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Management of fecal incontinence

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A B S T R A C T

Fecal incontinence is not a rare condition. In the majority of patients, no operative means result in symptom relief. Only if these fail surgical intervention is indicated. Various new surgical options have evolved over the last decades. The evidence of their efficacy varies substantially. The mainstays of surgical treatment of fecal incontinence are sphincteroplasty and sacral nerve stimulation. Data of other techniques, like posterior tibial nerve stimulation, radiofrequency energy delivery and bulking agents, are less robust. The article aims to outline the currently commonly accepted and frequently applied surgical techniques for treatment of fecal incontinence and their results and to present novel techniques, which carry potential for the future.

Definition/prevalence

Incontinence is a symptom. It ranges from occasional leakage while passing gas to complete loss of bowel control. It can be a secondary symptom to other pathologies of the colon, rectum and anus—such as tumors, inflammation—but it also can be due to functional deficits of the various components contributing to voluntary bowel control and emptying.

The true prevalence of FI is unknown. Overall, approximately 2% of the general population suffers from the inability to control bowel emptying.[1] The problem increases with age, with up to 11% of men and 26% in women after age of 50 years,[2] reaching up to 40% in nursing-home patients in whom urinary incontinence is frequently concomitant.[3]

Diagnostic and treatment considerations

The diagnosis of fecal incontinence (FI) is based on a standard anorectal examination that comprises inspection, palpation (including testing of perianal sensitivity and reflex activity), proctoscopy, rigid rectoscopy (to exclude pathologic conditions that may result in secondary incontinence), and a focused history. The last includes stool frequency, urge symptoms, incontinence for gas, liquid or solid stool, difficulties in passing stool, necessity of digital help when emptying, and day- and time-dependence of symptoms. Questions addressing aspects of evacuatory disorders may help to detect pathologic conditions such as rectocele, intussusception and enterocele, which frequently present with incontinence.[4]

Next to symptom alleviation, another main purpose of the treatment of FI is improvement in quality of life. Thus, the use of bowel-habit diaries, standardized questionnaires, and general and disease-specific quality-of-life (QoL) scores has become standard to document the symptoms in detail and to quantify the extent and severity of the disorder. This is done before, during, and after treatment. Interestingly, the correlation between symptom severity and quality of life is not linear, and thus both contribute to decision-making. The same instruments are used to monitor the clinical efficacy of interventions.

Most cases of incontinence can be treated with relatively simple pragmatic measures, and a commonly accepted principle is to begin with the simplest, least invasive treatment. Conservative options such as diet, medication, and retrograde irrigation can —without further diagnostic steps—be initiated to improve stool consistency and delayed colonic transit and/or to establish a normal periodicity to bowel emptying. If these fail or do not produce adequate symptom relief, further diagnostic procedures are indicated.

A range of diagnostic tools is available to identify morphologic and functional deficits of the various structures and functions contributing to the maintenance of continence. This is important to establish a meaningful therapeutic concept, as the cause of FI is multifactorial.

Comparable morphologic and functional lesions may result in clinical pictures of varying severity, as some deficits of components of the continence organ can partially be compensated for by other components. Anatomy and its potential defects can best be explored by imaging techniques, e.g. endoanal ultrasound and MRI. Whereas the former is widely available, relatively easy to

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perform, and considered an essential part of the initial diagnostic workup, MRI has limited availability and is considered part of an advanced diagnostic workup. Both help to differentiate muscular lesions from other causes.

Muscular function of the smooth-muscle internal anal sphincter and the striated-muscle external anal sphincter, perception of rectal filling and distension, compliance of the rectal reservoir, and the reflexive interaction of the rectum and anal sphincter can be tested and quantified by anorectal manometry. Electromyographic recording (EMG) of the striated muscles of the external anal sphincter and the pelvic floor permits differentiation of muscular from neurogenic defects and estimates the extent of reinnervation. Pudendal nerve terminal motor latency (PNTML) measures the conduction of peripheral nerves and helps to identify lesions, although questions of the relevance of its findings have recently caused it to be used less.

The following will focus on interventions directed to treat FI surgically. Some have achieved broad acceptance and are at present the mainstays of surgical therapy. Others are novel, not yet established, or under investigation.

Surgical treatment of anal sphincter insufficiency

Based on the diagnostic findings, two concepts for treating anal sphincter insufficiency can be distinguished.

(1) Morphologic reconstruction is indicated if identified morphologic defects have functional relevance. The aim is to reconstruct anatomy and thus reestablish function.
(2) Functional rehabilitation is indicated if no morphologic defects are identified. This aims to recruit residual function of the anorectal continence organ.

Sphincteroplasty

Sphincteroplasty describes a secondary or delayed reconstruction of the anal sphincter musculature, in either initially unrecognized or initially functionally irrelevant lesions, or if the outcome of primary repair has been unsatisfactory. The term sphincter repair is used to describe primary repair of the anal sphincter mechanism immediately after direct trauma. The indication for both procedures is FI owing to disrupted anal sphincter anatomy. In both, direct sphincter reconstruction aims to reestablish function by closing a morphologic defect by coaptation of the dehiscent muscle. The result may be a more rigid encircling of the anal canal, which usually is less elastic. Technically this can be done by adaption or overlap; functional outcome appears to be comparable.[5] The separate identification and repair of the internal anal sphincter is technically challenging and is of unproven therapeutic effect.

Sphincteroplasty can be combined with levatorplasty—an adaption of both levator ani muscles; however, care must be taken to avoid vaginal narrowing and dyspareunia. The addition of a biological implant may be advantageous to reinforce the anal muscles.[6]

The most common reason for sphincter repair and sphincteroplasty is obstetric trauma during childbirth, and thus anterior lesions are the most frequent. In colorectal surgery, injury is the result of blunt or penetrating trauma.

Despite the fact that the results of anal sphincteroplasty are not reported uniformly, outcomes appear to be comparable with approximately 50% of patients reporting a significant improvement in continence (Table 1). However, short-term outcome is not sustained, and it is common for function to deteriorate over time.[7]

Table 1
Sphincteroplasty: studies > 70 patients.

Study	Patients	Follow-up (months)	Continence % (excellent/good)
Londono-Schimmer et al.[52]	94	60	50
Gilliland et al.[12]	77	24†	55
Karoui et al.[53]	74	40	47
Halverson et al.[54]	71	27	41
Bravo Gutierrez et al.[55,+]	130	120	6
Norderval et al.[56]	71	27	41
Zorcolo et al.[57]	93	70†	55
Trowbridge et al.[58]	86	67	11
Oom et al.[10]	120	111†	38
Gleason et al.[59]	74	32	77
Madoff [60,#]	891		66

[#] Metanalyse.
[+] 130/190 available for 10 years follow-up.
[†] Median, otherwise mean. (Adapted with permission from Madoff et al.[17])

Nevertheless, a substantial proportion of patients remains satisfied: patient satisfaction 7 years after overlapping anterior sphincter repair reaches up to 84% despite a functional improvement in only 48%.[8]

Multiple factors have been studied as potential predictors of success. Although data are not fully conclusive and reproductible, poorer outcome was found to be associated with age $\geq$ 50 years,[9] deep wound infection, and isolated external anal sphincter defects.[10] Preoperative manometric variables do not predict outcome.[11] Coexisting uni- or bilateral neurogenic damage (measured by PNTML) has repeatedly been discussed as a predictor of lower success, but this remains controversial and is not considered contraindicative.[12,13]

If sphincteroplasty fails to achieve symptom improvement, or if function deteriorates over time, patients can be considered for functional rehabilitation, such as biofeedback and irrigation. If deterioration owes to breakdown of the reconstructed anal sphincter, repeat sphincteroplasty can be considered. Reported outcomes are similar to patients without previous sphincteroplasty, with good results reported in 50% and 58% of patients, respectively[14] and the long-term benefit is similar.[15] However, these findings raise doubts and need to be confirmed.[16] Repeat sphincteroplasty should be elected after other modalities have been explored.[17]

Lately, increasing evidence has indicated that sacral nerve stimulation may also be a treatment option for patients with sphincter defects after attempted anatomic reconstruction (and also as a possible first-line treatment for these indications).[18–20]

Sacral nerve stimulation/sacral neuromodulation

Sacral nerve stimulation (SNS), also termed sacral neuromodulation (SNM), aims to recruit residual function of the anorectal continence organ. In the last decade, the chronic low-frequency stimulation of the sacral spinal nerves (the peripheral nerve supply of the anorectum) gained broad acceptance owing to its efficacy, sustainability, limited invasiveness, and low comorbidity. Its role in the current treatment algorithm became (Fig. 1).[17,21]

No clinical or physiologic predictor of success of chronic stimulation exists, and thus, decision making for implantation of a permanent device is based solely on the outcome of temporary test stimulation, usually of 2 weeks' duration. Prerequisites for the test stimulation are residual sphincter function, an existing neuromuscular connection to the sphincter (tested by observation of voluntary squeeze or reflex activity after pinprick), and accessibility of the target sacral spinal nerves S3 and S4. Thus, the spectrum of