

2 Anorectal and Pelvic Floor Physiology

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2.1 Introduction

Normal continence and defecation is achieved through a complex interaction between peroral intake, the upper gastrointestinal tract (digestion, secretion and propulsion), and colorectoanal function. Accordingly, continence and defecation can be severely disturbed secondary to abnormal eating patterns or disturbed digestion and propulsion in the small bowel. This is, however, outside the scope of the present chapter. In clinical practice anorectal function cannot be interpreted without considering colonic function, which is therefore included in the following discussion.

The main functions of the colorectum are absorption (water, electrolytes, and short-chain fatty acids), transport, and storage. Absorption occurs mainly in the right colon. The main function of the left colon is storage of stools and, together with the rectum, the pelvic floor, and the anal canal, it is responsible for continence and defecation. The mechanisms underlying continence and defecation are interdependent, and in the majority of patients with functional problems both are affected to a varying degree.

2.2 General Aspects of Colorectal Motility

2.2.1 Colonic Motility

Contractions are either phasic or tonic [16]. Phasic colonic contractions last a few seconds and cause elevated intraluminal pressure [16, 26]. They are single, non-propagating contractions or mass contractions. Single, nonpropagating contractions occur very frequently and include shorter or longer segments of the colon [8, 31]. The main function of these contractions is to mix the colonic luminal content, thereby promoting absorption, or to move the colonic content over a small distance [8]. The mass contractions are high-amplitude propagating contractions that only occur a few times a day, usually originating in the right colon, and span

large parts of the colon, propelling the content distally [4, 8, 17]. Mass contractions are generated mostly in the daytime and especially upon awakening or after meals (the gastrocolic response) [17, 33]. The main function of mass contractions is colonic transport.

The tonic contractions are less well-defined, longer lasting – usually several minutes – and may not be associated with increased luminal pressures [16, 26]. Movements of colonic contents are often not associated with detectable pressure changes and may be due to changes in colonic tone [8]. Two types of tone have been described: a tetanic tone that is generated by fused phasic contractions, and a specific tone that is regulated mainly by chemical processes [16].

2.2.2 Rectal Motility

Rectal motility resembles the colonic pattern, with some colonic mass contractions progressing to the rectum, often initiating defecation [17, 32, 33]. There are, however, some differences. The main difference is the powerful phasic contractions, termed rectal motor complex (RMC), which occurs approximately every 60–120 min [13, 17, 33]. They have a frequency of 3–10 contractions per minute and last for several minutes [33]. They are very similar to phase three of the migrating motor complex within the small bowel. RMCs are often restricted to a single short segment of the rectum, but they may propagate either orally or anally, and they are often associated with contractions of the colon [33] and the anal canal [13]. Accordingly, their main function may be to prevent defecation.

2.3 Generation and Control of Colorectal Motility

Smooth muscle cells within the circular and longitudinal colorectal muscle layers are arranged in bundles that are connected by gap junctions. Bundles fuse at many points and thereby function as a syncytium. The resting membrane potential of the smooth muscle cells undergoes small undulating changes called slow waves. These are generated by the pacemaker cells (interstitial cells of Cajal) [9]. The slow waves do not cause contractions, but they do influence the frequency of spike potentials. During spike potentials, calcium enters the smooth muscle cell causing contraction of the colorectal wall.

Many factors, including neuronal, mechanical, hormonal, and immunological, influence the occurrence of spike potentials and thereby colorectal motility. However, the interactions are not fully understood. Colorectal motility is controlled by the nervous system, hormones, and the immune system. The neuronal system modifies colorectal motility at four levels [41]:

1. The enteric nervous system (ENS)
2. The prevertebral sympathetic ganglia
3. The parasympathetic and sympathetic systems within the brainstem and spinal cord
4. The higher brain centers

2.3.1 The Enteric Nervous System (ENS)

The ENS contains about the same number of neurons as the spinal cord and its function is only vaguely understood. Its sensory neurons are specialized for the detection of mechanical stimuli, temperature, and chemical properties. Through multiple interneurons, such stimuli affect the motor neurons that finally stimulate or inhibit smooth muscle cells. The interneurons also integrate stimuli from other parts of the ENS, the autonomic system, and hormones [41]. The ENS integrates several local reflexes (i.e., the distension reflex where distension registered by mechanoreceptors causes contractions orally and relaxation distal to the site). These reflex patterns can be modified by the autonomic nerve system via interneurons. Many different neurotransmitters either stimulate (e.g., acetylcholine, histamine, serotonin, cholecystokinin, motilin, gastrin) or inhibit (e.g., dopamine, noradrenalin, glucagon, vasoactive intestinal peptide, enkephalin, somatostatin) motility. Moreover, receptors for several of these transmitters (e.g., histamine and serotonin) have been divided into several subgroups, and specific agonist and antagonist have been developed that have, or in future may have, a clinical role.

2.3.2 The Prevertebral Sympathetic Ganglia

The second level of integration and control is within the prevertebral sympathetic ganglia and nerves [41]. These are considered to be the most important mediators of the gastrocolic response mediating colorectal phasic and tonic activity after a meal.

2.3.3 The Parasympathetic and Sympathetic System Within the Brainstem and Spinal Cord

The oral part of the colon approximately to the left flexure receives parasympathetic innervation from the vagal nuclei in the brainstem, while the distal colon and rectum are innervated from parasympathetic neurons in the sacral segment of the spinal cord. Parasympathetic activity stimulates colorectal motility, and if it is lost, colorectal reflex activity becomes severely reduced [22]. A clinical example of major importance is a lesion of the cauda equina that leads to severe defecation problems due to reduced reflex activity and tone in the left colon and rectum [22, 23].

Sympathetic activity reduces colonic phasic activity and tone [6]. The sympathetic fibers to the colorectum originate in segments T9–L2 and reach the mesenteric ganglia through the sympathetic chain, and from there postganglionic fibers reach the bowel. Observational studies suggest that they have only a minor effect on colorectal transport.

2.3.4 Higher Brain Centers

The frontal cortex, the stria terminalis, the amygdala, and the hypothalamus supply information that is integrated in the autonomic system [41]. Little is known about this interaction, but the action of higher brain centers is probably mainly inhibitory. Thus, patients with supraconal spinal cord lesions have increased left colonic and rectal reflex activity and tone [12, 22].

2.3.5 Colorectal Sensibility

Nonconscious sensory information is mediated via parasympathetic afferents in the vagal nerve to the brainstem or through the splanchnic nerve to the sacral spinal cord [41]. Painful stimuli are mediated through sympathetic afferents to the spinal cord [41]. Apart from inflammatory and chemical stimuli, the colon and rectum are only sensitive to stretch [34]. The subjective experience of rectal distension is a feeling of rectal fullness and the urge to defecate, while colonic distension produces pain and colic [15]. The location of rectal stretch receptors is controversial and it has been suggested that they are located outside the rectal wall in the adjacent pelvic structures.

2.3.6 Hormonal- and Immune-System Control of Colorectal Motility

Thyroid hormones stimulate colorectal motility and epinephrine reduces it. Once the immune system in the bowel wall becomes sensitized to a specific antigen, a second exposure will cause release of histamine and other messengers from the mast cells. The histamine will stimulate electrolyte, water, and mucus secretion, and promote strong contractions called power propulsions. These span large distances of the bowel [41], thereby quickly clearing potentially harmful antigens from the lumen.

2.3.7 Colorectal Transit Time

The total and segmental colorectal transit times are highly variable, and colonic transit time may be up to 4 days in asymptomatic subjects. The transit time is usually longer in the right colon than in the left colon and rectum [2], reflecting the fact that the main function of the left colon and rectum is storage.

In healthy subjects stool frequency and consistency correlates better with the rectosigmoid transit time than with the total transit time, while stool volume correlates with total colonic transit time. Stool weight in the Western world is usually 100–150 g/day, while it in rural Uganda its upper limit is as much as 500 g/day. This is most likely a reflection of the much higher intake of dietary fibers in Uganda, mainly bran, that do not undergo fermentation and thereby retain water.

2.4 The Anal Canal

The main functions of the anal canal are to maintain continence and to allow the passage of flatus and feces at an appropriate time and place. In the anal canal the somatic, intrinsic, and autonomic nervous systems are intimately linked.

The internal anal sphincter (IAS) is a direct caudal continuation of the circular muscle layer of the rectum and it consists of smooth muscles cells. Its main function is to contribute to the anal resting pressure [24, 30]. The anal resting pressure decreases with age, and in women also with increasing number of childbirths. The pressure is not constant, but undergoes undulating changes, the so-called slow waves, with low amplitude and a frequency of 10–20/min [37, 40]. Superimposed,

there are contractions and relaxations associated with changes in rectal pressure and diameter. The IAS is not under voluntary control, but is innervated by sympathetic fibers via the splanchnic nerves, and from parasympathetic fibers from the second to fourth sacral segments of the spinal cord. The sympathetic nervous system stimulates the IAS, while the action of parasympathetic nervous system is unclear.

The external anal sphincter (EAS) muscle is composed mainly of slow-twitch striated muscle fibers. It contributes to the resting anal pressure, but its main function is to generate the anal squeeze pressure [24, 30]. The EAS is partly under voluntary control via Onuf's motor nucleus in the spinal cord [29]. The nerves reach the muscle through the pudendal nerve and the perineal branch of the fourth sacral nerve.

Contraction of the puborectalis muscle creates the anorectal angle, and it is supposed that this creates a valve mechanism, which contributes to anal continence [30].

2.4.1 Anal Sensibility

Specific sensory receptions are numerous through the anal canal, and in contrast to the rectum, the anal canal is extremely sensitive to touch, temperature, and movement within its lumen [15, 34].

2.4.2 Rectoanal Reflexes

The pressure within the anal canal is related to the status of the rectum by several reflexes [37]. The anal sampling reflex consists of a regularly occurring, short-lasting relaxation within the upper anal canal, with a simultaneous contraction of the upper rectum and relaxation of the distal rectum [11]. Thus, contents can be moved from the rectum into contact with the mucosa of the upper anal canal and it is assumed that sensory receptors therein can determine the nature of the content (solid stool, liquid stool, or gas). After a short time the anal pressure is normalized and the content is forced back to the rectum [11]. The clinical relevance of this reflex remains to be established.

Another reflex is the rectoanal inhibitory reflex, which mediates relaxation of the IAS during a rectal distension (Fig. 2.1) [10, 22]. It is a local reflex that is conducted through intramural nerve fibers [10]. It is absent in Hirschsprung's disease.

2.5 Defecation

Defecation is normally preceded by colonic mass movements that bring fecal colonic contents into the rectum. The distention of the rectum further stimulates contractions of the colon via a reflex mediated by the ENS and by the parasympathetic defecation reflex [14]. This leads to a phasic contraction and an increase in rectal tone, thereby changing the rectum from a capacious reservoir into a conduit.

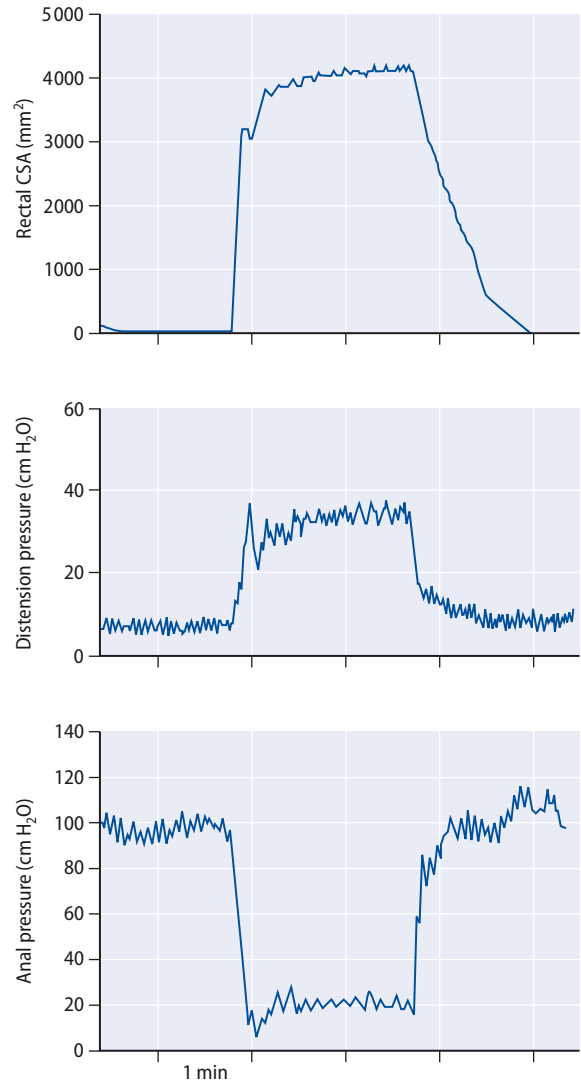


Fig. 2.1 The rectoanal inhibitory reflex. During rectal distension the rectal cross-sectional area (CSA; *top*) and pressure (*middle*) increase while the internal anal sphincter muscle and the anal pressure decrease (*bottom*)

The distention of the rectum stimulates the rectoanal inhibitory reflex, leading to a relaxation of the IAS [10, 22]. In addition there may be a direct coloanal reflex, whereby the IAS is relaxed simultaneously with colonic mass movement. The process is enhanced by increasing the abdominal pressure through a Valsalva maneuver [14]. The puborectalis voluntarily relaxes to increase the angle, and the external sphincter relaxes to open the anal canal. The defecation process can be blocked by voluntary contraction of the EAS and puborectalis, and the defecation reflex will gradually subside and the rectal compliance increase [14]. If the defecation reflex is interrupted due to damage to the reflex between the left colorectum and sacral spinal cord, defecation is severely disturbed, with a prolonged and incomplete evacuation, as seen in patients with cauda equina lesions [23].

2.6 Methods to Study Anal, Rectal, and Colonic Function

A variety of tests has been developed to study different aspects of colorectal function. Some of these tests are used in everyday clinical practice, while others have been used mainly for research in order to increase our understanding of the normal function and the pathophysiology of various diseases [5]. None of the tests are useful outside a clinical setting, which must include a detailed history, objective evaluation, and a diary regarding the patient's bowel function. There is generally a substantial overlap between normal subjects and patients. Furthermore, there are substantial inter- and intraindividual variations for the majority of the tests. The normal range depends upon age and gender. Therefore, the outcome of all tests must be considered in association with the patient's symptoms and interpreted with caution.

2.6.1 Anal Manometry

Various techniques (closed, open perfused, chip transducer, vector manometry) have been developed to measure the anal resting and squeeze pressures in order to estimate the function of the IAS and EAS [27, 36]. The normal range is dependent upon the methods used, age, and gender. The rectoanal inhibitory reflex is present if a balloon dilatation of the rectum leads to a reduction in resting anal pressure [10, 22].

2.6.2 Electrophysiology

These tests have been developed to improve our understanding of the mechanisms underlying sphincter weakness and dysfunction. The pudendal nerve terminal motor latency measures the latency from the stimulation of the pudendal nerve just distal to the ischial spine to the contraction of the EAS (Fig. 2.2). It is an indirect measurement of the conduction velocity of the nerve supplying the EAS. This latency may be prolonged secondary to traction or compression damage to the pudendal nerve [18, 19]. Using either conventional electromyography or single-fiber electromyography, the underlying neuropathic damage with denervation followed by reinnervation can be demonstrated. It is also possible to study the latency in afferent fibers. The electrophysiological test is used primarily in a scientific setting.

2.6.3 Transanal Ultrasonography and MRI

Transanal ultrasonography has been used increasingly to investigate the integrity of the IAS and EAS (Fig. 2.3). The investigation is quick and easy to perform, and the

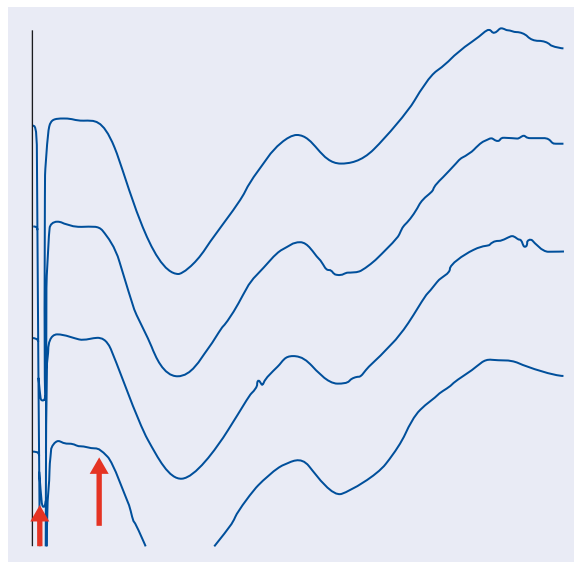


Fig. 2.2 The pudendal nerve terminal motor latency measures the latency from the stimulation of the pudendal nerve just distal to the ischial spine (spike due to electrical stimulation, *short arrow*) to the contraction of the external anal sphincter muscle (*long arrow*)

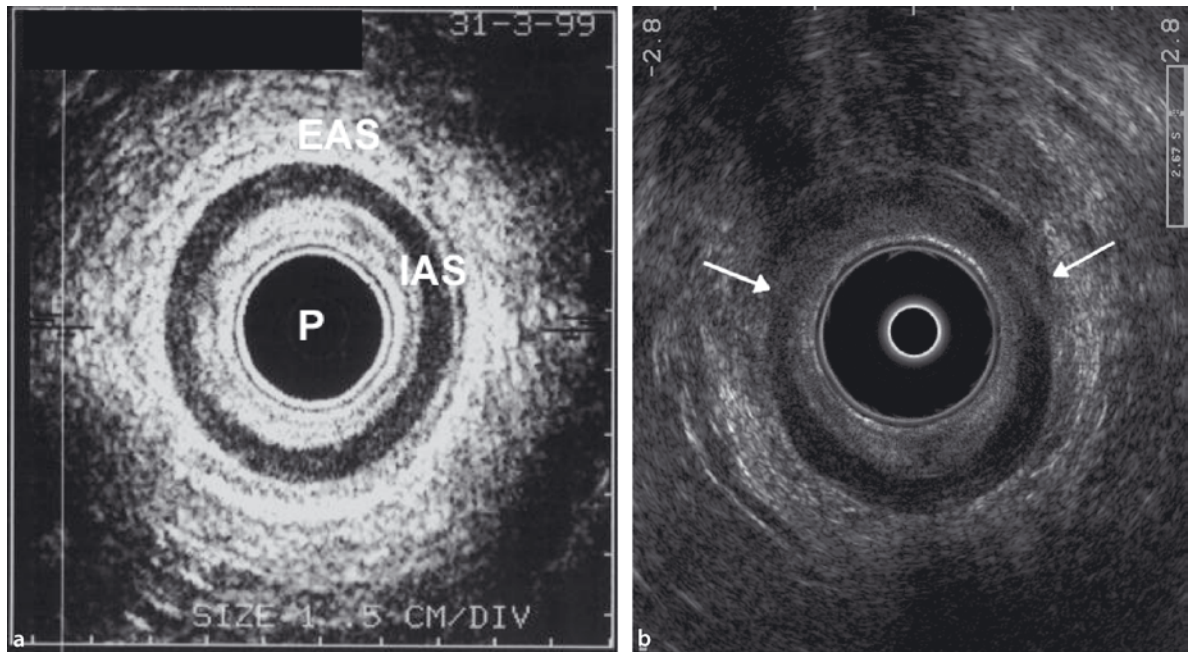


Fig. 2.3a,b Transanal ultrasonography. **a** Intact internal anal sphincter muscle (IAS) and external anal sphincter muscle (EAS). The central dark spot is the probe (P). **b** Anterior lesion (arrows) of the internal and external anal sphincter muscles (bottom)

dimensions of the anal sphincters can be measured simultaneously [3, 38]. A competitive technique is magnetic resonance imaging (MRI) of the sphincters. MRI can also detect atrophy of the EAS muscle and pelvic floor [35]. However, MRI is much more expensive and it is not available at all institutions.

2.6.4 Rectal Capacity, Distensibility and Compliance

Rectal distensibility and capacity can be measured using an air- or water-filled bag [7, 21, 25]. Rectal compliance during distention can be calculated from the pressure-volume curve. During the filling, the thresholds for first perception, desire to defecate, and maximal tolerated capacity is recorded. The test has the same limitations as anal manometry and, due to differences in techniques used, the normal range varies by up to 300% between centers [25].

2.6.5 Anal Sensation

The sensitivity within the anal canal can be measured by determining the threshold for sensation following

electrical stimulation. It is a simple and reproducible test, which has been used primarily in a scientific setting.

2.6.6 Colorectal Transit Time

In clinical practice, total and segmental colonic transit times are most often determined by means of radiopaque markers (Fig. 2.4) [1, 2]. These can be counted either in the stool or on plain abdominal x-rays. In its most simple form, 24 markers are taken as a single dosage followed by one plain x-ray after a fixed time interval – often 5 days. It is thus possible to distinguish patients with prolonged transit time, but the test does not provide any quantitative information about the total or segmental colorectal transit times. The total and segmental transit times can be determined if repeated markers are taken on consecutive days [1], but this increases the cost. The information obtained is only valid if the number of days when markers are taken exceeds the gastrointestinal transit time. Compliance is a problem, since patients have to take markers each day [1].

Scintigraphy can also be used for the determination of transit time. It is superior to radiopaque markers for the determination of gastric and small-bowel transit,

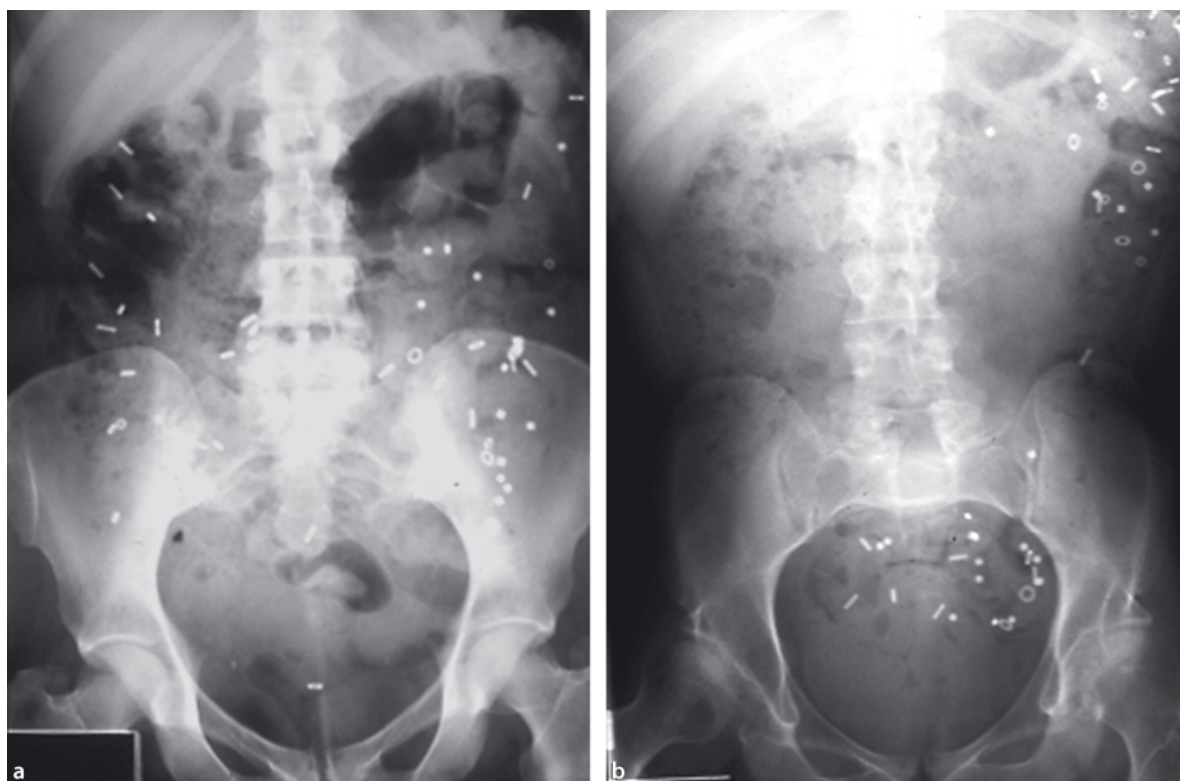


Fig. 2.4a,b Segmental colonic transit time determined by means of radiopaque markers. Generalized slow-transit constipation (a) and left-sided constipation (b)

but it does not provide better information about colorectal transit time. Furthermore, it is more expensive and gives a higher irradiation dose.

Interindividual variations in colonic transit times are large, and it is a concern that many patients with subjective complaints of constipation have a normal transit time. Moreover, the markers could behave differently from the bowel content and the content may not be homogeneous.

2.6.7 Colorectal Emptying

Various tests have been developed to study colorectal changes during defecation. In anal physiology laboratories, a balloon expulsion test has been used. A party balloon is placed in the rectum and inflated with 50 ml of warm water. The electromyographic (EMG) activity of the EAS is recorded with a cutaneous electrode. The patient is placed in a sitting position and asked to expel the balloon. It is recorded whether the patient can expel the balloon; the change in EMG activity is recorded

simultaneously. A typical patient with paradoxical puborectalis contraction during defecation cannot expel the balloon and shows increased EMG activity in the EAS. Difficult evacuation during defecation can also be accessed by means of defecography.

A more physiological test is the defecation scintigraphy test. The patient ingests radioisotopes and when radioactivity can be detected in the left colon and the rectum, the activity is quantified before and after a normal defecation (Fig. 2.5) [23]. The test is expensive and cumbersome and therefore generally used only for research.

2.6.8 Defecography

Defecography is a dynamic imaging of the rectum. The barium mixture is instilled in the rectum, and lateral radiographs are obtained while the subject is at rest, while cuffing, during a Valsalva maneuver, and during evacuation of the barium. Various measurements can be obtained. During defecation in a typical patient with

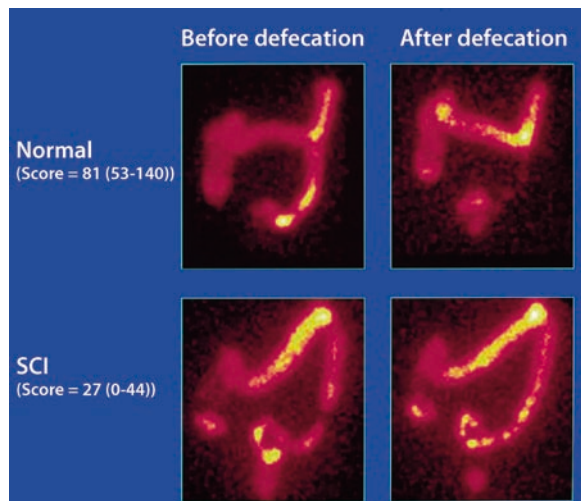


Fig. 2.5 Colorectal scintigraphy after oral intake of isotopes. Scintigraphy in a healthy subject before (*top left*) and after (*top right*) defecation, showing emptying of 81% of the contents of the rectosigmoid. Scintigraphy in a patient with disturbed defecation due to a traumatic lesion of the cauda equina before (*bottom left*) and after (*bottom right*) defecation, showing emptying of 27% of the content of the rectosigmoid

puborectalis paradox, there is a prolonged and incomplete emptying of the rectum, no descent of the pelvic floor, and the anal canal does not open up. Abnormal pelvic floor descent, rectocele, and rectal intussusception can also be recorded (Fig. 2.6). The main problem with both the balloon expulsion test and defecography is that they are unphysiological, without any normal call for defecation. Furthermore, there is a substantial overlap between healthy subjects and patients. Also, rectocele and rectal intussusception can be found in many subjects without any symptoms. Dynamic magnetic resonance defecography has recently been introduced to replace conventional defecography [28], whereby patients are not exposed to radiation, and the bladder, internal genitals, small bowel, and rectal wall are much better delineated. However, it has the same inherited problems as conventional defecography.

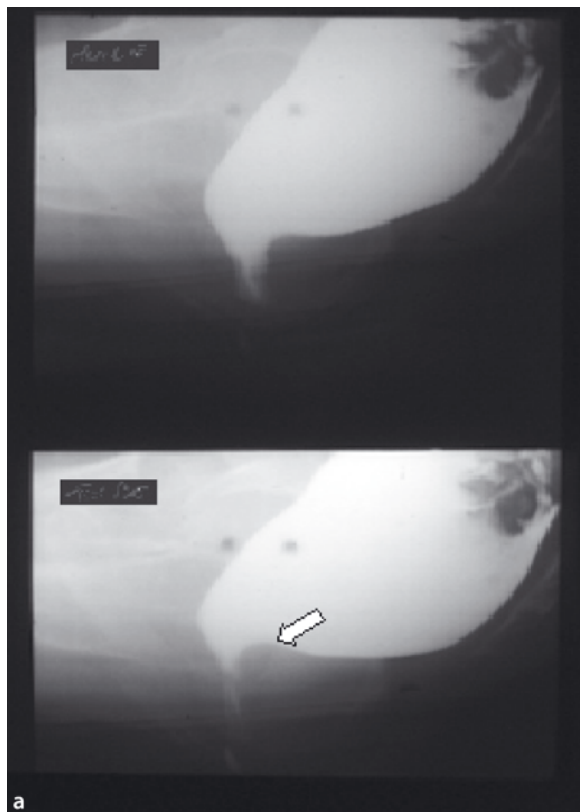
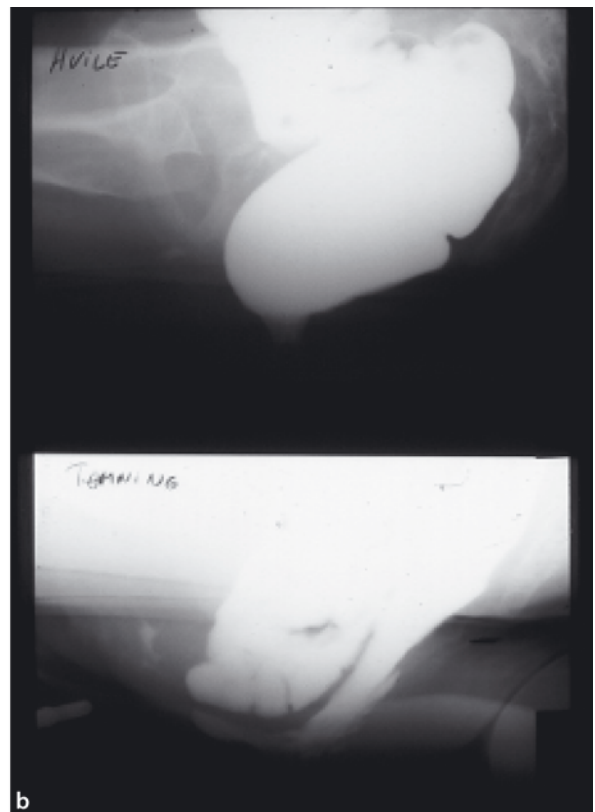


Fig. 2.6a,b Defecography. **a** Patient with obstructed defecation before (*top*) and during (*bottom*) rectal evacuation. Notice the closed anal canal and the increased impression of the pu-



borectal muscle (*arrow*). **b** Patient with enterocele before (*top*) and during (*bottom*) rectal evacuation. Notice the enterocele (small bowel filled with barium) obstructing the rectum

2.7 Anal Continence and Normal Bowel Movements

Principally, the same factors are involved in achieving normal continence and normal bowel function. The key factors causing anal incontinence and constipation/obstructed defecation are:

1. IAS:
 - a. Incontinence: mechanical lesions or atrophy
 - b. Constipation: lack of the rectoanal inhibitory reflex
2. EAS and puborectalis muscle:
 - a. Incontinence: mechanical lesions or atrophy
 - b. Constipation: lack of relaxation during defecation
3. Rectal volumes and threshold:
 - a. Incontinence: low volumes (low compliance) or high threshold
 - b. Constipation: high compliance and high capacity
4. Colorectal motility:
 - a. Incontinence: short transit or incomplete evacuation
 - b. Constipation: slow transit or difficult evacuation

For incontinence and constipation/obstructed defecation, inappropriate function of one or more of these factors might be partly or completely compensated by other factors (i.e., mechanical lesions of the IAS or EAS may cause severe incontinence in some, but hardly any symptoms in others) [31].

2.8 Practical Guidelines for Use of Tests of Incontinence and Constipation

It is difficult to propose strict guidelines since there is no simple relationship between patients' symptoms and test results, and no test can predict the outcome of any intervention. Therefore, the test used in individual laboratories is to a large extent based on tradition. The following tests are, however, used in the great majority of laboratories treating patients with fecal incontinence or constipation.

2.8.1 Fecal Incontinence

1. Transanal ultrasonography to determine the integrity of the IAS and EAS.
2. Anal manometry to provide a baseline measurement of the strength of the IAS and EAS.

3. Rectal thresholds and compliance to provide an indication as to whether the rectum is a low- or highly compliant organ.
4. Defecography in selected cases, particularly if there is clinical suspicion of a deep rectal intussusception or an unproven full-thickness rectal prolapse.

2.8.2 Constipation or Obstructed Defecation

This is a very common problem, and the great majority of patients should only be treated by conservative means [20, 39]. Physiological tests are only indicated in highly selected patients.

1. Total colonic transit time to see if it is prolonged. In selected cases, segmental transit time may be indicated.
2. Anal manometry. The rectoanal inhibitory reflex, which for practical reasons excludes Hirschsprung's disease. Measurements of sphincter strength if colectomy is considered.
3. The balloon expulsion test is a cheap and simple test for puborectalis paradox, but it should be interpreted with caution.
4. Rectal volumes to see if there are increased thresholds and maximum tolerated volume.
5. Defecography to demonstrate difficult evacuation and rectal intussusception – but the pathology must be interpreted with caution.

References

1. Abrahamsson H, Antov S, Bosaeus I (1988) Gastrointestinal and colonic segmental transit time evaluated by a single abdominal x-ray in healthy subjects and constipated patients. *Scand J Gastroenterol* 23:72–80
2. Arhan P, Devroede G, Jehannin B, et al (1981) Segmental colonic transit time. *Dis Colon Rectum* 24:625–629
3. Bartram CI (2005) Functional anorectal imaging. *Abdom Imaging* 30:195–203
4. Bassotti G, Gaburri M, Imbimbo BP, et al (1988) Colonic mass movements in idiopathic chronic constipation. *Gut* 29:1173–1179
5. Bharucha AE (2006) Update of tests of colon and rectal structure and function. *J Clin Gastroenterol* 40:96–103
6. Bharucha AE (1997) Adrenergic modulation. *Am J Physiol* 273:G997–1006
7. Bouchoucha M, Delvaux M, The Working Team (1997) Standardization of barostat procedures for testing smooth muscle tone and sensory thresholds in the gastrointestinal tract. *Dig Dis Sci* 2:223–241

8. Cook IJ, Furukawa Y, Panagopoulos V, et al (2000) Relationships between spatial patterns of colonic pressure and individual movements of content. *Am J Physiol* 278:G329–G341
9. Daniel EE, Berezin I (1992) Interstitial cells of Cajal are they major players in control of gastrointestinal motility. *J Gastrointest Motil* 4:1–24
10. Denny-Brown D, Robertson EG (1935) An investigation of the nervous control of defecation. *Brain* 58:256–310
11. Duthie HL, Bennet RC (1963) The relation of sensation in the anal canal to the functional anal sphincter: a possible factor in anal continence. *Gut* 4:197–182
12. Enck P, Greving I, Klosterhalfen S, et al (2006) Upper and lower gastrointestinal motor and sensory dysfunction after human spinal cord injury. *Prog Brain Res* 152:373–384
13. Ferra A, Pemberton JH, Levin KE, et al (1993) Relationship between anal canal tone and rectal motor activity. *Dis Colon Rectum* 36:337–342
14. Gayton AC, Hall JE (1996) Textbook of Medical Physiology, 9th edn. Saunders, Philadelphia
15. Goligher JC, Hughes ESR (1951) Sensibility of the rectum and colon. Its role the mechanism of anal continence. *Lancet* 1:543–547
16. Gregersen H, Ehrlein H-J (1996) Motility studies in laboratory animals. In: Jensen SL, Gregersen H, Shokouh-Amiri MH, Moody F (eds) *Essentials of Experimental Surgery: Gastroenterology*. Harwood Academic Publishers, New York, pp 17/1–17/39
17. Herbst F, Kamm MA, Morris GP, et al (1997) Gastrointestinal transit and prolonged ambulatory colonic motility in health and faecal incontinence. *Gut* 41:381–389
18. Keighley MRB, Williams NS (1997) *Surgery of the Anus, Rectum and Colon* (2nd edn), vol. 1. Bailliere Tindall, London
19. Kiff ES, Swash M (1984) Normal proximal and delayed distal conduction in the pudendal nerves of patients with idiopathic (neurogenic) faecal incontinence. *J Neurol Neurosurg Psychiatry* 47:820–823
20. Khaikin M, Wexner SD (2006) Treatment strategies in obstructed defecation and fecal incontinence. *World J Gastroenterol* 12:3168–3173
21. Krogh K, Ryhammer AM, Lundby L, et al (2001) Comparison of methods used for measurement of rectal compliance. *Dis Colon Rectum* 44:199–206
22. Krogh K, Mosdal C, Gregersen H, et al (2002) Rectal wall properties in patients with acute and chronic spinal cord lesions. *Dis Colon Rectum*. 45:641–649
23. Krogh K, Olsen N, Christensen P, et al (2003) Colorectal transport during defecation in patients with lesions of the sacral spinal cord. *Neurogastroenterol Motil* 15:25–31
24. Lestar B, Pennickx F, Kerremans R (1989) The composition of anal basal pressure. *Int J Colorectal Dis* 4:118–122
25. Madoff RD, Orrom WJ, Rothenberger DA, et al (1990) Rectal compliance: a critical reappraisal. *Int J Colorectal Dis* 5:37–40
26. Mandrek K, Golenhofen K (1990) Phasic-rhythmical and tonic components in gastrointestinal motility. In: Sperelakis N, Wood JD (eds) *Frontiers in Smooth Muscle Research*. Wiley-Liss, New York, pp 463–481
27. Maslekar S, Gardiner A, Maklin C, et al (2006) Investigation and treatment of faecal incontinence. *Postgrad Med J* 82:363–371
28. Morteale KJ, Fairhurst J (2007) Dynamic MR defecography of the posterior compartment: indications, techniques and MRI features. *Eur J Radiol* 61:462–472
29. Onuf B (1900) On the arrangement and function of the cell groups of the sacral region of the spinal cord in man. *Arch Neurol Psychopathology* 3:387–412
30. Person B, Wexner SD (2005) Advances in surgical treatment of fecal incontinence. *Surg Innov* 12:7–21
31. Rao SSC (2004) Pathophysiology of adult fecal incontinence. *Gastroenterol* 126:S14–S22
32. Rao SSC, Welcher K (1996) Periodic rectal motor activity: the intrinsic colonic gatekeeper. *Am J Gastroenterol* 91:890–897
33. Rao SSC, Sadeghi P, Beaty J, et al (2001) Ambulatory 24-h manometry in healthy humans. *Am J Physiol* 280:G629–G639
34. Rogers J (1992) Rectal and anal sensation. In: Henry M, Swash MM (1992) *Coloproctology and the Pelvic Floor*. Butterworth-Heinemann, Oxford, pp 54–60
35. Schwizer W, Steingoetter A, Fox M (2006) Magnetic resonance imaging for the assessment of gastrointestinal function. *Scand J Gastroenterol* 41:1245–1260
36. Snooks SJ, Barnes PRH, Swash M, et al (1985) Damage to the innervation of the pelvic floor musculature in chronic constipation. *Gastroenterology* 89:977–981
37. Sorensen SM, Gregersen H, Sorensen S, et al (1989) Spontaneous anorectal pressure activity: evidence of internal anal sphincter contractions in response to rectal pressure waves. *Scand J Gastroenterol* 24:115–200
38. Sultan AH, Kamm MA, Talbot IC, et al (1994) Anal sphincter endosonography for identifying external sphincter defects confirmed histologically. *Br J Surg* 81:463–465
39. Wald A (2005) Pathophysiology, diagnosis and current management of chronic constipation. *Nat Clin Pract Gastroenterol Hepatol* 3:90–100
40. Waldron DJ, Kumar D, Hallan RI, et al (1989) Prolonged ambulant assessment of anorectal function in patients with prolapsing hemorrhoids. *Dis Colon Rectum* 32:968–974
41. Wood JD, Alpers DH, Andrews PLR (1999) Fundamentals of neurogastroenterology. *Gut* 45:II6–II16

Self-Assessment Quiz

Question 1

The rectoanal inhibitory reflex:

- a. Mediates relaxation of the internal anal sphincter during rectal distension
- b. Mediates relaxation of the external anal sphincter during rectal distension
- c. Inhibits rectal contractions during anal distension
- d. Inhibits rectal contractions during external anal sphincter contraction
- e. Inhibits rectal secretion during colonic mass movements

Question 2

Pudendal nerve terminal motor latency may be prolonged:

- a. In spinal-cord-injured patients
- b. In irritable bowel syndrome
- c. In Hirschsprung's disease
- d. After traction or compression damage to the pudendal nerve
- e. In various connective-tissue diseases

Question 3

The following method is useful for the detection of rectal intussusception:

- a. Anal manometry
- b. Transanal ultrasonography
- c. Colorectal transit time assessed by radiopaque markers
- d. Defecography
- e. Colorectal scintigraphy

Question 4

Transanal ultrasonography is mainly used for:

- a. Detection of internal or external anal sphincter muscle lesions
- b. Assessment of paradoxical puborectalis contraction
- c. Assessment of rectal emptying after defecation
- d. Computation of anal resting and squeeze pressures
- e. Detection of Hirschsprung's disease

Question 5

Lesions of the cauda equina cause:

- a. Increased rectal tone
- b. Reduced rectal reflex activity and tone
- c. Reduced rectal compliance
- d. Paradoxical puborectalis contraction
- e. No clinically significant changes in anorectal function

1. Answer: a
Comments: The rectoanal inhibitory reflex mediates relaxation of the internal anal sphincter during rectal distension. The reflex is mediated via intramural nerve fibers from the rectum to the internal anal sphincter muscle. It is absent in Hirschsprung's disease.
2. Answer: d
Comments: Observational studies have shown an association between prolonged pudendal nerve terminal motor latency and traction or compression damage to the pudendal nerve, usually due to childbirth.
3. Answer: d
Comments: Rectal intussusception can be detected by defecography; the intussusception is usually seen when the intra-abdominal pressure increases during staining.
4. Answer: a
Comments: Internal or external anal sphincter muscle lesions can usually be detected by transanal ultrasonography. It is important to note, however, that fecal incontinence due to anal sphincter insufficiency can be present in spite of normal findings at transanal ultrasonography. Furthermore, many subjects with anal sphincter lesions detected by transanal ultrasonography do not suffer from fecal incontinence.
5. Answer: b
Comments: Lesions of the cauda equina or the conus medullaris interrupt the reflex arch between the left colon and rectum, and the sacral spinal cord segments 2–4. This interrupts parasympathetic stimuli, thereby reducing rectal (and left colonic) reflex activity and tone (answer b). This usually leads to severely compromised rectosigmoid emptying at defecation.

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