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An Evidence-Based Approach to the Evaluation, Diagnostic Assessment and Treatment of Fecal Incontinence in Women

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Abstract

Fecal incontinence (FI) is a debilitating disorder which negatively impacts quality of life. The etiology is often multifactorial and although most women with FI are able to be treated, many remain untreated because a significant proportion of women do not report their symptoms and seek care. The evaluation and treatment of FI can be also hindered by a lack of understanding of the mechanisms and current options. This article provides a review on the evidence-based evaluation and management for FI.

Keywords

Fecal incontinence; Anal incontinence; Defecatory disorders; Accidental bowel leakage

Introduction

Fecal incontinence (FI) is a physically and psychosocially debilitating condition which negatively impacts quality of life (QOL). The prevalence of FI in community dwelling women in the United States (US) varies considerably depending on the population studied and the definition of FI with rates of 2.2 to 24 %.[1–8] The estimated prevalence is disproportionately higher in the older woman. By 2030, more than one-fifth of women will be 65 years or older. As this proportion of the population increases, there will be substantial burden of healthcare and pelvic floor symptoms in community dwelling women.[3]

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The National Institute of Health (NIH) released a consensus and state-of-the-science statement regarding incontinence in adults in 2007 to reduce the suffering and burden of incontinence in adults.[9,10] The statement emphasized the importance of efforts to raise public awareness of incontinence and the benefits of prevention and management in order to eliminate stigma, promote disclosure and care seeking, and reduce suffering.[9,10] It has been recently reported that women with FI prefer the term, accidental bowel leakage (ABL), to describe their condition.[9,11] As providers of care for FI, when speaking with our patients or publically regarding this condition, it is recommended that we use the term ABL.

The definition of FI is inconsistent among existing reports. According to the terminology by International Urogynecology Association and International Continence Society, FI is the complaint of involuntary loss of solid/liquid feces, whereas anal incontinence (AI) pertains to loss of feces or flatus, which is perceived as a social or hygienic problem.[12] Neither definition quantifies the impact on quality of life or the time period during which the condition is measured. In recent years, improvements have been made in the traditional diagnostic and therapeutic modalities, and several new treatments have emerged. This article will focus on the advances of assessment and management of FI.

Continence Mechanism

The FI mechanism is dependent upon anal sphincter function, rectal sensation, adequate rectal capacity and compliance, colonic transit time, stool consistency, cognitive and neurologic factors. Incontinence occurs when any one or more of these factors are impacted. In a prospective study, 80% of patients had more than one pathogenic abnormality.[13,14] Proper diagnosis and treatment of FI requires an understanding of the complex pelvic floor musculature, innervation, and function, as well as compensatory mechanisms.

The normal resting pressure is produced by the internal anal sphincter (IAS, 75–80%) and external internal sphincter (EAS, 20–25%). Anal cushions are connective tissue complexes that contains smooth muscle cells and vascular channels providing an effective tight seal of the anal canal in concert with the IAS.[15–18] The rectoanal inhibitory reflex (RAIR) induces the IAS to relax in response to rectal distention, allowing anorectal sampling and preparation of the canal for defecation.[19] Fecal impaction, particularly in older women, leads to overflow incontinence by chronic inhibition of the IAS.[20] The anal sphincter complex has autonomic innervation via both parasympathetic (pelvic) and sympathetic (hypogastric) nerves. The EAS with the puborectalis provides voluntary control over defecation via the pudendal nerve. Pudendal nerve injury can occur when the nerve is stretched by descent of the perineal/pelvic floor. This can occur with obstetric injury, as well as with a history of chronic straining during stooling where sustained pelvic floor descent can cause the traction neuropathy.[20]

Risk Factors

Risk factors for FI include advanced age, diarrhea, obstetric injury, obesity, physical limitation, neurological disorders, urinary incontinence, and increasing parity.[21] FI associated with pregnancy and childbirth has been extensively studied. Obstetric anal sphincter injury (OASIS) is the most established and potentially the most modifiable risk

factor. The role of vaginal delivery (VD) on FI is controversial according to both short and long-term studies.[21–24] Operative delivery, especially forceps-assisted VD, is a well-documented risk factor for OASIS and subsequent FI.[25–27] The occiput posterior fetal position further increases the risk.[28] Other documented risk factors include median episiotomy, fetal macrosomia, and increased maternal age.[29,30] In prospective studies, nearly 35% of primiparous women showed sphincter disruption following normal VD.[14,28,31] However, the majority of women with sphincter defects following VD do not experience incontinence in the immediate postpartum period.[20,32] Currently, the American Congress of Obstetricians and Gynecologists does not suggest primary elective cesarean delivery for prevention of FI (Grade 2C) because planned cesarean delivery conferred no protection against FI.[33,34] However, in women with a history of FI or OASIS, the optimal mode of delivery for subsequent pregnancies is uncertain.[35,36] Other risk factors for FI are listed in Table 1.

Diagnostic Evaluation

Physical Examination and Anorectal Manometry

Evaluation of patients with FI consists of a careful history followed by a thorough physical examination and diagnostic testing as indicated. Validated questionnaires are useful to assess the symptom severity and the effects of FI on QOL. There are three main types of FI symptoms: 1) passive leakage – stool leakage with little or no forewarning, 2) urgency incontinence – unable to defer defecation due to lack of voluntary control, 3) seepage – leakage shortly after bowel movements due to incomplete evacuation of stool.[15] Inability to evacuate stool can be due to obstructive or dyssynergic defecation as well as impaired anorectal sensation.

Digital examination provides a crude assessment of the anal resting and squeeze tone, disruption of the anal sphincter, the anorectal angle, pelvic floor descent, rectal masses, and other pathologic conditions. The accuracy of digital examination is operator dependent. Overall, rectal examinations have been proven as reliable as anal manometry in assessing anal resting and squeeze tone.[37] However, a correlation between the physical examination and manometric measures have been controversial.[14,38–41] The positive predictive value (PPV) of digital examinations to identify low resting and squeeze pressure by experienced clinicians was reported to be 67% and 81%, respectively.[42,43] Complaints of ‘leakage’ have a sensitivity of 98.9%, specificity of 11%, and PPV of 51% for detecting low resting pressures.[42] Anorectal manometry (ARM) results do not always correlate with symptom severity and do not consistently predict postoperative success of anal sphincter repair.[44] When comparing history vs physiologic testing alone, the correct diagnosis of FI was made 11% vs 55%, respectively.[43,45] This may be because most women with FI have more than one continence mechanisms compromised.[14,43] Another study demonstrated that anorectal testing results led to an alteration of diagnosis and treatment in 19% and 16%, compared to history and examination alone.[46] Therefore, objective testing plays an important role in the evaluation of FI in more complicated patients.

Although multiple diagnostic studies are available to assess FI, few guidelines exist to delineate when specific testing should be performed.[47] The American Gastroenterological

Association (AGA) issued recommendations for anorectal testing techniques with suggestions addressing standardization of testing and interpretation of manometry measures. [48]

Anorectal manometry with rectal sensory testing is the preferred method for defining the functional weakness of the anal sphincter complex and for detecting abnormal rectal sensation.[49,50] Currently, several types of probes and pressure-recording devices are available. Each system has distinct advantages and drawbacks.[14,51,52] A water-perfused probe with multiple closely spaced sensors is most commonly used. A solid-state probe with micro-transducers does not require perfusion equipment, is easier to calibrate and possibly more accurate, however more expensive and fragile.[14,51–53] The sensation threshold is valuable for determining whether biofeedback is useful, as a poor rectal sensory threshold correlates with low biofeedback success rates.[48] “Normal” values vary depending on the population studied.

Endoanal Ultrasound

Endoanal ultrasound (EAU) assesses structural integrity and morphology of the anal sphincters. A traditional probe has a 7mHz rotating transducer with a focal length of 1–4 cm. Higher frequency transducers (10–15mHz) can provide better delineation of the sphincter complex.[14,54,55] Both 2D and 3D ultrasound approaches are currently available, as well as translabial/perineal approaches. The advantages of the 3D ultrasound are the ability to investigate the suspicious areas with 3D volume images as a cine loop. It also allows the operator to distinguish EAS defects from other close structures.[53,56] EAU is reliable for identifying IAS defects,[53,57] whereas the assessment of EAS can be operator dependent, confounded by normal anatomic variations such as an EAS gap. EAS thickness < 10mm is suspicious for sphincter injuries.[53,58] A sphincter defect must be viewed in 2 out of 3 locations (upper, middle, distal canal) for accurate diagnosis. Whether sphincter defects on EAU are the etiology of FI is somewhat controversial, as EAU has a low specificity for diagnosis[14,59] and the degree of separation on ultrasound may not correlate with symptom severity.[53,60] Magnetic resonance imaging (MRI) was previously considered superior in detecting EAS injuries. However, with improvements on the sonographic technology, there is no significant difference between endoanal MRI and ultrasound in depicting EAS defects in patients with FI.[61] The sensitivity for detecting EAS defects for MRI and ultrasound was 81%, 90% respectively, and the PPV was 89% for MRI and 85% for EAU.[62] The cost, clinical utility, and availability are often the hindering factors for MRI.

Studies comparing electromyography (EMG) mapping with EAU showed a high concordance rate for identifying sphincter defects.[14] EMG assesses anal sphincter activity using a surface electrode or a concentric needle and can be helpful to distinguish neurogenic from myogenic damage.

Pudendal Nerve Assessment

Pudendal nerve terminal motor latency (PNTML) measures the time required after stimulating the pudendal nerve with an electrode to induce an EAS contraction. Prolongation of PNTML suggests pudendal neuropathy. However, one may have normal fast twitch

muscle function demonstrated, but abnormal slow twitch which would not be measured by PNTML. Bilateral, not unilateral, neuropathy has been associated with diminished sphincter function and higher incontinence scores.[53] Some studies reported PNTML is useful in predicting the outcome of sphincteroplasty in conjunction with other modality.[49] Good to excellent sphincteroplasty outcomes with and without pudendal neuropathy on PNTML have been reported 11% vs 80% respectively.[49] However, PNTML is operator dependent and has a poor correlation with clinical symptoms and histologic findings.[48] The AGA does not recommend PNTML for routine evaluation.[49, 63]

Emerging neurophysiological tests have been recently introduced. The neural circuitry of afferent and efferent pathways can be evaluated using cortical and motor evoked potentials of the rectum (CEP, MEP), and anal sphincter response to magnetic stimulation of the motor cortex (transcranial magnetic stimulation [TMS]). The combination of afferent and efferent circuitry evaluation provides a novel, integrated, comprehensive method to evaluate “gut-brain-gut” interactions. CEP, MEP, and TMS are largely used in research and clinical trials, currently not available for clinical use.[19,49,64–67]

Management

The goal should focus on restoring continence and improving QOL. Unfortunately, no single option has been shown to provide consistent, long-term effectiveness with low complication rates, making FI extremely difficult to manage.[19] The main approaches are: conservative including medical, pelvic muscle exercises/physical therapy ± biofeedback, and surgery. One of the most important aspects of treatment is patient education on stool consistency, bowel function and diet to avoid offending foods.

Dietary Considerations

The frequency and consistency of stool can greatly affect symptom severity. High fiber, particularly soluble, can increase stool bulk and firmness and is especially helpful in women with low-volume loose stools.[14] Methylcellulose is 100% soluble and non-fermentable, which absorbs water and increases the stool transit time. Recommended supplementation is 2–6 grams per day (daily fiber intake: 25–35 grams). Other fiber types include psyllium (70% soluble) and calcium polycarbophil, an insoluble hydrophilic fiber, which also works as a bulking agent.[68–69]

Medications

Loperamide (Imodium®) is a synthetic opioid which inhibits intestinal peristalsis increasing oral-cecal transit time. Loperamide also increases resting anal sphincter tone, improves rectal perception and rectal compliance.[70] Compared to placebo, loperamide was more effective for reducing urgency FI, with more people achieving full continence, improved symptoms, and fewer FI episodes.[71] Loperamide has fewer central nervous system side effects compared to diphenoxylate (Lomotil).[72] The most recent ongoing randomized controlled trial with Loperamide (CAPABLE: Controlling Anal incontinence by Performing Anal exercises with Biofeedback or Loperamide) is currently being undertaken by the NICHD Pelvic Floor Disorders Network (NCT02008565). This trial compares the use of

loperamide to oral placebo, and the use of anal sphincter exercise training with biofeedback to usual care (educational pamphlet) in the treatment of FI.[73]

Anticholinergic medications such as hyoscyamine, if taken before meals, can be helpful in postprandial leakage. Amitriptyline has been shown to increase colon transit time by decreasing rectal contractions in patients with idiopathic FI.[68,69] Cholestyramine or colestipol can be helpful in patients with bile salt malabsorption.[47,68] Low dose clonidine, an α -adrenergic agent, reduces rectal sensation and urgency.[47] Phenylephrine gel applied directly to the sphincter was shown to increase IAS tone, possibly beneficial for patients with intact IAS but low resting pressure.[68,71,74] Localized dermatitis or burning sensation, although short-lasting, have been observed.[71]

Pelvic Muscle Exercises and Biofeedback

Pelvic muscle exercises and biofeedback improve FI symptoms by improving contraction of pelvic floor muscles, sensori-motor coordination required for continence, and enhancing the ability to perceive rectal distension.[75] Biofeedback is performed using visual, auditory, or verbal feedback techniques with an ARM or EMG probe inserted into the anorectum to display pressure changes.[14] More recent studies have found a difference between pelvic muscle exercises alone and exercises with biofeedback (the addition of a rectal balloon, electrical stimulation, or EMG), in favor of adding biofeedback.[76] Currently, there is no standardization of biofeedback treatment, likely contributing to the wide range of reported success rates of exercises $\pm$ biofeedback from 38 to as high as 100 %.[14,20,76] Severe FI, pudendal neuropathy, and underlying neurological problems have been associated with worse outcomes, but more beneficial in patients with urgency FI, and useful in post-sphincteroplasty or anal repair.[14,76] No study has reported a major difference in outcomes of biofeedback or pelvic muscle exercises compared to other methods for treatment of FI. [76] Nonetheless, the American College of Gastroenterology and AGA recognize biofeedback as safe and effective and recommend its use especially in patients with weak sphincters and/or impaired rectal sensation.[14,20] A recent NIH consensus statement concluded that biofeedback is effective in preventing and reversing pregnancy related FI for the first year after delivery, however, there is a lack of long-term benefits of biofeedback on prevention.[10]

Surgical Management

Sphincter Repair

Significant innovative changes in the surgical treatment of FI have recently emerged. Studies vary in the primary outcome measured (QOL, dichotomous continent/incontinent, number of leakage episodes) making the interpretation and comparative efficacy of results more problematic.

In general, surgery should be considered in selected patients who have failed conservative measures. In most patients with FI due to sphincter trauma, especially with a history of obstetric trauma, overlapping sphincter plication is effective, at least in the short-term. [14,77] If disrupted, a separate imbrication of the IAS may be undertaken.[14] Initial

symptom improvement has been seen in 70–80% of patients, however, the success rate deteriorates over time with long-term (≥ 5 years) success ranging 20 – 58%. [14,78] No patients remained completely continent to liquid and solid stool at 10 years. [14] The most common complication is wound infection, ranging from 6 to 35%. [78,79] Deep infection resulted in a poor outcome on long-term follow-up. [78,79] Other predictors of long-term failure include, advanced age at the time of repair, duration of FI symptoms, and pudendal neuropathy, although controversial. [78,79] Preoperative manometry and sonography results failed to predict success following sphincteroplasty. [14] Nonetheless, diagnosis based on imaging to confirm the presence of a sphincter defect is warranted. [78]

Specifically in obstetric women with third and fourth degree obstetric lacerations (OASIS), the results of randomized trials comparing overlapping vs end-to-end repair are conflicting. For immediate repair of obstetric sphincter laceration, where no scar is present, the two approaches do not differ in outcomes as supported by most studies. [80,81] However, according to the recent randomized trial of the 3-year outcomes by Farrell et al, end-to-end repair of complete third and fourth degree obstetric lacerations is associated with significantly lower rates of AI at 12 months. However, no differences were observed over 3 years. [82]

In patients with significant incontinence due to structural damage, neosphincter construction has been attempted, either from autologous gracilis muscle or an artificial bowel sphincter. A fast twitch skeletal muscle when stimulated can be transformed into a slow twitch muscle providing a sustained sphincter like response. The continuous stimulation is maintained by an implanted pacemaker (no longer available in the US). [14,83] Clinical success rates were between 38–90%, however, gracilis muscle transfer has drawbacks such as deterioration in effectiveness with time, a long learning curve for surgeons, and high morbidity. The revision rate due to major complications was up to 50%. [14]

Neuromodulation

Sacral Nerve Stimulation

Sacral nerve stimulation (SNS) was first introduced as a minimally invasive surgical option for refractory FI in 1995 in Europe. [84] In the US, Interstim® was approved by the FDA for treatment of refractory chronic FI in April 2011. A somato-sympathetic reflex pathway has been suggested as a mechanism of action to reduce colonic activity, increase both resting and squeeze pressures, and change rectal sensitivity or compliance. [85,86] Some studies have reported clinical improvement without changes in sphincter pressures. [86] The exact mechanism of action is still controversial. SNS improves FI symptoms in patients even with disrupted sphincters, including previously failed sphincteroplasty. The extent of sphincter gap differs in existing studies, but lesions up to 180° have been treated with SNS and outcome appears to be independent of size. [84] SNS is a staged procedure. After a 2–4 week period with a temporary percutaneous peripheral nerve electrode attached to an external stimulator, if a significant benefit is achieved, typically defined as at least a 50% reduction in FI episodes, the implantation of the definitive pulse generator can be undertaken. [87]

In the pivotal US multicenter trial, 90% of subjects proceeded from temporary to permanent stimulation.[88,89] This study was extended, and the long-term durability of SNS was published in 2013 reporting 36% complete continence and 89% therapeutic success at 5 years.[90] Other studies have demonstrated over 80% of patients achieving a 50% reduction in incontinence episodes per week with sustained long-term results (up to 14 years).[84,85,88,91] The most common adverse events are implant site pain and paresthesia (28%).[91] Pain is usually managed conservatively, and explanation of the device is rarely necessary.[91] The most serious complication is implant site infection, 3% (up to 10.8% reported).[91] Explanation or revision has been reported 4%, however less frequent with the newer electrodes.[91] The safety of SNS in pregnancy as well as risk to the unborn fetus, has not been established.

Percutaneous Tibial Nerve Stimulation

Percutaneous tibial nerve stimulation (PTNS), initially used in treatment of overactive bladder symptoms, is now gaining ground as a treatment for FI, but is not currently approved by the FDA. A 34-gauge needle electrode stimulates the posterior tibial nerve near the medial malleolus to achieve effects via L4-S3 nerve roots. Compared to Interstim®, PTNS requires repetitive treatments to maintain effectiveness. However, PTNS is a minimally invasive outpatient technique with almost no associated morbidity.[92] A success rate of up to 60% has been reported.[92] Most recently, the largest prospective study with 115 patients with a median follow-up of 26 months (range, 12–42) reported 52% of patients with FI demonstrated 50% reduction in FI episodes.[93] A multicenter study in the United Kingdom, the CONFIDENT trial, comparing the effectiveness of PTNS with sham electrical stimulation, is currently being undertaken to evaluate the efficacy and cost-effectiveness of PTNS in FI.[93] The ideal treatment protocol (interval and duration) has not been established. A potential disadvantage of PTNS includes frequent returns to the clinic. A randomized controlled trial comparing SNS and PTNS in the treatment for FI is currently being performed (NCT01069016).[93, 94]

Injectables

The treatment particularly associated with passive incontinence and IAS dysfunction remains challenging. Injection of a bulking agent to augment the closure of the proximal anal canal was first introduced in 1993.[95] Bulking agents into the submucosal or intersphincteric space augments the anal cushions. In theory, this provides a better seal of the anal canal lumen at rest, increases the length of the high-pressure zone, and improves canal symmetry by targeting IAS defects.[15,96] Ten materials have been introduced (Table 2).[96] The advantage of anal bulking is its simplicity and minimal invasiveness. Graf et al demonstrated the short-term efficacy of Solesta™ vs sham injection for FI. 52% in the treatment group had a 50% reduction in incontinence episodes compared to 32% in the control group at 6 months. The placebo effects demonstrated in the study are compatible with other trials and cannot be negated, as there was no difference between arms observed at 3 months.[95,96] The treatment response increased to 69% at 12 months in this study population. Treatment-related adverse events were mostly self-resolved. Of the 136 study patients, only 2 had serious adverse events (abscesses) requiring interventions.[95,96] The

American Society of Colon and Rectal Surgeons practice parameters for the treatment of FI state “when passive FI caused by internal sphincter dysfunction is the predominant symptom, injectable therapy seems to be effective and safe, although its long-term efficacy has yet to be defined.”[15] Consensus regarding the indications, the techniques (submucosal vs. intersphincteric, use of trans-anal ultrasound) or the optimal material are still under investigation.[96] While it may not offer complete resolution, anal bulking agents can alleviate symptoms in most patients especially with mild to moderate FI.[15]

Secca® Procedure

The Secca® procedure, an application of a temperature-controlled radiofrequency (RF) energy to the IAS, was approved by the FDA for treatment of refractory FI in 2002. RF-induced injury to the IAS is thought to cause collagen deposition and fibrosis, potentially tightening the affected anal canal.[97] A similar procedure has been used in the treatment of gastro-esophageal reflux disease and benign prostatic hypertrophy.[97] A five-year follow-up study published in 2008 showed 84% had 50% symptomatic improvement.[98] Other studies have shown similar positive results. However, none of the studies contained neither greater than 50 patients nor follow-up longer than 5 years.[98,99] There is short-term efficacy and safety data in a limited sample size.[99] Further study is needed to define the indication and long-term outcomes.

Colostomy

Diverting colostomy is performed if other treatments failed or unsuitable.[14,100] Colostomy should not be regarded as a failure. Improving QOL and amelioration of symptoms can be very rewarding. A laparoscopic approach can result in decreased morbidity.[14]

Investigational Treatment Options

Injection of autologous myoblasts (cultured from a pectoralis muscle biopsy) into the EAS defect may offer symptomatic improvement. Frudinger et al demonstrated an increase in squeeze pressures at 1 and 6 months post-injection in women with FI due to obstetric sphincter injuries.[101] However, the changes were not sustained at 12 months even though significant symptomatic improvement was noted among the subjects throughout the study period.[101] The use of autologous cells bypasses ethical concerns related to embryonic stem cells and avoids adverse events associated with foreign materials. A randomized control trial comparing autologous muscle-derived progenitor cell vs sham (saline solution) injection into the anal sphincter is being undertaken (NCT01523522).[102]

Traditionally, anal plugs, designed to temporarily occlude the anal canal, have been considered difficult to tolerate but useful for patients with impaired anal-rectal sensation and who are institutionalized or immobilized.[14,103] The REST study is the most recent multicenter, prospective, non-randomized trial to assess the efficacy, tolerability, and safety of a new single-use disposable device, Renew Insert, in moderate-to-severe FI patients (NCT01475474).[104] The study showed a significant reduction in both FI frequency and Wexner scores with high satisfaction and low adverse event rates.[105] Another device, the

LivSure intra-vaginal bowel control system, has been designed to provide bowel control for women with FI. The device is placed intra-vaginally, similar to a pessary, with an inflatable balloon oriented posteriorly and a hand-held patient pump. When the balloon is inflated, the vaginal wall occupies rectal space, preventing unwanted stool passage. Deflating the balloon allows stool passage. Typically, the vaginal insertion produces little or no sensation. The LIFE study is a trial for women with FI to evaluate the use of the investigational LivSure device, not yet received marketing approval/clearance from the FDA (NCT01655498).[106]

Conclusions

FI is a debilitating disorder which significantly impacts QOL. The etiology is often multifactorial and although most FI symptoms and conditions are treatable, many women remain untreated because they do not seek care. The evaluation and treatment of FI can also be hindered by a lack of understanding of the mechanisms and current knowledge on available options. In the past decade, research and development of diagnostic testing has allowed better understanding of this multifactorial disorder, and has yielded newer diagnostic tools and treatment options. Future efforts should focus on educational efforts to promote care seeking behavior and comparison of the efficacy of therapeutic modalities in well-designed controlled clinical trials.

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Table 1

Etiology of fecal incontinence

Anal	
•	Injury (obstetrical, anorectal surgery, accidental trauma)
•	Fistula
•	Rectal prolapse, Intussusception
•	Hemorrhoids
•	Anal carcinoma
•	Perianal infection
•	Congenital; (imperforate anus, rectal agenesis, cloacal agenesis, myelo/meningocele, spina bifida)
Rectal	
•	Proctitis (inflammatory bowel disease, radiation, infection)
•	Rectal carcinoma
•	Rectal Infection
Neurological	
•	Central Nervous System (Stroke, dementia, spinal cord injury, tumor, multiple sclerosis, cauda equina)
•	Peripheral Nervous System (Pudendal neuropathy, Diabetes mellitus)
Functional	
•	Fecal impaction
•	Diarrhea
•	Irritable bowel syndrome
•	Physical Disabilities
•	Psychiatric disorders
•	Metabolic, medication, malabsorption

Table 2**Anal Bulking Materials**

- Autologous fat
- Teflon
- Bovine glutaraldehyde cross-linked collagen (Contigen®)
- Carbon-coated zirconium beads (Durasphere®)
- Polydimethylsiloxane elastomer
- Dextranomer in non-animal stabilized hyaluronic acid (Solesta™)
- Hydrogel cross-linked with poly-acrylamide (Bioplastique®)
- Porcine dermal collagen (Permacol™)
- Synthetic calcium hydroxylapatite ceramic microspheres
- Polyacrylonitrile in cylinder form