Chapter 7

Faecal Incontinence

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Additional information is available at the end of the chapter

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Abstract

Fecal incontinence (FI) is an extremely common condition, whose true prevalence is difficult to assess. It was defined as the uncontrolled passage of fecal material recurring for >3 months. Fecal incontinence is related to many etiologic factors, but one of the most frequent causes is secondary to pelvic and/or anal and rectal surgery, childbirth-related damage, or other pelvic trauma. Fecal incontinence after surgery may be elicited by many pelvic, rectal, and anal surgical/obstetric procedures, which contribute through different mechanisms to incontinence. After accurate evaluation, the first line approach with medical and behavioral treatments often fails in treating FI. Rehabilitative therapy and less invasive procedures are preferred before performing standard surgical intervention, while invasive procedures are to be discouraged.

Keywords: fecal incontinence, anorectal manometry, endoanal ultrasound, bulking agents, sphincteroplasty

1. Introduction

Fecal incontinence (FI) is an extremely common condition, whose true prevalence is difficult to assess. It was defined as the uncontrolled passage of fecal material recurring for >3 months [1]. The employment of absorbent pads, alimentary restriction, and other restraint principals, up until the last few years, was the only treatment within nonspecialized centers. One of the side effects of dysfunctional sphincter is the inability to hold gas and feces. Incontinence is the result of irregularity of any of the systems, anatomic and neurophysiological structures, together with other systemic diseases, which may have altered intestinal motility and stool consistency as well as diseases that affect superior cerebral capability. Incontinence reduces significantly the patient's quality of life and leads patients to renounce all forms of social daily life. With the passing of the years, changes in pelvic floor structures, connective tissue, smooth

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and striated muscle component lead to an increase in prevalence of the incontinence. The psycho-emotional effects, namely, stress, anguish, tears, anxiety, fatigue, fear of public humiliation, and the sensation to be dirty and smelling are devastating. Limited sexual activity is inevitable as also fear of the anticipated incontinence, fury, abasement, depression, insulation, and frustration. In addition, activity is seriously impaired; for many patients, trivially walking can be a time to deal with unpleasant inconveniences and that results in avoiding every daily activities. After accurate evaluation, the first line approach with medical and behavioral treatments often fails in treating FI. Rehabilitative therapy and less invasive procedures are preferred before performing standard surgical intervention, while invasive procedures are to be discouraged.

2. Fecal incontinence following surgery or trauma

Fecal incontinence is related to many etiologic factors, but one of the most frequent causes is secondary to pelvic and/or anal and rectal surgery, childbirth-related damage or other pelvic trauma. Fecal incontinence after surgery may be elicited by many pelvic, rectal, and anal surgical/obstetric procedures, which contribute through different mechanisms to incontinence. Until quite recently, the surgical management of fecal incontinence has, for almost 25 years, focused on the repair of injuries sustained to the anal sphincter complex. The advent of anal ultrasound in the late 1980s allows better case selection through improved recognition and characterization of anterior obstetric and anal defects amenable to repair, previously relied only to clinical examination [2].

3. Clinical assessment

Medical, surgical and obstetric history is the first attempt to evaluate a patient suffering from fecal incontinence. Information regarding stool form according to the Bristol scale [1, 3], number of bowel movements/week, pathological pre-existing conditions and procedures of former rectal and/or anal surgery were collected from previous outpatient charts. The surgical operations were categorized according to type of surgical procedure, operation date, underlying disease, time elapsed between surgical procedure and outpatient observation. Obstetric trauma has the highest incidence, both following a surgical procedure (episiotomy) and a sequel of traumatic delivery (prolonged labor, a disproportion between the size of the baby and the pelvis, breech delivery, forceps use). The Sultan classification [4] has been adopted by the International Consultation on Incontinence [5] and the Royal of obstetricians and gynecologists. Different scores are used to classify fecal incontinence: Fecal Incontinence Severity Index (FISI) score [6], Fecal incontinence quality of life (FIQL) [7], Rapid assessment fecal incontinence score (RAFIS) [8], Gastrointestinal Quality of Life Index (GIQLI) [9], and Wexner and Jorge scale [10]. Rectal examination remains the first attempt to evaluate the condition of anal sphincter in patients with FI, showing good sensitivity and poor specificity in discerning small from severe global anal sphincter defects. Moreover, digital rectal examination had

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fair sensitivity and poor specificity in grading external anal sphincter defects, and its best accuracy was on complete external anal sphincter lesions. Anal resting and squeeze tone were correlated to anal pressures [11].

4. Diagnosis

4.1. Functional tests

4.1.1. Anorectal manometry

A useful functional test for accurate definition of anal canal pressures, recto-anal inhibitory reflex, rectal sensations and rectal compliance is anorectal-manometry (ARM). Fecal incontinence is evaluated using all these parameters. Different methods to acquire data regarding ARM in FI management are employed: classic water perfusion manometry, solid-state manometry [12], and high-resolution manometry [13, 14]. Solid-state probe with strain gauge transducers or water-perfused probes is actually in use. A central lumen ends in a 4-cm long, compliant balloon attached to the extremity of the catheter, at 4 cm from the distal recording point [12, 15, 16]. The water perfused manometry systems use pneumohydraulic pumps ensuring a rate of 0.2–0.4 ml/min with a pressure head of 10 psi. The sonde is introduced leaving pressure sensors and balloon in the rectum, and a rest time of 5 min is necessary to allow the anal basal tone to become again to its starting value. The resting anal pressure may be measured with a station pull-through technique withdrawing the probe step-by-step 0.5 cm at the time to record the pressure profile of the anal canal. If the patient is not totally at relax, there could be a stronger concurrence of the striated muscles and higher pressures certainly would be registered. Therefore, in order to obtain steady values, it would be preferable to place the sensors inside the anal canal and register the pressure at rest for longer period (5–15 min).

The basal tone is given by tonic activities of the internal anal sphincter (IAS) and of the external anal sphincter (EAS). Works on the effect of the IAS myotomy, general anesthesia [17], and of the block of pudendal nerve [18] on the anal pressures show that 75–85% of the rest pressure derives from the IAS and the remaining part from the EAS. To assess the strength and length of the voluntary contraction, the gauge sonde is positioned in the anal canal with registration bores in the high-pressure area, and the patient is asked to squeeze (≥2 attempts). The average of the highest pressures recorded at any site in the anal canal is used to calculate the maximum squeeze pressure [12, 16]. The duration of squeezing can be intended as the period in which the squeeze pressure is maintained above 50% of the maximum value or as the interval between the beginning of the pressure increasing in the anal canal and the pressure curve returns to the starting values. The squeeze maneuver assesses the function and the voluntary control of the EAS. Recent works, using manometry and 3D ultrasound of the anal canal [19], reported the association between increased pressure along the entire anal canal and the contraction of puborectalis muscle and EAS. These data suggest that the puborectalis muscle takes parts to the squeezing time in the proximal portion of the anal canal, while the EAS in the distal portion and the maximal values are registered where the puborectalis overlaps EAS. Involuntary contraction of the EAS occurs during blunt change in abdominal pressure: this is a multisynaptic sacral reflex that prevents anal incontinence in such conditions and that is voluntarily inhibited during defecation. To check the integrity of this reflex, the patient is invited to make a cough: this reflex response results in the anal sphincter pressure to rise above that of the rectum. The cough reflex is evaluated as the highest positive difference between the increases of the anal pressure in comparison with the increase of the rectal pressure in two attempts [12]. Picking at the perianal skin is possible to attend at a contraction of EAS: this is the anocutaneous reflex. Anal pressures vary by age and sex even though there exists a sizable overlapping in values [15, 16, 20, 21]. Measured pressures tend to be higher when you run a quick pull-through [10]. Barostat test would be the ideal method for assessing the sensorial and viscoelastic characteristics of the rectum, even though it is still use in routinely valuation because of its cost. During ARM, a balloon is inflated in the rectum with increasing volumes of air to keep some information over the viscoelastic properties and sensory functions. This procedure is less accurate than the detailed barostat test, but it is considered sufficient to keep information about clinical rectal properties. The rectal balloon is intermittently air inflated. Each inflation is performed every 30–60 s and implies a 10 ml volume gain up to 200 ml of air or the beginning of pain/discomfort. The rectal balloon is completely deflated after each step [12, 16]. Rectum responds to filling with visceral relaxation for comfortably storing feces until the voluntary defecation: this accommodation is described by the rectal compliance, which is a volume/pressure curve. The pressure in the balloon during the distension seems to be connected to the internal rectal pressure, and its recording is used to assess the rectal compliance. The balloon inflation causes a fast increasing of the balloon pressure, followed by a decline to a steady-state value as the rectum fits to the increased volume. The rectal steady state is calculated as the difference between the recorded pressure and the pressure obtained during the inflation of the balloon. High compliance rates means that the rectum has excessive relaxation and then results in poor increased pressure in its lumen, conversely low compliance rates describe a poor adaptation of the rectum to volume gains and then results in high intra-rectal pressures. The distension of the rectum by increasing volumes is aimed also to evaluate rectal sensibility. The patient is invited to refer the feelings in the rectum. Usually, three steps of sensations are identified: (1) feeling of fullness or distention, (2) steady bid to evacuate, and (3) maximum tolerable volume that can be associated with painful sensation [12, 16]. Large size and/or high compliant rectum requires large volume to evoke the call to evacuate; in noncompliant rectum, small volume can induce urgency [22]. However, balloon inflections used to assess the rectal compliance and sensibility seem to have some limits: in addition to the fact, the rectum is an open cavity, the results depend on both technique and operator. Recorded data may vary according to the size and the nature of the balloon and the method of air inflation and its velocity. The rectal sensibility can also be modified by the discomfort caused by the maneuver and/or by the use of clyster before the test. Distention of the rectum causes a transitory decrement of the basal anal pressure due to the relaxation of the IAS: this characteristic is known as the rectoanal inhibitory reflex (RAIR). It is a reflex mediated via the myenteric plexus. It can be also identified as a “sampling mechanism” to discriminate the rectal content: flatus and consistence of feces. The characteristics of the RAIR depend on some technical aspects: the rectum should be empty, megarectum needs

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high volume to reach the correct level of distension in order to evocate the reflex, the pressure drop can be obscured by the EAS contraction, or it cannot be evident at all when the resting pressure is very low. The nondetectability of RAIR is diagnostic for Hirschsprung's disease with a sensitivity of 91% and specificity of 94%. In constipated patients, it is convenient to evaluate the defecatory maneuver. The patient is invited to defecate to evaluate sphincter responses during the maneuver, while the rectal balloon can be inflated with air or water. Normal pattern should result in increasing the internal rectal pressure, which is synergic with the decrease in the internal anal pressure. Some patients may have anal pressure increment during straining for the paradoxical contraction of the EAS or lack of anal relaxation: in both cases, there is an obstacle to evacuate [12]. In a third pattern of dyssynergic defecation, the internal rectal pressure is lower than anal pressure [12]. The maneuver can be altered by outpatient condition (position of the patient, lack of privacy); actually, it is poorly reproducible and altered patterns are recorded also in asymptomatic subjects. Anorectal manometry is suggested in the workup for fecal incontinence because it provides objective assessment of the anal sphincter function. The manometric parameters usually considered are resting pressure, squeeze pressure, rectal compliance, and rectal sensibility.

4.1.2. Electromyography

Another functional evaluation is the anal neurophysiological testing. External anal sphincter electromyography, motor-evoked potentials, somatosensory evoked potentials, and sacral anal reflex latency measurement are currently available to evaluate neurogenic anorectal disorders. Pudendal nerve supplies voluntary control of external anal sphincter, diagnosis of its damage may be reached with neurophysiological tests and a prolongation of electrical impulse across it may have several impacts on evacuative control [23]. Finally, anal electromyography may be helpful in patients with obstructed defecation. It senses electrical activity during rest, squeeze, and strain and can be useful to identify patients with paradoxical contraction of the puborectalis, sign of pelvic floor dyssynergia.

5. Imaging

5.1. Endoanal ultrasound

Endoanal ultrasound allows to visualize the complete ring of the internal anal sphincter (IAS), the complete ring of the superficial external anal sphincter (EAS) (concentric band of mixed echogenicity) and the thickness of both anal sphincters in the middle level of the anal canal [24, 25]. A discontinuity of the muscle, with an area of mixed echogenicity due to replacement of muscle cells by fibrous tissue, was read as a defect of IAS or EAS. The sphincter defect was measured in degrees. Diffuse thinning and/or replacement of muscle fibers by fat defined external anal sphincter atrophy. Internal anal sphincter atrophy was identified as diffuse thinning of the sphincter. Correct acquaintance of the normal ultrasonographic anatomy of the anal canal is necessary to identify abnormalities. In particular, EAUS is currently the gold standard exam for internal and external anal sphincter defects identification in fecal incontinence. Most recent studies showed 80–100% sensitivity in identifying sphincter's damages. Endosonographic scanning is performed with a 7 or 10 MHz rotating endoprobe, providing a 360° axial view of the anal canal; three-dimensional endosonography allows multiplanar imaging of the anal sphincters. Color or power Doppler imaging technology can also be used with endosonography [26]. The examination is performed with the patient placed in the left lateral position, in the knee-chest position. A digital anorectal examination must be performed before the insertion of the probe to visualize the lesion's size and location and the status of the anal sphincters [27, 28]. At the moment of the insertion of the probe into the anal canal, it is usually put in line with standard orientation, in which the anterior anatomical structures are at the uppermost or 12 o'clock side of the image, the patient's left side is at 3 o'clock, the patient's posterior side is at 6 o'clock, and the patient's right side is at 9 o'clock. To cover the entire length of the anorectal canal, the probe should be introduced up to 8–9 cm, approximately at the level of peritoneal reflection. Then, the probe is slowly retracted, and images are obtained at different levels through the anal canal [29].

The anatomy of the anal sphincter complex is based on four layers: (1) sub-epithelial tissues (medium reflectivity), (2) IAS – hypoechoic (low reflectivity), (3) the longitudinal-muscle layer (variable reflectivity), (4) EAS – hyperechoic (variable reflectivity).

The anal canal is conventionally divided into three different parts: (1) upper anal canal which is a hyperechoic horseshoe sling of the puborectalis muscle posteriorly and loss of the EAS in the midline anteriorly; (2) middle anal canal level which is the completion of the EAS ring anteriorly in combination with the maximum IAS thickness (IAS is seen as a hypoechoic ring-like structure); (3) the lower anal canal level is defined as that immediately caudal to the termination of the IAS and comprises the subcutaneous EAS. Moreover, the IAS gets slightly thicker, and the EAS gets thinner with increasing age. It is important to consider some snares in the reading of EAS images. The female anterior EAS anatomically situated below the level of the puborectalis sling may be wrongly interpreted as an anterior EAS damage. The anococcygeal ligament, with its triangular hypoechoic structure on the axial images posteriorly, should not be confused with a sphincter defect. On endoanal sonography, atrophic or degenerative sphincters are seen as thin and poorly defined and often with heterogeneous increased echogenicity. Increased echogenicity on endoanal sonography has been shown histologically to be correlated with replacement of smooth muscle by fibrous tissue. It will be important to recognize abnormal thinning and physiologic age-related EAS differences. This should be a problem in the EAS because the EAS muscle is also thinner at older ages, and it may be difficult to distinguish sufficiently between atrophy and age-related changes [30]. The IAS is very clearly seen on endoanal sonography, and it is easier to appreciate atrophy and small tears of this sphincter. Moreover, 3D endoanal sonography facilitates sagittal and coronal reconstruction of the anal canal, resulting in better delineation of the normal anatomy and defects of the anal sphincter. On endoanal sonography, scar tissue seems to be a mixed echogenic area. A discontinuity of the anal sphincters results as a localized defect. The localized defect of the IAS appears as hyperechoic break, and EAS tears appear as relatively hypoechoic areas. An injury of the EAS due to vaginal delivery is typically anterior, usually in the right anterolateral side. In contrast, an isolated IAS injury almost never follows childbirth and indicates a primary traumatic cause from within the anal canal, most commonly surgical interventions. During

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the exam should be carefully registered the number, the circumferential and the longitudinal extent of all defects. Anal sphincter injury related to vaginal delivery in female is the most common cause of fecal incontinence due to direct or indirect anal sphincter muscles damage or sphincter innervation. They are identified in 0.6–9.0% of vaginal deliveries where mediolateral episiotomy is performed, but the detection in EAUS is much higher. Typically, anal sphincter defects childbirth related are ultrasonically seen as an interruption of the normal U-shaped, upper—or round—middle, and low aspect of the EAS characterized by a "loss" of the right anterolateral arm of the EAS (from 9 o'clock to 11 o'clock) because the episiotomy is usually realized, by a right-hander gynecologist, in this anterolateral area. If the EAUS after vaginal delivery will detect an important anal sphincter defect—even little symptomatic—it should be immediately repaired to decrease the risk of severe FI. Anorectal surgery represents the second most frequent cause of sphincter lesion. In all cases of anorectal surgery, especially in cases of a procedure with more risk of postsurgical sphincter lesions—fistula in ano—and even for simple anorectal surgery, as in the case of a patient who is multiparous or with previous perianal surgery or trauma, EAUS is mandatory to be performed to evaluate the status of the sphincter complex to avoid surgical procedures that could make unmask a preexisting sphincter incontinence in the postoperative period. When a hemorrhoidectomy or a prolapsectomy is performed, respectively, the removal of hemorrhoidal cushions or the postoperative fecal urgency that can occur after prolapsectomy can improve a light or subclinical fecal incontinence [31]. Moreover, an anal sphincterotomy performed for anal fissure could became the final act responsible for moving a previous asymptomatic sphincter lesion in a clinical fecal incontinence. In particular, EAUS in patients surgically treated for anal fissure might show insufficient sphincterotomy and sphincterial thickening because of the persistence of fissure and anal pain or, on the contrary, demonstrate an excessive sphincterotomy with temporary or permanent incontinence. In case of a surgery for fistula in ano, an endoanal US should be performed in the preoperative for mapping the abscess and identify the fistula, but also to exclude the presence of a previous internal, external, or both sphincter lesions. This relief could change the quality and outcome of surgery [32]. The preoperative EAUS is, however, recommended for every fistula because the fistula that was preoperatively judged easy might demonstrate as complex at surgery or at the postoperative follow-up with potential even dramatic sphincter consequences [33, 34]. In recurrent or complex fistula in ano, 3D EAUS (sometimes with hydrogen peroxide) proved to be more accurate than 2D for detecting difficult (hidden) primary or secondary tracks and internal openings [35]. It should be underlined, however, that an endoanal US realized in an operated patient could offer important difficulties of interpretation of the US images for the presence of fibrosclerotic tissue and/or artifacts.

5.2. Magnetic resonance imaging in fecal incontinence

An imaging assessment is mandatory in evaluating anal incontinence as sphincter tears are overlooked at clinical examination. Loss of ring continuity and loss of homogeneous intensity signal of the sphincters are pathologic detections due to damage of muscle fibers. Breakage of the normal shape with hypointense alteration of the muscle fibers is pathognomonic of the presence of scar tissue. It is visible as hypointense tissue because of its content in fibrous tissue, more hypointense than the normal external sphincter, distorting the normal multilayered architecture of the sphincter muscle. Fat replacement is also a finding consistent with atrophy even if the sphincter thickness is preserved [36]. Many studies have demonstrated that despite its lower local spatial resolution, external phase-array MR imaging is comparable to endoanal magnetic resonance imaging (MRI) for the depiction of anal sphincter defects and EAS atrophy. Endoanal exam have limits as the discomfort in the introduction of the coil, the reduced quality of images due to artifacts from movement and interface between the probe and the rectum, and probable stretching of the sphincter muscles caused by the probe itself with consequent underestimation of their thickness [37, 38]. External phased-array MRI imaging has demonstrated atrophy of EAS in most women complaining fecal incontinence and an IAS defect in women with previous obstetric trauma. Besides external phased-array MRI can identify other defects of pelvic floor structures. Puborectalis muscle atrophy, shown as an abnormal thinning, has also been found in a considerable number of fecal incontinent patients. Pubo-rectalis and levator-ani muscle defects are relatively common in women with severe fecal incontinence, however usually associated to sphincter injury than solitary defects. MRI has demonstrated that levator-ani muscle injury is present in lot of women with EAS injuries who delivered vaginally, and those women patients were frequently suffering from fecal incontinence. Anorectal angle (ARA) change during squeeze was lower in subjects with fecal incontinence who had a history of a third- or fourth degree perineal tear, indicating a lower function of the pubo-rectalis muscle [39]. It is important to assess the sphincter integrity with MRI because patients who have only a focal defect may benefit from surgical repair [40], or in the case of incontinence and rectal prolapse, patients may achieve restoration of continence after rectopexy [41]. In the selection of patients for anal sphincter repair, both endoanal MR and endoanal sonography are sensitive tools for pre-operatory assessment, but endoanal MRI is capable of depicting EAS atrophy, with a sensitivity of 81% and a positive predictive value of 89% compared to surgical findings, which is associated with a poor outcome of anterior anal sphincter repair [42, 43]. Patients with external sphincter atrophy at a preoperatory assessment have worse outcome after repair, while those with normal external thickness show a better postsurgical outcome [44]. MRI defecography (dynamic imaging of the pelvic floor) has also been evaluated in selecting surgical options in anal incontinence and MRI defecography reveals various pelvic floor abnormalities including rectal descent, cystocele, enterocele, rectocele, and rectal invagination. Moreover, 50% of patients revealed ARA changes <10% between rest and squeezing and rest and defecation, indicating a dysfunction of puborectalis sling mechanism. Experience of radiologist is important in evaluating the sphincters complex, being the interobserver agreement stronger if both internal and external sphincters are intact or damaged [45]. MRI provides an accurate depiction of anal sphincter complex and pelvic floor anatomy with evaluation of muscle integrity and being a valuable tool to assess functional abnormalities of the pelvic floor as well. Either endoanal or external MRI can be used to evaluate muscle integrity with comparable results. External phased-array MRI provides information on pelvic floor muscle, while dynamic imaging is an additional tool to assess if pelvic floor prolapsed (bladder, uterine, or rectal) is associated. These information are of main diagnostic importance in evaluating fecal incontinence and aid treatment decision-making.

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6. Treatment

The first step of therapy is conservative approaches, especially in patients with mild symptomatology, as dietary changing, medical therapy, muscles exercises (*exercises of Kegel*), biofeedback, and nonsurgical electrical nerve stimulation. Dietary changing avoiding caffeine, fruits rich on fibers, spicy foods, alcohol, and milky products (in patients with lactose intolerance) may help, but evidence on these restrictions is lacking. Smoking and sedentary lifestyle can be associated with FI [46].

6.1. Kinesitherapy

Kinesitherapy is a rehabilitative method that alleviates symptoms and obtains the greatest possible recovery of lost or altered function, by utilizing therapeutic exercise and movement of the body or part of it to treat disease [47]. Pelviperineal kinesitherapy or pelvic floor muscle training (PFMT) occupies a very important position in rehabilitation in the fields of urogynecology and proctology. PFMT typically consists of verbally guided instruction in pelvic floor and sphincter contractions (*Kegel contractions*). Anal sphincter exercises are performed to strengthen the puborectalis muscle, which is continuous with the external anal sphincter [48]. The technique is to consider pelvic floor like an elevator that can stop at different floors as it can go up and down. Other reported methods include working on coordination of anal sphincter activity and working to isolate a contraction of the anal sphincter. Some therapists use to place an hand externally or to guide the patient with finger placed vaginally or rectally for the correct exercise techniques, but most would argue that this constitutes a form of low-tech biofeedback training. Biofeedback therapy (BFB) includes many different types of training exercises for the pelvic floor. Biofeedback is defined as the process of gaining greater awareness of many physiological functions, primarily using instruments that provide information on the activity of those same systems, with a goal of being able to manipulate them at will. For pelvic floor rehabilitation purposes, the most common type of biofeedback is EMG that based on biofeedback therapy, which was introduced in 1979 [49]. Biofeedback is performed using visual, auditory, or verbal feedback techniques with an anorectal manometer or electromyographic sonde inserted into the rectum to display pressure modifications [50]. Data are registered either through surface electrodes or via the use of intravaginal or intrarectal sensors. Electrical stimulation is another modality that has been used for the rehabilitative therapy of FI. The target of electrical stimulation is to improve the strength and/or endurance of striated muscles contraction with the objective being typically identified with the external anal sphincter. Another goal can be to allow patients with decreased kinesthetic awareness to become more cognizant of where their pelvic floor muscles are in space and what it feels like when the muscles and sphincter are contracting. Electrical stimulation can be delivered to the pelvic floor and anal sphincter in many different forms, including via surface electrodes or intrarectal probes and with many different stimulation parameters and treatment protocols. All forms of electrical stimulation are often used with PFMT or biofeedback training, although stimulation can be used alone as rehabilitative treatment. Transcutaneous and percutaneous tibial nerve stimulations have been tried in patients with FI. In a randomized, double-blind, sham- controlled trial, 144 patients were randomly assigned to receive either active or sham stimulations for 3 months. No statistically significant difference was shown between real and sham transcutaneous electrical nerve stimulation (TENS) in terms of an improvement in the number of FI/urgency episodes per week [51].

6.2. Sacral nerve stimulation

Anorectal and pelvic floor innervation derive from the autonomic and the somatic nervous systems. Motor innervation of the levator-ani muscle and pubo-rectalis sling starts in the sacral nerve roots (S2–S5) [52–54]. The EAS is innervated by a branch of the pudendal nerve, the inferior rectal nerve [52]. Autonomic innervation is sympathetic and parasympathetic. Parasympathetic innervation is through the pelvic plexus, derived from the sacral nerves (S2–S4) [52]. Anal and distal rectal sensory innervation is mainly through the pudendal nerve [55]. Electrical stimulation of this dual innervation seems to excite both systems and causes both direct and reflex-mediated responses in the fecal continence mechanism [56, 57]. The real mechanism of action of SNS in the treatment of bowel and urinary dysfunctions is not cleared yet. The great part of the studies was conducted in patients affected by urinary dysfunctions. For infants, who have not yet achieved voluntary control, a critical level of bladder distention is required to stimulate the voiding reflex. This sensory input, on reaching the pontine micturition center, simultaneously allows for a coordinated detrusor contraction and concomitant urethral relaxation. Gaining voluntary control, the voiding reflex becomes a complex process mediated at a higher level in the cerebral cortex. Voluntary voiding is a result of inhibition of the sympathetic system and activation of the sacral parasympathetic system [58, 59]. In patients with fecal incontinence, limited information is available to explain the mechanism of action. A small study demonstrated that SNS was associated with higher tolerance of rectal distention, but the neurologic mechanism behind this is unclear [60]. Probably, the pudendal afferent somatic fibers work by inhibiting colonic propulsive activity and activating the internal anal sphincter [61]. The action on colonic motility may explain why patients with significant anal sphincter defects may benefit from SNS.

6.3. Injectable/implantable bulking agents

Injectable agents have been used for the first time as a treatment for urinary incontinence (UI), with the advantages of an ambulatory procedure and low morbidity rate but with variable success. Thereafter, different injectable agents have been employed for FI. The use of bulking agents in patients with FI is still controversial, mostly because of conflicting results and lack of agreement regarding adequate indications. Moreover, different techniques of injection have been performed, and several agents have been used via injection: Fat, PTQ®, Durasphere®, Coaptite®, NASHA TM -Dx, Permacol®, and BulkamidTM. Different techniques of delivery have been described, providing a submucosal injection inside the anal canal, intersphincteric or within the sphincter defect scar tissue; transanal/transmucosal, transsphincteric or intersphincteric were the route of injection at different areas of the anal canal, in two/three/four or more points [62–66].

Recently, a novel approach has been introduced to treat patients with FI, by the placement of implantable agents, in the form of thin cylinders, within the sphincteric complex. The THD Gatekeeper TM was the first device used, but very recently, the THD SphinKeeperTM has been available for procedure. Gatekeeper TM implants are made of a material (HYEXPAN TM) that is both solid at the time of delivery and slowly absorbs water to expand itself once implanted. Within 48 h, the implant should have reached its definitive size and shape. At this step, the consistency of the material has moved from hard to soft with shape memory, giving the implant a pliable texture that makes it compliant to external pressures without losing its original shape. For these reasons, it was decided to place the implants in the intersphincteric space, in the belief that this would achieve a more effective distribution of a presumed "bulking effects" than would be achieved with submucosal positioning, thus exploiting the physical characteristic of the implant most effectively. However, the "bulking effects" should be not the only and/or main effect contributing to the therapeutic efficacy. The intersphincteric location should also minimize the potential risk of erosion, ulceration, fistulation of the anal canal, and possible displacement of the prosthesis [67–70].

6.4. Artificial bowel sphincter

The artificial bowel sphincter (ABS together with dynamic graciloplasty and sacral nerve stimulation (SNS)) is still considered an optional treatment for refractory conservative treatment and severe fecal incontinence. Christiansen and Lorentzen first reported in 1987 a perianal implantation of an adapted artificial urinary sphincter (AMS 800, America Medical System) for a patient with fecal incontinence [71]. In 1996, Lehur and colleagues described the results obtained with an artificial bowel sphincter designed just for FI (Acticon Neosphincter – American Medical System) [72]. To date, despite the good results reported in the literature, in terms of improved continence and quality of life, the rate of surgical explantation and surgical procedures for infections of ABS still remains too high [73]. These were the reasons that reduced a wide acceptance of ABS in coloproctology practice. In accordance with Wexner et al., the cumulative risk of device explant increases with time but less dramatically in the longer follow-up [74]. Moreover, Wong et al. have shown, in long-term follow-up, as after explantation for infection the reimplantation can be performed without difficulty [75].

ABS implantation represents the last resort after failure of conservative and less-invasive surgical procedures in fecal incontinence [76]. It is indicated especially in patients with almost complete sphincter damage or post-surgical sphincter excision or for patients with congenital malformation or with significant neurological dysfunction [77]. In order to achieve long-term satisfying results and to use the device completely and competently, potential candidates must not have recent or active perineal infection and should not have manual limitations [78, 79]. Artificial sphincter was used before for treating urinary incontinence and later modified for fecal incontinence. The ABS, Acticon Neosphincter (American Medical Systems, Minnetonka, MN, USA) aims to control incontinence by mimicking the natural action of the sphincter muscle. The device composed of three parts: an inflatable cuff that works as the new sphincter and seals the anal canal, a control pump, and a pressure-regulating balloon that also functions as a fluid reservoir connected by two special tubes system [80]. The patient is placed in the lithotomy position under general anesthesia. The cuff is positioned creating a tunnel around the rectum; the balloon is implanted ahead to the bladder in the Retzius space and the pump is inserted into the major labia in women or inside the scrotum in men [79, 81, 82].

6.5. Reconstructive surgery

Reconstructive surgery is indicated more specifically in cases of fecal incontinence incurred by anal sphincter lesions, abnormalities, or deformities, as well as sphincter deficiency with no evident lesions and abnormalities of the pelvic floor.

The following are several reconstructive techniques:

- Sphincteroplasty.
- Suture of the levator ani.
- Reconstruction of the sphincter complex using muscle repair.

Surgical techniques which resulting in a direct repair are only indicated for lesions located in the external anal sphincter. The main cause of sphincter lesions is obstetric trauma. Despite the lack of any particular continence consequences caused by childbirth, 1–4% of deliveries result in lesions of the sphincter complex or of the pelvic floor (lesions of the third and fourth degrees) [83–86]. Fetus weight, surgical incision on the midline of the perineum (episiotomy), the use of forceps, and breech presentation are considered the main risk factors of sphincter damage [87–89]. Obstetric damages can be detected immediately in the postpartum and are caused by third degree laceration, but approximately in 40% of cases [88], continence dysfunctions are detected as early as 6 months post-delivery [85]. The most frequently performed surgical procedure for the treatment of obstetric lesions is direct anterior sphincter suture repair [89]. Optimal timing for the repair is within 3–4 months following the trauma. Anal sphincter repair can be performed using the "end-to-end" technique, thereby facing the two laps after resecting scar tissue as well as through the "overlapping" technique, which is performed by overlaying the residual functional extremities. The first technique is used to repair recent injuries in which the scar that has outdistanced the extremities of the muscles is not yet formed, thus allowing for the facing of the extremities without excessive tension. In old injuries, the sphincter defect is often consolidated, and a direct suture of the extremities is to be avoided at all cost, as it would be invariably destined to failure. The overlapping technique is generally quite safe for sphincter suture repair; suture repairs of the pelvic muscles, performed alone or with a sphincteroplasty, are carried out in order to treat muscle deficit or defects. The goal of this type of technique is the restoration of tension to the functionally deficient sphincter muscles through the use of plication. In the history of surgery, the first recommended and validated procedure was the postanal repair, presented by Parks in 1971 and subsequently modified [90]. This procedure was at first suggested to patients with neurogenic or idiopathic fecal incontinence, with no sphincter defect. The anterior levatorplasty procedure is often performed to treat pelvic trauma frequently resulting from obstetric injury. These types of surgical procedures are performed when an attempt to restore the sphincter using the aforementioned technique has not led to any effective results. The logic behind this strategy is to recreate the anal sphincter by replacing degenerative tissue with ectopic muscle located at the perineal level or by using a prosthetic device [91]. Muscle transposition and prosthetic replacement are two different techniques, yet both utilize the same functionality: to create an area with high pressure around the terminal part of the gastrointestinal tract by tightening around the distal rectum. Another option is the muscle of the lower limb, which extends from the ischium to the knee joint, also called “rectus femoris muscle” alternatively; the gluteus maximus muscle may be used. Dynamic graciloplasty is often indicated as the type of procedure with the most favorable outcomes, above all thanks to its anatomical characteristics that predispose its transposition [92].

6.6. Intestinal ostomy

When all surgical treatments fail, bowel ostomy may be considered an effective, safe, and appropriate surgical solution for patients with severe incontinence [93]. Indications for colostomy/ileostomy include spinal cord injury, complete pelvic floor denervation, severe perineal trauma, and actinic FI that can lead to severe neurogenic incontinence. It is performed on patients immobilized with skin problems or other complications too [94] or on those who are physically or mentally incapable without any bowel control resulting in a poor quality of life [95]. The creation of a colostomy or ileostomy provides definitive control of fecal incontinence. It is usually performed if other treatment options had no satisfying results. Patients are usually understandably very unwilling to the idea of a permanent ostomy, fearing it will be difficult to manage due to the great impact on self-image and social interactions.

Conflict of interest

The authors had no conflict of interest.

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References

[1] Pucciani F. Fecal soiling: Pathophysiology of postdefaecatory incontinence. Colorectal Disease. 2013;**15**:987-992

[2] Stergios DK. Childbirt Trauma. London: Springer Verlag Ltd; 2017. 329 p. DOI: 10.1007/978-1-4471-6711-2

[3] O'Donnell LJ, Virjee J, Heaton KW. Pseudo-diarrhoea in the irritable bowel syndrome: Patients' records of stool form reflect transit time while stool frequency does not. Gut. 1988;**29**:A1455

[4] Sultan AH. Obstetric perineal injury and anal incontinence. Clinical Risk. 1999;**5**(6):193-196

[5] Koelbl H, Igawa T, Salvatore S, Laterza RM, Lowry A, Sievert KD, et al. Pathophysiology of urinary incontinence, faecal incontinence and pelvic organ prolapse. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence. 5th ed. Paris: ICUDEAU; 2013. pp. 261-359

[6] Rockwood TH, Church JM, Fleshman JW, et al. Patient and surgeon ranking of the severity of symptoms associated with fecal incontinence: The fecal incontinence severity index. Diseases of the Colon and Rectum. 1999;**42**:1525-1532

[7] Rockwood TH, Church JM, Fleshman JW, Kane RL, Mavrantonis C, Thorson AG, Wexner SD, Bliss D, Lowry AC. Fecal incontinence quality of life scale: Quality of life instrument for patients with fecal incontinence. Diseases of the Colon and Rectum. 2000;**43**:9-16

[8] de la Portilla F, Calero-Lillo A, Jiménez-Rodríguez RM, Reyes ML, Segovia-González M, Maestre MV, García-Cabrera AM. Validation of a new scoring system: Rapid assessment faecal incontinence score. World Journal of Gastrointestinal Surgery. 2015 Sep 27;**7**(9):203-207

[9] Eypasch E, Williams JI, Wood-Dauphinee S, Ure BM, Schmulling C, Neugebauer E, Troidl H. Gastrointestinal quality of life index (development, validation and application of a new instrument). The British Journal of Surgery. 1995;**82**:216-222

[10] Jorge JM, Wexner SD. Etiology and management of fecal incontinence. Diseases of the Colon and Rectum. 1993;**36**:77-97

[11] Coura MM, Silva SM, Almeida RM, Forrest MC, Sousa JB. Is digital rectal exam reliable in grading anal sphincter defects. Archives of Gastroenterology. 2016 Oct-Dec;**53**(4):240-245

[12] Rao SS, Azpiroz F, Diamant N, et al. Minimum standards of anorectal manometry. Neurogastroenterology and Motility. 2002;**14**(5):553-559

[13] Rao SS. Advances in diagnostic assessment of fecal incontinence and dyssynergic defecation. Clinical Gastroenterology and Hepatology. 2010;**8**(11):910-919

[14] Jones MP, Post J, Crowell MD. High-resolution manometry in the evaluation of anorectal disorders: A simultaneous comparison with water-perfused manometry. The American Journal of Gastroenterology. 2007;**102**(4):850-855

[15] Rao SS, Hatfi eld R, Soffer E, et al. Manometric tests of anorectal function in healthy adults. The American Journal of Gastroenterology. 1999;**94**(3):773-783

[16] Gruppo Lombardo per lo Studio della Motilità Intestinale. Anorectal manometry with water- perfused catheter in healthy adults with no functional bowel disorders. Colorectal Disease. 2010;**12**:220-225

[17] Bennett RC, Duthie HL. The functional importance of the internal anal sphincter. The British Journal of Surgery. 1964;**51**(5):355-357

[18] Frenckner B, Euler CV. Influence of pudendal block on the function of the anal sphincters. Gut. 1975;**16**(6):482-489

[19] Liu J, Guaderrama N, Nager CW, Pretorius DH, Master S, Mittal RK. Functional correlates of anal canal anatomy: Puborectalis muscle and anal canal pressure. The American Journal of Gastroenterology. 2006;**101**(5):1092-1097

[20] Fox JC, Fletcher JG, Zinsmeister AR, et al. Effect of aging on anorectal and pelvic fl oor functions in females. Diseases of the Colon and Rectum. 2006;**49**(11):1726-1735

[21] Raza N, Bielefeldt K. Discriminative value of anorectal manometry in clinical practice. Digestive Diseases and Sciences. 2009;**54**(11):2503-2511

[22] Felt-Bersma RJ, Sloots CE, Poen AC, et al. Rectal compliance as a routine measurement: Extreme volumes have direct clinical impact and normal volumes exclude rectum as a problem. Diseases of the Colon and Rectum. 2000;**43**(12):1732-1738

[23] Ricciardi R, Mellgren AF, Madoff RD, et al. The utility of pudendal nerve terminal motor latencies in idiopathic incontinence. Diseases of the Colon and Rectum. 2006;**49**:852-857

[24] Pucciani F. Faecal soiling: Pathophysiology of postdefaecatory incontinence. Colorectal Disease. 2013;**15**:987-992

[25] Santoro GA, Wieczorek AP, Dietz HP, et al. State of the art: An integrated approach to pelvic floor ultrasonography. Ultrasound in Obstetrics and Gynecology. 2011;**37**:381-396

[26] Sudakoff GS, Quiroz F, Foley WD. Sonography of anorectal, rectal, and perirectal abnormalities. American Journal of Roentgenology. 2002;**179**:131-136

[27] Frudinger A, Bartram CI, Halligan S, et al. Examination techniques for endosonography of the anal canal. Abdominal Imaging. 1998;**23**(3):301

[28] Thakar R, Sultan AH. Anal endosonography and its role in assessing the incontinent patient. Best Practice and Research. Clinical Obstetrics and Gynaecology. 2004;**18**:157-173

[29] Abdool Z, Sultan AH, Thakar R. Ultrasound imaging of the anal sphincter complex: A review. The British Journal of Radiology. 2012;**85**:865-875

[30] Frudinger A, Halligan S, Bartram CI. Female anal sphincter: Age related differences in asymptomatic volunteers with high-frequency endoanal US. Radiology. 2002;**224**:417-423

[31] Mascagni D, Naldini G, Stuto A, et al. Recurrence after stapled haemorrhoidopexy. Techniques in Coloproctology. 2015;**19**:321-322

[32] Lengyel AJ, Hurst NG, Williams JG. Pre-operative assessment of anal fistulas using endoanal ultrasound. Colorectal Disease. 2002;**4**:436-440

[33] Pescatori M, Interisano A, Mascagni D, et al. Double flap technique to reconstruct the anal canal after concurrent surgery for fistulae, abscesses and haemorrhoids. International Journal of Colorectal Disease. 1995;**10**:19-21

[34] Navarro-Luna A, Garcia-Domingo MI, Rius-Macias JR, et al. Ultrasound study of anal fistulas with hydrogen peroxide enhancement. Diseases of the Colon and Rectum. 2004;**47**:108-114

[35] Ratto C, Gentile E, Merico M, et al. How can the assessment of fistula-in-ano be improved? Diseases of the Colon and Rectum. 2000;**43**:1375-1382

[36] Stoker J. Magnetic resonance imaging in fecal incontinence. Seminars in Ultrasound, CT, and MR. 2008;**29**:409-413

[37] Terra MP, Beets-Tan RG, van der, Hulst VPM, et al. MR imaging in evaluating atrophy of the external anal sphincter in patients with fecal incontinence. American Journal of Roentgenology. 2006;**187**(4):991-999

[38] Terra MP, Beets-Tan RG, van der Hulst VPM, et al. Anal sphincter defects in patients with fecal incontinence: Endoanal versus external phased-array MR imaging. Radiology. 2005;**236**:886-895

[39] Bharucha AE, Fletcher JG, Melton LJ III, Zinsmeister R. Obstetric trauma, pelvic floor injury and fecal incontinence: A population based case-control study. The American Journal of Gastroenterology. 2012;**107**:902-911

[40] Madoff RD. Surgical treatment options for fecal incontinence. Gastroenterology. 2004; **126**:S48-S54

[41] Schiedeck H, Schwandner O, Scheele J, Farke S, Bruch HP. Rectal prolapse: Which surgical option is appropriate? Langenbeck's Archives of Surgery. 2005;**390**:8-14

[42] Dobben AC, Terra MP, Slors JF, et al. External anal sphincter defects in patients with fecal incontinence: Comparison of endoanal MR imaging and endoanal US. Radiology. 2007;**242**:463-471

[43] Cazemier M, Terra MP, Stoker J, et al. Atrophy and defects detection of the external anal sphincter: Comparison between three-dimensional anal endosonography and endoanal magnetic resonance imaging. Diseases of the Colon and Rectum. 2005;**49**:20-27

[44] Dobben AC, Terra MP, Deutekom M, et al. The role of endoluminal imaging in clinical outcome of overlapping anterior anal sphincter repair in patients with fecal incontinence. American Journal of Roentgenology. 2007;**189**:W70-W77

[45] Malouf AJ, Halligan S, Williams AB, et al. Prospective assessment of interobserver agreement for endoanal MRI in fecal incontinence. Abdominal Imaging. 2001;**26**:76-78

[46] Townsend MK, Mattews CA, Whithehead WE, Grodstein F. Risk factors for fecal incontinence inolder women. The American Journal of Gastroenterology. 2013;**108**:113-119

[47] Umphred D. Neurological rehabilitation. St. Louis: MosbyElsevier; 2007

[48] Rao SSC. Current and emerging treatment options for fecal incontinence. Journal of Clinical Gastroenterology. 2014;**48**:752-764

[49] Scott KM. Pelvic floor rehabilitation in the treatment of fecal incontinence. Clinics in Colon and Rectal Surgery. 2014;**27**:99-105

[50] Meyer I, Richter HE. An evidence-based approach to the evaluation, diagnostic assessment and treatment of fecal incontinence in women. Current Obstetrics and Gynecology Reports. 2014;**3**:155-164

[51] van der Wilt AA, Giuliani G, Kubis C, van Wunnik BPW, Ferreira I, Breukink SO, Lehur PA, La Torre F, Baeten CGMI. Randomized clinical trial of percutaneous tibial nerve stimulation versus sham electrical stimulation in patients with faecal incontinence. The British Journal of Surgery. 2017 Aug;**104**(9):1167-1176

[52] Person B, Wexner SD. Advances in the surgical treatment of faecal incontinence. Surgical Innovation. 2005;**12**(1):7-21

[53] Barber MD, Bremer RE, Thor KB, Dolber PC, Kuehl TJ, Coates KW. Innervation of the female levator ani muscles. American Journal of Obstetrics and Gynecology. 2002;**187**(1): 64-71

[54] Grigorescu BA, Lazarou G, Olson T, Downie SA, Powers K, Greston WM, Mikhail MS. Innervation of the levator ani muscles: Description of the nerve branches to the pubococcygeus, iliococcygeus, and puborectalis muscles. International Urogynecology Journal. 2008;**19**:107-116

[55] Chan CLH, Ponsford S, Scott SM, Swash M, Lunniss PJ. Contribution of the pudendal nerve to sensation of the distal rectum. The British Journal of Surgery. 2005;**92**:859-865

[56] Matzel KE, Schmidt RA, Tanagho EA. Neuroanatomy of the striated muscular anal continence mechanism. Diseases of the Colon and Rectum. 1990;**33**(8):666-673

[57] Fowler CJ, Swinn MJ, Goodwin RJ, Oliver S, Craggs M. Studies of the latency of pelvic floor contraction during peripheral nerve evaluation show that the muscle response is refl exly mediated. The Journal of Urology. 2000;**163**(3):881

[58] Chancellor MB, Chartier-Kastler EJ. Principles of sacral nerve stimulation (SNS) for the treatment of bladder and urethral sphincter dysfunctions. Neuromodulation: Technology at the Neural Interface. 2000;**3**(1):16-26

[59] Blok BF, Groen J, Bosch JL, Veltman DJ, Lammertsma AA. Different brain effects during chronic and acute sacral neuromodulation in urge incontinent patients with implanted neurostimulators. BJU International. 2006;**98**:1238-1243

[60] Abdel-Halim MR, Crosbie J, Engledow A, Windsor A, Cohen CR, Emmanuel AV. Temporary sacral nerve stimulation alters rectal sensory function: A physiological study. Diseases of the Colon and Rectum. 2011;**54**:1134-1140

[61] Gourcerol G, Vitton V, Leroi AM, Michot F, Abysique A, Bouvier M. How sacral nerve stimulation works in patients with faecal incontinence. Colorectal Disease. 2011;**13**: e203-e211

[62] Watson NF. Anal bulking agents for faecal incontinence. Colorectal Disease. 2012 Dec; **14**(Suppl 3):29-33

[63] Guerra F, La Torre M, Giuliani G, Coletta D, Amore Bonapasta S, Velluti F, et al. Long-term evaluation of bulking agents for the treatment of fecal incontinence: Clinical outcomes and ultrasound evidence. Techniques in Coloproctology. 2015;**19**:23-27

[64] Hussain ZI, Lim M, Stojkovic SG. Systematic review of perianal implants in the treatment of fecal incontinence. The British Journal of Surgery. 2011;**98**:1526-1536

[65] Morris OJ, Smith S, Draganic B. Comparison of bulking agents in the treatment of faecal incontinence: A prospective randomized clinical trial. Techniques in Coloproctology. 2013;**17**:517-523

[66] La Torre F, de la Portilla F. Long-term efficacy of dex-tranomer in stabilized hyaluronic acid (NASHA/dx) for treatment of faecal incontinence. Colorectal Disease. 2013;**15**:569-574

[67] Ratto C, Parello A, Dionisi L, et al. Novel bulking agent for faecal incontinence. The British Journal of Surgery. 2011;**98**:1644-1652

[68] Ratto C, Buntzen S, Aigner F, Altomare DF, Heydari A, Donisi L, Lundby L, Parello A. Multicentre observational study of the gatekeeper for faecal incontinence. The British Journal of Surgery. 2016;**103**:290-299

[69] Al-Ozaibi L, Kazim Y, Hazim W, Al-Mazroui A, Al-Badri F. The gatekeeper™ for fecal incontinence: Another trial and error. International Journal of Surgery Case Reports. 2014;**5**:936-938

[70] de la Portilla F, Reyes-Díaz ML, Maestre MV, Jiménez-Rodríguez RM, García-Cabrera AM, Vázquez-Monchul JM, Díaz-Pavón JM, Padillo-Ruiz FC.Ultrasonographic evidence of Gatekeeper™ prosthesis migration in patients treated for faecal incontinence: A case series. International Journal of Colorectal Disease. 2017 Mar;**32**(3):437-440

[71] Christiansen J, Lorentzen M. Implantation of artificial sphincter for anal incontinence. Lancet. 1987;**2**:244e245

[72] Lehur PA, Michit F, Denis P, Grise P, Leborgne J, Teniere P, et al. Results of artificial sphincter in severe anal incontinence. Diseases of the Colon and Rectum. 1996;**39**:1352-1355

[73] Hong KD, Dasilva G, Kalaskar SN, et al. Long-term outcomes of artificial bowel sphincter for fecal incontinence: A systematic review and meta-analysis. Journal of the American College of Surgeons. 2013;**217**(4):718-725

[74] Wexner SD, Jin HY, Weiss EG, et al. Factors associated with failure of the artificial bowel sphincter: A study of over 50 cases from Cleveland Clinic Florida. Diseases of the Colon and Rectum. 2009;**52**:1550e1557

[75] Wong MT, Meurette G, Wyart V, et al. The artificial bowel sphincter: A single institution experience over a decade. Annals of Surgery. 2011;**254**(6):951

[76] Lee YY. What's new in the toolbox for constipation and fecal incontinence? Frontiers of Medicine (Lausanne). 2014;**1**:5

[77] Galandiuk S, Roth LA, Greene QJ. Anal incontinence sphincter ani repair: Indications, techniques, outcome. Langenbeck's Archives of Surgery. 2009;**394**(3):425-433

[78] Tan JJ, Chan M, Tjandra JJ. Evolving therapy for fecal incontinence. Diseases of the Colon and Rectum. 2007;**50**(11):1950-1967

[79] Edden Y, Wexner SD. Therapeutic devices for fecal incontinence: Dynamic graciloplasty, artificial bowel sphincter and sacral nerve stimulation. Expert Review of Medical Devices. 2009;**6**:307-312

[80] Person B, Wexner SD. Advances in the surgical treatment of fecal incontinence. Surgical Innovation. 2005;**12**(2):182

[81] Altomare DF, Dodi G, La Torre F, Romano G, Melega E, Rinaldi M. Multicentre retrospective analysis of the outcome of artificial anal sphincter implantation for severe faecal incontinence. The British Journal of Surgery. 2001;**88**(11):1481-1486

[82] La Torre F, Masoni L, Montori J, et al. The surgical treatment of fecal incontinence with artificial anal sphincter implant. Preliminary clinical report. Hepato-Gastroenterology. 2004;**51**:1358-1361

[83] Fernando RJ, Sultan AH, Kettle C, Thakar R. Methods of repair for obstetric anal sphincter injury. Cochrane Database of Systematic Reviews. 2013 Dec 8;**12**

[84] Premkumar G. Perineal trauma: Reducing associated postnatal maternal morbidity. RCM Midwives. 2005;**8**(1):30-32

[85] Fornell EU, Matthiesen L, Sjodahl R, Berg G. Obstetric anal sphincter injury ten years after: Subjective and objective long term effects. BJOG: An International Journal of Obstetrics and Gynaecology. 2005;**112**:312-316

[86] Rieger N, Wattchow D. The effect of vaginal delivery on anal function. The Australian and New Zealand Journal of Surgery. 1999;**69**:172-177

[87] Valsky DV, Lipschuetz M, Bord A, Eldar I, Messing B, Hochner-Celnikier D, Lavy Y, Cohen SM, Yagel S. Fetal head circumference and length of secondary stage ah labor are risk for levator ani muscle injury, diagnosed by 3-dimensional transperineal ultrasound in primiparous women. American Journal of Obstetrics and Gynecology. 2009;**201**:91-97

[88] Midwifery J, McCandlish R. Perineal trauma: Prevention and treatment. Women's Health. 2001;**46**(6):396-401

[89] Lamblin G, Bouvier P, Damon H, Chabert P, Moret S, Chene G, Mellier G. Long-term outcome after over lapping anterior anal sphincter repair for fecal incontinence. International Journal of Colorectal Disease. 2014;**29**:1377-1383

[90] Parks AG, McPartlin JF. Late repair of injuries of the anal sphincter. Proceedings of the Royal Society of Medicine. 1971;**64**(12):1187-1189

[91] Brown SR, Wadhawan H, Nelson RL. Surgery for faecal incontinence in adults. Cochrane Database of Systematic Reviews. 2013 Jul 2;**7**

[92] Walega P, Romaniszyn M, Siarkiewicz B, Zelazny D. Dynamic versus adynamic graciloplasty in treatmentof end-stage fecal incontinence: Is the implantation of the pacemaker really necessary? 12-month follow-up in a clinical, physiological, and functional study. Gastroenterology Research and Practice. 2015;**2015**:698516

[93] Vaizey CJ, Kamm MA, Nicholls RJ. Recent advances in the surgical treatment of faecal incontinence. The British Journal of Surgery. 1998;**85**:596-603

[94] Senapati A, Phillips RK. The trephine colostomy: A permanent left iliac fossa end colostomy without recourse to laparotomy. Annals of the Royal College of Surgeons of England. 1991;**73**:305-306

[95] Poirier M, Abcarian H. Fecal incontinence. In: Cameron JL, editor. Current Surgical Therapy. Philadelphia: Mosby Elsevier; 2008. pp. 285-291