinal cord and reach the external anal sphincter via the inferior hemorrhoidal nerve (51–53).

Contraction of the cavernosus muscle that is activated during intercourse (20, 21) may evoke sphincter contraction via the "cavernoso-anal reflex" (22). Consequently, anal sphincter contraction during coitus may contribute to the closing of the anal canal, to guard against flatus or feces (22). The cooperation of the anal sphincter with cavernous muscles seems possible because the anaesthetization of the cavernosus muscles results in the absence of the response of the anal sphincters.

Regarding the function of the anal sphincter in fecal continence, additional parameters have been proposed. One is sphincter distensibility, which has been proposed to have a superior value to anal sphincter pressure in terms of contribution to fecal continence (54). This opinion was based on measurements using the EndoFLIP system, which indicated that the distensibility index in fecally continent subjects is higher than in incontinent patients. It is however likely that the introduction of the EndoFLIP into the anal canal of continent subjects will activate multiple receptors present in the mucosa and submucosa of the anal canal, including the receptor of the AESCR. This would explain why the distention index is lower in incontinent patients—their fecal continence mechanisms located "in'' the anal canal are impaired, and therefore the endoFLIP does not activate them or activated them less strongly than in healthy subjects. Therefore, the anal basal pressure and the squeeze pressure, which are typically measured during the EndoFLIP test, are probably increased by the pressure generated by the involuntary contraction of the EAS. Hence, we believe that the distensibility index, although having a predictive value in the diagnosis of fecal (in)continence, does not illustrate a mechanism, but a composite outcome as a result of different variables, including the involuntary contraction of EAS. This hypothesis however should be confirmed with anorectal physiology tests, which can visualize the physiological reaction of the EAS to the EndoFLIP-forced anal distention.

The functional anal canal length used to be considered to play a role in fecal continence. This belief came from the fact that women have a shorter anal canal than men, and the prevalence of fecal incontinence is often higher in women than in men. Measurement of the length in the continent and incontinent women has not confirmed that the functional length of the anal canal has a predictive value for fecal (in)continence (23).

Finally, also the rectoanal pressure gradient (RAPG) has been named as a factor that would predict fecal (in)continence (24). However, clinical investigations indicated that the rectoanal pressure gradient during simulated evacuation is not associated with defecatory problems, which undermines the value of RAPG for diagnosing fecal incontinence (24).

Secondary mechanisms supporting fecal continence.

Colon and rectosigmoid junction. Many studies have been devoted to the role of the colon in the process of defecation, but much less is known about its function in the process of fecal continence. The human colon is different from most animals in that it does not permit the continuous evacuation of stool. In contrast, continence is facilitated by a retrograde propagating cyclic motor pattern in the sigmoid colon, generating a braking mechanism to transit (Fig. 3) (25, 26). Also, haustral boundaries, i.e., sustained or rhythmic pressurizations caused by local circular muscular contractions that form the boundaries of the characteristic colonic haustral compartments, can retard transit and so support continence by promoting stool formation and water absorption within the haustra (27). The intrahaustral pressure waves have mixed propagation directions and occur often simultaneously, again preventing continuous transit compared with their counterpart movements in the small intestine and stomach.

The water absorption process in the colon enables the formation of solid stool and prevents the continued existence of liquid stool that may result in diarrhea, known to be a risk factor for fecal incontinence (28, 29). The water absorption process also regulates stool viscosity; higher stool viscosity can contribute to fecal continence, as proven by Fox et al. (30) who studied rectal filling with stool substitutes having different viscosity. This might be also seen as an indirect mechanism supporting fecal continence.

The rectosigmoid junction is a functional sphincter that is referred to as the sphincter of O'Beirne (31) (Fig. 3). This sphincter is also part of the "braking mechanism,"

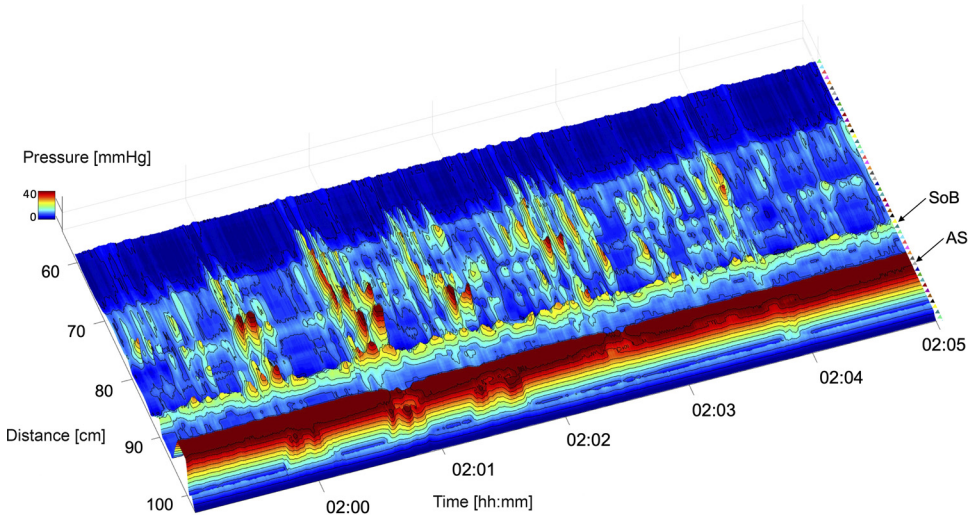

Figure 3. Components contributing to the continence mechanisms recorded in a healthy subject using high-resolution colonic manometry. The anal sphincters (AS); the top of the pressure waves is cut off to make other motor patterns better visible. And the retrograde propagating cyclic motor pattern oral to the sphincter of O'Beirne. Adapted from the study by Chen et al. (31) with permission. SoB, The sphincter of O'Beirne.

contributing to continence by keeping content away from the rectum (31). Recent studies, using high-resolution manometry, showed that in healthy subjects, the sphincter of O'Beirne is visible intermittently as an elevated pressure band of ~27 mmHg at the level of the rectosigmoid junction, compared with an average pressure of 8 mmHg of the haustral boundaries (Fig. 3) (31). The sphincter of O'Beirne is also a site of initiation of the retrograde cyclic motor pattern (31), emphasizing that the retrograde cyclic motor pattern is an important mechanism of continence.

Shafik identified two reflexes associated with the sphincter of O'Beirne when the sphincter contracts in response to a gradual or rapid distention of the sigmoid colon: the rectosigmoid junction tightening reflex (32) and the rectosigmoid junction guarding reflex (33). However, neither the innervation pathway nor mechanisms underlying these reflexes have been characterized, and no further research on these reflexes has been performed.

Rectum.

Many specialists in the field of fecal continence reckon the rectum to be an important element of the total fecal continence machinery because the rectum is a storage site for feces (34). However, patients can maintain fecal continence after total rectal resection (55). We do not deny the contributions of the rectum to fecal continence, but the rectum is not essential. This might be surprising because it is known that fecal continence is impaired in many patients after rectal resection (56, 57). This impairment, however, may result from other conditions. For instance, patients undergoing rectal resection are often diagnosed with rectal cancer. To maintain a safe distal resection margin, a larger region than only "the rectal reservoir'' is resected, sometimes including even the proximal part of the anal canal, which can undoubtedly impair fecal continence (58).

One way that the rectum is involved in fecal continence is the occurrence of retrograde propagating periodic rectal motor activity. This motor activity has been called the "rectal cyclic motor pattern" or the "rectal brake" that may propel rectal contents back into the sigmoid (35).

Rectal compliance reflects the rectal wall distensibility and is composed of both active (relaxation) and passive (elastic) properties (36, 38). Rectal compliance is strongly associated with the rectal volume (37, 39) and is believed to be essential for fecal continence (34). This belief has possibly arisen from the fact that some patients with impaired compliance can experience fecal incontinence (39, 40, 59). However, other factors known to contribute to fecal incontinence are present in these patients, such as an inflammatory process, which can increase intrarectal pressure (39). Then also, the rectal filling sensations, including urge sensation, and consequently the maximum tolerable volume, will be obtained quicker in patients having decreased elasticity of the rectal wall. Such patients will have increased bowel movements, but this does not make them incontinent, as long as they are able to control the moment of defecation. Moreover, patients with inflammatory diseases often experience diarrhea, which is a risk factor for fecal incontinence (39) independent of rectal compliance. Normal rectal compliance may not have an essential function in maintaining fecal continence.

The role of the rectum in fecal continence has also been assumed through the perspective of rectal filling sensation, also referred to as rectal sensitivity. Rectal sensitivity is often associated with multiple factors such as rectal resting pressure, squeezing pressure, and rectal capacity (34). These factors, however, seem to influence the rectal volume at which a certain sensation is felt, but they do not control rectal sensation pressure (42). It is known that the rectal filling sensation is evoked by rectal distention, as proven by experiments involving a balloon inserted into the rectum, driven by changes in rectal pressure (41). However, after rectal resection, patients still experience "rectal" filling sensations (55, 60).

Anal cushions.

It has been estimated that anal cushions contribute to ~15% of the anal resting pressure (5), and consequently to fecal continence (43). The vascular cushions have the ability to expand at rest (44), because increased pressure of

the anal sphincter impedes the venous drainage of anal cushions, enhancing their size, sealing the anus, and aiding in fecal continence in coordination with the IAS and EAS.

Gluteus maximus muscle.

We observed that some incontinent patients contract the gluteus maximus muscle voluntarily, creating the most distal barrier when they feel the presence of feces in the anal canal. According to Shafik et al. (45), gluteal contraction may be regulated by the so-called straining-gluteal reflex, which might be activated during elevated intra-abdominal pressure, and this could assist in anal canal closure through the extended and laterally rotated femur. This, however, is not the primary continence mechanism but rather "the last chance of rescue" for subjects whose fecal continence mechanisms described earlier are altered, i.e., subjects who struggle with fecal incontinence.

As described earlier, fecal continence is regulated by multiple mechanisms that are synchronized by the cooperation of the autonomic and somatic nervous systems with smooth and striated muscles. As there are many different mechanisms underlying fecal continence, it is currently not possible to measure all facets of fecal continence with a single test. This is despite significant advances in the evaluation of anorectal function in the past decade.

SUMMARY

In this review, we presented and evaluated mechanisms contributing to fecal continence. The primary mechanisms providing fecal continence rely on physical barriers generated by the contraction of muscles that close the anal canal and in this way keep the feces in the rectum. These barriers are generated by the synchronized interplay of muscles and their innervation, which enable voluntary and involuntary contractions. There are also important secondary mechanisms that indirectly support fecal continence, such as retrograde motility patterns present along the lower gastrointestinal tract and the water absorption process. This review emphasizes the complexity and supplementary nature of the mechanisms maintaining fecal continence, explaining why relatively few subjects are completely incontinent and also why there are different forms of fecal incontinence. Additional contributors to fecal continence are psychological, behavioral, and cognitive functions that may play a critical role in fecal continence, especially in children (61, 62). The complexity of fecal continence mechanisms has resulted in the development of many different techniques allowing investigation of anorectal functions. Such studies have led to increased knowledge but also confusion, necessitating further research.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

M.T., G.S., and P.B. conceived and designed research; G.S., J.-H.C., J.D.H., and P.B. prepared figures; M.T., G.S., J.-H.C., J.D.H., and P.B. drafted manuscript; M.T., G.S., J.-H.C., and J.D.H. edited and revised manuscript; M.T., G.S., J.-H.C., J.D.H., and P.B. approved final version of manuscript.

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