Committee 17

Surgery for Faecal Incontinence

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.

CONTENTS

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INTRODUCTION

Therapy for faecal incontinence is readily divided into non-surgical and surgical therapy. Selection of specific therapy is based upon a number of considerations, including the severity of incontinence and structural integrity of the anal sphincter.

Conservative therapy is most applicable to relatively mild cases of incontinence. Biofeedback retraining can be attempted for incontinence of any cause or severity, as the therapy is painless and risk-free. These treatments are discussed in detail elsewhere in this monograph.

The most widely accepted surgical therapy for faecal incontinence is overlapping sphincteroplasty. Typical of other well-established therapies, the evidence base supporting this approach is paradoxically less robust than that supporting more recent treatment options. Sphincteroplasty is useful only in cases in which there is an anatomic sphincter defect, and it has been reported to provide satisfactory results in many case series. However, several recent studies have now shown that results of sphincteroplasty deteriorate with time [1, 2].

A number of operations were developed in the early to mid 20th century to provide a treatment option for patients whose native sphincter was either intact but weak or not reparable. Muscle transposition procedures using either gluteus maximus or gracilis were devised to create a functional biological neosphincter, but the approach did not gain widespread popularity. The Parks postanal repair was devised in 1975 to treat patients with incontinence due to pelvic neuropathy [3].

Dissatisfaction with available operations for faecal incontinence led to development of a variety of novel procedures during the last 20 years. The stimulated (dynamic) graciloplasty and the artificial anal sphincter were devised as salvage procedures for patients who had failed or were not candidates for standard therapy. A more recent approach is the use of sacral nerve stimulation, which was adopted for this purpose from its previously better-defined role in urinary voiding dysfunction. Moreover, there has also been a trend towards development of minimally invasive approaches to faecal incontinence, such as the use of injectable biomaterials.

Several important caveats apply to interpretation of the results of surgery for faecal incontinence reported in the literature. First, the vast majority of reports are uncontrolled case series. Randomized controlled studies are rare, and those reported include only small numbers of patients [4]. Second, numerous quantitative measures have been used to report outcomes, but only recently have any of these been validated, such as the Faecal Incontinence Quality of Life (FIQL) instrument. Third, criteria for "successful" outcomes have been variable and often arbitrary. Fourth, the quality of data reported is variable, though it has generally improved with the passage of time. Chart review has been supplanted by patient questionnaires and interviews by independent data auditors; daily continence diaries, the most stringent form of data collection, have become increasingly commonplace (though not routine). Despite the fact that studies using lax data collection are certain to report better results than those using methodology that is more stringent, of necessity, composite reviews of surgical results include studies using various methods of data collection. Finally, results are not always reported on an intention to treat basis, particularly in the implantable device literature.

SEARCH METHODS

PubMed search was conducted to identify studies published on the use of surgery for faecal incontinence in children and adults. Keywords used were faecal incontinence and surgery. Full text copies of studies deemed to be potentially relevant were obtained. Priority was given to systematic reviews, randomized controlled trials, and controlled clinical trials; if those were unavailable or inadequate, comparative observational studies, case series, case reports and narrative reviews were also included. Reviewers were not blinded to the names of studies' authors, institutions or publications. In view of the nature of the guideline, priority was given to the reports with large number of

patients and long follow-ups assessing efficacy of surgical interventions. Particular emphasis was placed on those reporting techniques and functional outcome including quality of life after an operation.

Non-English language papers were noted but excluded from the review unless they contained an English-language abstract providing sufficient information.

A. SURGERY FOR ADULT FAECAL INCONTINENCE

I. SPHINCTER REPAIR

Anal sphincter repair is the term used to describe primary repair of the anal sphincter mechanism following direct trauma. The most common indication is following childbirth and repair in this situation is usually performed by the attending obstetrician. Colorectal surgeons are more commonly involved in primary repair of injury that is the result of blunt or penetrating trauma. Occasionally, the anal sphincter mechanism is damaged during anorectal surgery for other pathology particularly surgery for anal fistula.

In Western obstetric practice, the incidence of overt anal sphincter injury (grade 3 or 4 tear) is low, 3- 5% following primiparous delivery and 0.5-1% following second and subsequent deliveries [5]. When prospectively looked for with endoanal ultrasound, the incidence of anal sphincter injury is higher [6, 7]. A meta-analysis of 717 vaginal deliveries found an incidence of new anal sphincter defects of 27% in primiparous and 9% in multiparous women using 2D endo-anal ultrasound [8]. 3D ultrasonography suggests that the incidence is somewhat less, perhaps 11%, following primiparous delivery [9]. The risk factors for sphincter injury include instrumental vaginal delivery, prolonged second stage of labor, fetal macrosomia, and a persistent occipto-position of the fetal head [7, 10-12]. Midline episiotomy is associated with higher incidence of anal sphincter injury and the angle of mediolateral episiotomy may also influence perineal outcome [13]. A policy of restrictive use of episiotomy may reduce the incidence of anal sphincter injury [14].

Obstetric injury of the perineum is classified as a first degree tear if confined to vaginal epithelium and skin, second degree if the perineal muscles are torn, third degree if the anal sphincter muscles (external: EAS; internal: IAS) are torn, (3a: less than 50% EAS torn; 3b: more than 50% EAS torn; 3c: IAS torn) or fourth degree if both EAS and IAS and rectal or anal mucosa are torn [15]. Primary repair of an obstetrical tear is correctly termed anal sphincter repair and is usually performed by the obstetrician immediately after delivery most commonly in the delivery room under local or epidural anesthetic. By tradition, the technique of repair has been a direct oppositional repair of the severed external anal sphincter. The internal anal sphincter, if divided, is difficult to identify separately and when separately repaired it is usually *en block* with the anal canal mucosa in a complete or 4th degree tear.

There have been four randomized clinical trials [16-19] and one meta-analysis [20] that have investigated different techniques of immediate primary repair of the external anal sphincter following obstetric injury. There was a trend towards better outcome with an overlap repair; however, the meta-analysis concluded that it would be inappropriate to favor one type of repair over another [20]. **[LEVEL OF EVIDENCE: 1]** With regard to management of the internal anal sphincter, Mahoney et al [21] have shown persistence of an IAS defect to be adversely associated with continence outcome in a series of 500 consecutive women assessed following repair of a 3rd or 4th degree tear.

It has been suggested that primary anal sphincter repair might be best performed by a colorectal surgeon rather than an obstetrician [22]. Nordenstam et al [23] concluded, in a single institution study of 156 women, that technique and expertise impact on the outcome of primary repair and that if needed, the repair could be safely delayed until such expertise was available. Soerensen et al [24] have found no adverse outcome with delayed primary repair. **[LEVEL OF EVIDENCE: 2]**

There have been two randomized trials of post-operative management of the bowel after primary anal sphincter repair. These have shown benefit in use of a laxative rather than a constipating regimen but no advantage to the addition of a stool bulking agent [25, 26]. **[LEVEL OF EVIDENCE: 1]**

Alteration in faecal continence occurs in approximately 13 - 17% of women following primiparous vaginal delivery [6, 27, 28]. The prevalence is greater if urgency of defecation is included as a symptom [7]. Incontinence to flatus has been reported in up to 27% of 7,879 women surveyed 12 weeks after delivery [29]. **[LEVEL OF EVIDENCE: 2]** The prevalence is significantly higher in women who have undergone anal sphincter repair. Fenner et al [30] found that women who had sustained third and fourth degree tears were more likely to have bowel incontinence than women without anal sphincter injury 6 months following delivery. This was more pronounced in women with a history of 4th degree tear. Mahoney et al [21] studied 500 consecutive women after repair of a recognized 3rd or 4th degree tear and found some alteration in continence in 50% at 3 months post partum. The median Cleveland Clinic Continence score [31] in this cohort was 2 (range 0-19) and 4.4%

had a score >9, a score deemed to be socially disruptive [32]. **[LEVEL OF EVIDENCE: 2]**

Management of subsequent labor following a previous anal sphincter tear must take account of obstetric risk factors, symptoms of incontinence and patient preferences. Harkin et al [12] found an approximately 5 fold increase in the incidence of recurrent sphincter tear compared to the incidence of first sphincter injury during second labor. Fynes et al [33] found that women with altered continence after first vaginal delivery were at risk of deterioration if delivered vaginally on their second pregnancy. Caesarian delivery before the onset of the second stage of labor was found to be protective [33]; however, in a systematic review, Nelson et al [34] found that pregnancy rather than delivery was a more important indicator of post partum continence. **[LEVEL OF EVIDENCE: 3]**

A number of studies have looked at long term outcomes after repair of a 3rd or 4th degree tears and all have shown an increasing prevalence of continence disorders with age. These findings parallel those of the general population of parous women who have not had a recognized tear [35-37]. Eogan et al [38] found in a study of women 10, 20 and 30 years following delivery that onset of menopause was the most significant determinant of symptoms, whereas Mous et al [39] found the incidence of incontinence increased with age irrespective of menopausal status. Fornell et al [40] found that subjective and objective anal function after an sphincter injury deteriorates with time and subsequent deliveries. A persistent defect in the internal anal sphincter was found to be an important determinant, an observation supported by Mahony et al [21]. **[LEVEL OF EVIDENCE: 3]**

II. SPHINCTEROPLASTY

The term anal sphincteroplasty is used to describe secondary or delayed reconstruction of the anal sphincter musculature, injury to which has either not been recognized at the time of injury or the outcome of primary repair has been unsatisfactory. Anterior sphincteroplasty is the most common type of reconstruction performed because of the association with obstetric injury. In this situation, the anal sphincter muscles and perineal body have separated, leaving a large defect in the anterior quadrant with horseshoe type configuration to the anal sphincter mechanism. Occasionally, the defect is such that the anal and vaginal mucosa have healed to form a cloacal defect. Anal sphincter defects related to previous anal fistula surgery or direct trauma are usually less complex and are not associated with a deficient perineum. The results of sphincteroplasty are shown in **Table 1** [1, 2, 41-56].

Table 1. Published results of anal sphincteroplasty since 1990, including series with 50 or more

Authors (ref)	Year	Number of patients	Follow-up (months)	Continent % (excellent / good)
Fleshman et al [41]	1991	55	12	72
Engel et al [42]	1994	55	15	79
Londono-Schimmer et al [43]	1994	94	60	50
Oliveira et al [44]	1996	55	29*	71
Gilliland et al [45]	1998	77	24*	55$
Young et al [46]	1998	54	18*	86$
Malouf et al [1]	2000	55	77	49
Karoui et al [47]	2000	74	40	47
Osterberg et al [48]	2000	51	12	58
Morren et al [49]	2001	55	40	56
Tan et al [50]	2001	50	28	50
Halverson and Hull [2]	2002	71	69	25
Bravo Gutierrez et al [51]	2004	130+	120	6
Norderval et al [52]	2005	71	27	41
Zorcolo et al [53]	2005	93	70*	55
Trowbridge et al [54]	2006	86	67	11
Barisic et al [55]	2006	65	80*	48
Madoff [56]	2004	891		66

metanalysis * Median follow-up + 130/190 available for 10 year follow-up $ defined as “successful”

The decision to perform anal sphincteroplasty is a function of symptoms and the anatomical extent of the sphincter defect [56]. In assessing symptoms, one of several continence scores should be used [31, 57, 58]. The two most commonly applied are the Cleveland Clinic Continence Score [31] and the St Mark's Continence Score [59]. In addition, a quality of life instrument should be applied [32]. Endoanal ultrasound is helpful in defining the extent of anal sphincter injury. 3D endoanal ultrasonography may provide further information [9]. Pelvic floor assessment using fMRI [60] or multiple contrast defecating proctography [61] is valuable in the assessment of a more global pelvic floor injury. **[LEVEL OF EVIDENCE: 4]**

Other causes of disordered continence should be excluded, e.g., inflammatory bowel disease, colorectal cancer and neurological lesions. Patients with background IBS are more likely to be symptomatic than those more predictable bowel habit and equivalent anal sphincter defects [62]. Pelvic floor electrophysiological assessment, while not essential, should be comprehensive and not confined to measurement of pudendal nerve terminal motor latency [63].

For symptomatic patients with a less than one quadrant anal sphincter defect, a trial of dietary modification, stool regulating drugs and physiotherapy is appropriate. There are limited data regarding the role of biofeedback with or without electrical augmentation [64, 65]; however, a recent Cochrane review concluded there were insufficient data to allow definitive assessment [66].

For patients with a more than one quadrant anal sphincter defect, anal sphincteroplasty is appropriate [4, 15, 56]. Preoperative counseling should identify post operative wound healing as the most common difficulty. The majority of patients can expect significant improvement in continence after the procedure, with a mean of 66% reporting excellent or good results in the short term [56]. **[LEVEL OF EVIDENCE: 3]** Concomitant repair of a cloacal defect or vaginal fistula should be undertaken [67, 68]. There is no evidence that a defunctioning colostomy improves outcome.

Anal sphincteroplasty can be performed in the lithotomy or in the prone jack-knife position. Full bowel preparation is often performed but not mandatory, although most surgeons at a minumum would give a cleansing enema pre-operatively. The conventional incision is an inverted 'V' that may be closed as an inverted 'Y' as described by Parks [69]. If anterior levatorplasty and particularly if rectocele repair is contemplated, then a posterior fourchette incision with the patient in lithotomy may have advantages [50]. The external anal sphincter is usually repaired using an overlapping technique without separate identification and repair of the internal anal sphincter [15]. There has been one small randomized trial of direct versus overlapping sphincteroplasty which showed similar outcomes [70].

Initial success of sphincteroplasty is related to whether the anal sphincter defect is corrected [42, 71]. Early failure is usually associated with a persisting defect, identifiable using endoanal ultrasound [72]. This may be amenable to a further attempt at repair [71, 73, 74]. There is, however, increasing evidence that continence outcomes deteriorate with long-term follow-up [15, 56]. In the largest study reported to date, Bravo Gutierrez et al [51] found that only 6% of patients retained full continence 10 years following anal sphincteroplasty. The effect of age at time of operation on long-term function is controversial [75, 76]; however, long-term atrophy of the sphincters may be relevant [15]. **[LEVEL OF EVIDENCE: 2]**

Pre-operative physiologic testing may be helpful in the overall management of patients with faecal incontinence. However, the value of anal manometry and pelvic floor electrophysiological assessment as prognostic indicators for outcome following sphincteroplasty is controversial. There are no established parameters that reliably predict outcome following sphincteroplasty [77, 78].

III. POSTANAL REPAIR

Postanal repair was first reported by Sir Alan Parks in 1975 [3]. This procedure was designed to increase the length of the anal canal, restore the anorectal angle and re-create the flap valve mechanism, which at the time was thought essential for maintaining faecal continence. Success rates ranged from 15% to 83%, depending on the definition of the success, the length of follow-up, and possibly the cause of incontinence. The published studies regarding postanal repair include two systematic reviews of randomized controlled trials (level 1) [4, 79], two randomized controlled trials (level 1 [80] and 2 [81]), two non-randomized cohort studies (level 2) [82, 83], 8 case series of good quality (level 3) [84-91] and 10 case series of poor quality (level 4) [3, 92-100]. The results of postanal repair are shown in **Table 2** [3, 80-100].

Subsequent observational studies with a median follow-up of more than 5 years revealed that continence deteriorated with time. Despite 60% to 80% of patients reporting persisting improvement, only one-third were actually continent to liquid or solid stool [86, 89, 100]. Even in the most recent study reporting the "long-term" outcome of postanal repair [91], only 23% were continent to liquid or solid stool, while 68% improved symptomatically with a median follow-up of 3 years. Possible explanations for deterioration of continence following initial improvement included unrecognized denervation and/or muscular injury of the sphincter and pelvic floor musculature,

Table 2. Postanal Repair for Faecal Incontinence

Authors (ref)	Year	Number of patients (female)	Median or mean follow-up: months (range)		Outcomes	
					Continent to solid and liquid (%)	Symptomatic improvement
Parks [3]	1975	75 (68)	ns	(180 or less)	83%	ns
Browning and Parks [84]	1983	42 (36)	ns	(1or less)	81%	ns
Keighley [92]	1984	89 (ns)	ns	(6 or more)	63%	84%
Ferguson [93]	1984	9 (8)	ns	(ns)	67%	ns
van Vroonhoven and Schouten [94]	1984	16	ns	ns	63%	75%
Henry and Simson [95]	1985	242 (193)	11	(0.5 - 27)	60%	ns
Habr-Gama et al [96]	1986	42 (39)	12	(12)	52%	ns
Womack et al [82]	1988	16 (14)	26	(15 or more)	38%	88%
Scheuer et al [85]	1989	39 (ns)	ns	(ns)	15%	70%
Yoshioka and Keighley [86]	1989	116 (?)	60	(ns)	24%	81%
Rainey et al [97]	1990	42 (37)	42	(6 - 95)	31%	71%
Scott et al [98]	1990	62 (56)	ns	(ns)	45%	82%
Laurberg et al [99]	1990	28 (28)	ns	(ns)	32%	75%
Orrom et al [83]	1991	17 (ns)	15	(ns)	59%	ns
Deen et al [80]	1993	PAR: 12 (12)	24	(22 - 28)	42%	42%
		ALP: 12 (12)	22		33%	50%
		TPFR: 12 (12)	28		67%	83%
Engel et al [87]	1994	38 (34)	43	(15 - 126)	21%	50%
Jameson et al [88]	1994	36 (33)	6	(6)	50%	83%
			25	(6 - 72)	28%	53%
Setti-Carraro et al [89]	1994	34 (34)	73	(61 - 95)	26%	82%
Rieger et al [100]	1997	19 (ns)	96	(24 - 120)	37%	58%
van Tets et al [81]	1998	PAR: 11 (11)	3	(3)	27%	45%
		TPFR: 9 (9)	3	(3)	22%	33%
Matsuoka et al [90]	2000	20 (20)	36	(12 - 90)	35%	35%
Abbas et al [91]	2005	44 (44)	36	(24 - 216)	23%	68%

and the presence of occult anal sphincter disruption, particularly in the studies reported before endoanal ultrasonography or magnetic resonance imaging were available. Moreover, physiological and radiological evaluations before and after postanal repair have not demonstrated consistent changes in anal canal length, resting pressure, voluntary contraction pressure, anorectal sensitivity and movement of the anorectal angle [81-84, 101]. These reports of increasingly poor outcomes have diminished the popularity of this procedure significantly. **[LEVEL OF EVIDENCE: 3]**

Deen et al [80] in a randomized controlled trial comparing three procedures in 36 women with neuropathic faecal incontinence, found that complete continence was achieved in 42% of patients after postanal repair, 33% after anterior levatorplasty, and 67% after total pelvic floor repair. In contrast, van Tets et al [81] conducted a randomized controlled trial comparing postanal repair and total pelvic floor repair in 20 women with neurogenic faecal incontinence. Complete continence to solid or liquid stool was achieved in 27% of patients after postanal repair and in 22% after total pelvic floor repair.

IV. NON-STIMULATED MUSCLE TRANSPOSITION

A variety of muscle transposition procedures have been devised for the treatment of faecal incontinence. Early efforts focused upon the use of transposed skeletal muscle to supplement the function of a weak or disrupted anal sphincter. Early in the 20th century, a number of surgeons utilized gluteus maximus muscle, transposed in a variety of configurations, to create a neosphincter [102, 103]. In 1952, Pickrell et al [104] described the use of transposed gracilis muscle to create a neosphincter for incontinent children.

Published series of gracilis transposition are uncontrolled and demonstrate variable success rates [105-114]. **[LEVEL OF EVIDENCE: 3]** One study reviewed the functional results of graciloplasty longitudinally in 22 patients followed for a median 63 months [115]. 18 patients (81%) were improved at 6 months, though only one regained normal continence. Results deteriorated in 5 patients during subsequent follow up. Bilateral gracilis transposition has been used successfully in several small series [106, 116].

Success rates following gluteus transposition have likewise been variable [117-121]. **[LEVEL OF EVIDENCE: 3]** A prospective randomized trial in women with post-obstetric neuropathic incontinence showed similar significant degrees of improvement following both gluteus maximus transposition and total pelvic floor repair [122]. A recent retrospective review of 25 gluteoplasty patients reported restoration of continence in 18 patients (72%) and partial restoration in an additional 4 patients (16%). Donor-site and peri-rectal complications occurred in 16 patients (64%) [123].

V. STIMULATED MUSCLE TRANSPOSITION

The transposition of the gracilis muscle to reconstruct the anal sphincter was first performed in children in 1952 [104]. The blood supply is primarily from a single proximal artery that allows excellent mobility for transposition [124]. Successful electrical stimulation of a previously transposed gracilis muscle was first reported in 1988 [125], and case series from 2 independent centers were simultaneously reported in 1991 [126, 127]. Baeten et al [126] showed improved continence in 8 of 10 patients; Williams et al [127] in 12 of 20.

Even after successful muscle transposition, functional outcomes are limited by two physiological factors. First, patients are unable to consciously maintain tonic contraction of their neosphincters over long periods of time. Furthermore, even if patient volition were not a problem, gracilis muscle is poorly suited to tonic contraction. While the external anal sphincter comprises predominantly slow-twitch, fatigue-resistant type I fibers, the gracilis muscle comprises predominantly type II, fast-twitch fibers that are rapidly fatigable [128]. Graded electrical stimulation transforms type II into type I muscle fibers [129], and use of an implantable electrical pulse generator has been shown to convert transposed gracilis to a muscle with predominantly type I fibers [126-128]. The gracilis muscle is well suited to electrical stimulation due to the relatively constant proximal location of the neurovascular bundle, which is easily identified at surgery [130].

The results of stimulated graciloplasty are shown in **Table 3** [131-141]. **[LEVEL OF EVIDENCE: 2]** In 1995, Baeten reported his results in 52 patients, with 38 (72%) becoming continent after surgery [131]. In a subsequent paper by this group published in 2003, 200 patients followed for a median of 261 weeks were reported [139]. The overall success rate was 72%. Patients with incontinence due to trauma had the best results (82% success), while patients with incontinence due to congenital anorectal malformation had the worst results (52% success). 138 complications were reported, including disturbed evacuation in 32 patients (16%), infection in 24 (12%), pain in 16 (8%) and pulse generator displacement in 12 (6%). Ten patients (5%) had anorectal perforations, 7 of whom eventually obtained a successful outcome. Rosen et al [142] reported restoration of continence in 9 of 10 patients treated by dynamic graciloplasty using a “split-sling” wrap configuration. Sielezneff et al [143] treated 16

Table 3. Dynamic Graciloplasty: General measures of continence

Authors (ref)	Year	Number of patients	Follow-up	Percentage continent*
Baeten et al [131]	1995	52	25.2 months (mean)	73
Geerdes et al [132]	1996	67	32.4 months (mean)	78
Cavina et al [133]	1998	31	37.8 months (mean)	85
Madoff et al [134]	1999	131	24 months (median)	66
Mander et al [135]	1999	64	16 months (median)	69
Baeten et al [136]	2000	123	23 months (mean)	74
Wexner et al [138]	2002	83	24 months	53
Rongen et al [139]	2003	200	16.3 months (median)	72
Pennickx et al [140]	2004	60	48 months (median)	55
Tillin et al [141]	2006	49	43 months (median)	70

* variable definitions; does not necessarily denote perfect continence. Issues of divergence in technique arose from these studies, each of which has seen increasing consensus in the literature despite a lack of randomized trial data. Thus, intramuscular (vs. epineural) electrodes are now universally employed, and diverting stomas and 'vascular delay' prior to muscle transposition are no longer utilized.

patients and 13 had improved continence. However, 8 patients suffered morbidity, resulting in 33 subsequent admissions and 23 reoperations.

Three multicenter prospective trials of dynamic muscle plasty have been performed to date [134-136]. In each of these studies, patients served as their own controls. No randomized prospective trials have been performed.

Madoff et al studied 139 patients from 12 centers, 128 of whom had gracilis wraps and 11 gluteus wraps. [134] Of those patients, 104 were treated for faecal incontinence, and 35 underwent total anorectal reconstruction following abdominoperineal resection for cancer. Success rates for graciloplasty were 71% for patients with acquired incontinence and 50% for those with incontinence due to a congenital abnormality. There were a total of 138 complications for the entire group. Wound complications (41 major and 35 minor) were both the most prevalent and the most consequential. Other complications included pain in 28 patients (22%), hardware problems in 14 (11%) and tendon detachment in 4 (3%). Centers with significant prior experience with the procedure had substantially fewer major wound complications (17.4 vs. 33.1%) and significantly higher success rates (80% vs. 47%).

Mander et al [135] reported the results of dynamic graciloplasty in 64 patients with refractory faecal incontinence treated at 7 centers. There were 24 infectious complications, 5 of which involved perineal wound breakdown and 3 of which required reoperation. 44 (69%) patients became continent to solid stool 1 month following stoma closure. Evacuation problems developed in 16 patients (25%), and this lead to failure in 14. At a median of 10 months follow-up, 29 patients had a good functional result.

Baeten et al [136] reported the results of dynamic graciloplasty in 123 patients treated at 20 centers as part of the Dynamic Graciloplasty Therapy Study Group (DGTSG). The aims of this study were to assess both the safety and efficacy of this treatment; 189 adverse events occurred in 91 patients, including one death due to pulmonary embolism. There were 18 major and 31 minor infectious complications. There were 42 instances of therapy-associated pain, occurring variably in the donor leg, at the anal canal, or at the device site. There were 11 lead dislodgements but no problems with lead breakage or pulse generator malfunction. A follow-up study showed full or partial recovery from these complications in 87% of patients. [137] This study, in contrast to others, was based upon data from daily continence diaries. A successful result (defined as a 50% or greater decrease in incontinent events in patients without pre-existing stomas) was achieved in 63% of patients after one year. Another follow-up of this patient cohort demonstrated stable success rates at 18 months (55%) and 24 months (56%) [138]. Statistically significant improvements in the physical and social function scales of the SF-36 were also recorded at 12 months.

A multicenter retrospective trial from Belgium using dynamic graciloplasty treated 60 patients with 27 failures [140]. Continence was achieved in 78% of the group. However, more than half (26 patients) required the use of antegrade continence enemas or other measures to maintain continence. Seven patients had a permanent stoma constructed. Seventy-five complications occurred with 61 total reoperations. Loss of muscle stimulation occurred in 22 patients; 10 were due to issues specific to the stimulator and leads, 4 were due to technical failure of the muscle wrap. Functional outcome was directly associated with a

maintenance of stimulation and initiation of stimulation within 50 days of surgery.

Very few studies have examined the long term results with dynamic muscle wraps. Thornton et al [144] reported on the 5-year follow up of 38 patients who had undergone dynamic graciloplasty. Of the 33 patients available for follow-up by telephone interview, obstructive defecation was a problem for 11% of the cohort and 16% had been converted to a permanent colostomy. Of those with a functioning graciloplasty (22 patients) who reported a faecal incontinence score of less than 12 (range 0-24), 50% reported problems with obstructive defecation and 64% felt their bowel habits had negatively impacted their quality of life. Long-term complications were primarily related to stimulator issues; ten patients required 15 operations to replace stimulator components. However, 72% of patients reported pain, swelling or paresthesias of the donor leg and 27% reported sexual dysfunction.

Tillin et al [141] performed a prospective case-comparison study of 49 patients who had a dynamic graciloplasty and 87 patients who either refused the surgery or were not offered the surgery. The primary outcomes evaluated were symptoms, quality of life, anxiety, and depression. Of the treated group, the procedure failed completely in 15 patients. At two year follow-up, two-thirds of patients were either never or rarely incontinent to liquid or solid stool. Up to 50% of patients with a satisfactory outcome reported disordered evacuation and 8 other patients were deemed failures due to this problem. In comparison to the 87 patients who did not undergo treatment, there were significantly more patients in the dynamic graciloplasty group who reported a greater than 20% improvement in their incontinence scores. However, the treated group also had a significantly worse pain as assessed on a validated pain scale.

Chapman et al [145] performed a systematic review of dynamic graciloplasty for faecal incontinence on behalf of the Australian Safety and Efficacy Register of New Interventional Procedures- Surgical (ASERNIP-S). The authors reviewed 37 original articles published between 1991 and October 2000. All of the papers were judged to be of low-evidence quality, as all but one paper were case series, and the sole comparative study utilized historical controls. Mortality excluding cancer deaths was 1% (95% confidence interval 1-3%) and morbidity 1.12 (95% CI 0.14 - 2.08) events per patient. Success was variably defined between studies, but was reported as ranging from 42-58%. The ASERNIP-S Review Group determined that “the safety of the procedure cannot be determined at the present time due to an incomplete and/or poor-quality evidence base” and that “efficacy is established.” Tan et al [146] examined three treatments for faecal incontinence including dynamic graciloplasty, artificial bowel sphincter and end stoma. They concluded that the most cost effective intervention was an end stoma, the artificial bowel sphincter was most cost-effective after 10 years and that dynamic graciloplasty should only be considered as an alternative in highly specialized centers.

VI. ARTIFICIAL ANAL SPHINCTER

Artificial sphincters have been used for the treatment of urinary incontinence since 1973 [147]. A success rate of 79% with a mean follow-up of 7.2 years has been reported. The device (AMS Sphincter 800® Urinary Control System, American Medical Systems, Minnesota, USA) and its subsequent modifications is a totally implantable system consisting of 3 parts: an inflatable occlusive cuff that is implanted around the native sphincter, a pressure-regulating balloon that is implanted in the prevesical space, and a control pump that is implanted in the labia majora or the scrotum. In 1987, Christiansen & Lorentzen [148] applied this device to a patient with faecal incontinence. The patient had an excellent result with no complications at a follow-up of three months.

Early promising results [149] prompted the modifications of the AMS Sphincter 800®, which eventually culminated in the development of Acticon Neosphincter® (American Medical Systems, Minneapolis, USA) that was specifically designed for faecal incontinence and became available in May 1996.

The published studies evaluating the safety and effectiveness of the newest sphincter system (Acticon Neosphincter®) include one randomized controlled trial (level 1) [150], one non-randomized cohort case control study (level 2) [151], 9 non-randomized cohort studies (level 2) [152-160], three systematic reviews of various types of studies with some heterogeneity (level 3) [161-163], one retrospective case control study (level 3) [164], 3 case series of good quality (level 3) [165-167], 4 case series of low quality (level 4) [168-171], and 3 case reports (level 4) [172-174]. The results of these studies except for case reports are shown in **Table 4** [150-160, 164-171]. Two studies by Romano et al [175, 176] and two case reports [177, 178] were excluded from the analysis because artificial anal sphincters in those studies were not implanted for faecal incontinence, but as a part of total anorectal reconstruction in patients who had abdominoperineal resection for rectal cancer. **[LEVEL OF EVIDENCE: 2]**

No mortality was reported, but overall complication rate varied between 11 and 87%. Surgical site infections (9 to 58%) and erosion of the adjacent skin (6 to 32%) were common. Up to 46% of patients underwent revisional surgery, and the proportion of patients with a functioning device at the time of evaluation after follow-up of between 6 and 34 months ranged between

Table 4. Artificial anal sphincter for faecal incontinence with Acticon Neosphincter®

Authors (ref)	Year	Number of patients	Mean or median follow-up (months)	Number (%) of functioning devices	Overall complications	"Success" in patients with a functioning device	"Success" in intention to treat
Vaizey et al [152]	1998	6	10	5 (83%)	ns	100%	83%
Lehur et al [153]	2000	24	20	20 (83%)	42%	90%	75%
Dodi et al [154]	2000	8	10.5	6 (75%)	38%	67%	50%
O'Brien et al [165]	2000	13	ns	10 (77%)	69%	90%	69%
Malouf et al [168]	2000	18	26	7 (39%)	67%	ns	39%
Altomare et al [166]	2001	28	19	21 (75%)	32%	67%	50%
Devesa et al [155]	2002	53	26.5	26 (49%)	58%	65%	53%
Wong et al [156]	2002	115	12	75 (65%)	87%	85%	54%
Ortiz et al [157]	2002	22	28	15 (68%)	77%	60%	41%
Lehur et al [158]	2002	16	25	12 (75%)	31%	92%	69%
Parker et al [159]	2003	37	39	17 (46%)	43%	49%	47%
Michot et al [169]	2003	25	34.1	20 (80%)	20%	79%	60%
Ortiz et al [151]	2003	8:AAS	44	5 (63%)	75%	CCF-FI score:16→8	
		8:DG	39	4 (50%)	50%	CCF-FI score:18→18	
Casal et al [160]	2004	10	29	9 (90%)	60%	44%	40%
O'Brien et al [150]	2004	7:AAS	6	6 (86%)	43%	CCF-FI score:19→4.8	
		7:SC	6	_	_	CCF-FI score:17.1→14.3	
La Torre et al [170]	2004	7	26.3	5 (71%)	43%	100%	71%
Altomare et al [171]	2004	25	ns: long-tem	6 (24%)	76%	50%	12%
da Silva et al [164]	2004	11:AAS	12	11 (100%)	55%	CCF-FI score:18 → 7.5	
		5:GN	38.8	5 (100%)	60%	CCF-FI score:17.4→9.4	
Michot et al [167]	2007	9	21.5	8 (89%)	11%	CCF-FI score:19→8.6	

CCF-FI:Cleveland Clinic Florida Faecal Incontinence(0: full continence - 20: worst incontinence);#AAS: artificial anal sphincter; DG: dynamic graciloplasty; SC: supportive care; GN: gracilis neosphincter; ns: not stated; "Success" is defined as "continence to solid and liquid stool without significant obstructed defecation, otherwise defined in each study"

24 and 100%, with up to 67% patients having their devices explanted. Most of the patients (78 to 100%) with a functioning device were continent to solid stool, 56 to 95% were continent to solid and liquid stool, and 22 to 67% were completely continent. The success rate in patients with a functioning device was 44 to 100%, and the intention-to-treat success rate was 41 to 83%. Studies of smaller number of patients or shorter follow-up period tend to report better outcomes.

Wong et al [156] has reported the largest, multicenter prospective trial to date. Of 115 patients, 75 patients (65%) retained a functioning device after a median follow-up of 12 months. Overall complication rate was 87%. Forty-six percent of patients underwent revisional surgery and device explantation was required in 37%. Thirty patients (40%) experienced obstructed defecation with 21 reporting to have been impacted. A successful outcome was achieved in 85% of the 61 patients with a functioning device, while the intention-to-treat success rate was 54%.

With a longer follow-up period of presumably 5 years, Altomare et al [171] reported a poorer and "disappointing long-term" results. In their initial report of 28 patients in 2001 [166], 21 patients (75%) retained a functioning device with a median follow-up of 19 months. The success rate in patients with a functioning device was 76%, and the intention-to-treat success rate was 50%. In their follow-up study of the 21 patients who retained the device in their initial report, a further 4 patients had the device removed, because of mechanical failure (2), late infection (1) or untreatable obstructed defecation (1). Out of the 17 patients who continued to have an implanted device, 14 were available for long-term evaluation. Out of the 14 patients, 5 had a revision operation, and 8 no longer activated the device because of obstructed defecation (7) or anal pain (1). Obstructed defecation occurred in 7 patients, who were unable to defecate without an enema. Although 8 patients were reasonably continent to stool, 5 of them were significantly constipated. Over all, out of the initial 25 patients for whom longer follow-up was available, only 6 patients (24%) retained a functioning device, and a good functional result was achieved only in 3 (50%) out of the 6 patients, while the intention-to-treat success rate was only 12%.

O'Brien et al [150] conducted a prospective randomized controlled trial, comparing the artificial anal sphincter (AAS) and a program of supportive care (SC). Out of the 7 patients who underwent the implantation surgery, 6 (86%) retained a functioning device after a 6 month follow-up with complication rate being 43%. The Cleveland Clinic Faecal Incontinence (CCF-FI) score (0: full continence – 20: worst incontinence) significantly decreased from a preoperative mean of 19.0 +1.2 to a postoperative mean of 4.8+4.0 in the AAS group, while it did not change in the 7 patients of SC group with an initial mean of 17.1+2.3 and a final mean of 14.3+4.6 at 6 months.

Ortiz et al [151] performed a non-randomized cohort case control study, comparing the AAS and the dynamic graciloplasty (DG). Out of the 8 patients who underwent AAS implantation, 5 (63%) continued to have a functioning device after a median follow-up of 44 months with 75% complication rate. Out of the 8 patients who underwent DG, 4 (50%) retained the stimulator after a median follow-up of 39 months with 63% complication rate. The median CCF-FI score significantly decreased from 16 to 8 in the AAS group, while it did not change from 18 to 18 in the DG group.

A retrospective case control study was reported by da Silva et al [164], comparing the AAS and the gracilis neosphincter (GN) procedure in patients with imperforate anus. All of the 11 patients who underwent the AAS retained a functioning device after a mean follow-up of 12 months with complication rate being 45%. In the 5 patients who underwent the GN, the complication rate was 60% after a mean follow-up of 38.8 months. The mean CCF-FI score significantly decreased in both groups (AAS: before 18 vs. after 7.5; GN: 17.4 vs. 9.4).

There is another artificial sphincter developed and reported by Hajivassiliou et al [179]. Different from the artificial anal sphincter (AAS), Acticon®, this prosthetic bowel sphincter (PBS) is implanted around the rectum at the supralevator plane through an abdominal approach. Finlay et al [180] implanted the PBS in twelve patients with severe faecal incontinence. At a median follow-up of 59 (range 30–72) months, nine of the 12 patients had a functioning device. There were no device-related infective complications after the initial operation, but one patient developed pseudomembranous colitis and had the device removed. The PBS was effective in restoring continence in ten of 11 patients. Median (range) Cleveland Clinic continence scores improved from 16 (7–20) before to 3 (0–7) after surgery. In two patients, the device was eventually removed owing to infection after revisional surgery that was performed due to the displacement of the sphincter component.

The PBS has two potential advantages over the AAS: it may cause less infective complications due to its sterile transabdominal implantation, and it can be applied to a severely incontinent patient with major perineal tissue loss. On the other hand, possible disadvantages include the need for a transabdominal implantation and the possibility of a severe pelvic or intraabdominal infection should an erosion occur.

At present the AAS is more widely used, and its safety and efficacy have been examined by many institutions. The PBS may have some place as an artificial sphincter because of its apparent advantages over the AAS, but its safety and efficacy need to be investigated extensively before widespread implantation is undertaken.

VII. SACRAL NERVE STIMULATION

Sacral nerve stimulation (SNS) was first applied for the treatment of faecal incontinence in 1994 by Matzel et al [181] in patients with functional deficits of the anal sphincter but no morphologic defect. The concept of recruiting residual function of an inadequate anorectal continence organ by electrostimulation of its peripheral nerve supply, i.e. the sacral spinal nerves, was adapted from the field of urology in the early 1990's [182], where it has been used since 1981 [183]. The rationale for applying sacral nerve stimulation (SNS) to faecal incontinence was based on both clinical observations and anatomic considerations (from the former, the beneficial effect on bowel habits and anorectal continence function and increased anorectal angulation and anal canal closure pressure seen in urologic patients; from the latter, the demonstration by dissection of a dual peripheral nerve supply of the striated pelvic floor muscles that govern these functions [182] with the sacral spinal nerves being the most distal common location of this dual nerve supply. It was hypothesized that stimulating the sacral spinal nerves could both enhance physiologic function and improve the symptoms of faecal incontinence.

1. TECHNIQUE

SNS has become a minimally invasive technique with low morbidity. The surgical technique can be divided into two stages:

As no other predictors of SNS outcome exist at present, patients are uniformly selected for operative implantation of a permanent neurostimulation device on the basis of clinical improvement during test stimulation. This first stage, termed percutaneous nerve evaluation (PNE), is used to confirm a satisfactory nerve response and then evaluate the clinical effect of stimulation prior to the implantation of a permanent device. Therapeutic trial stimulation is performed for a one to three-week period, a time period sufficient to prove its therapeutic effect - commonly considered if the frequency of episodes of faecal incontinence documented by bowel-habit diary is alleviated by at least 50% and if the improvement is reversible after discontinuation. Two technical options are used for subchronic percutaneous nerve evaluation (PNE): a temporary, percutaneously placed, test stimulation lead (or multiple leads) that will be removed at the end of this phase; or operative placement of a quadripolar lead, the so-called "foramen electrode" close to a target nerve. This electrode can stay in place and be used for permanent stimulation, if the test stimulation is effective. Today most commonly this foramen electrode is placed by a minimally invasive technique that uses a foramen electrode with a modified anchoring device, the so-called "tined lead" placed through a trochar. For screening, both types of leads are connected to an external pulse, the latter with a percutaneous extension cable.

The second stage is implantation of a permanent electrode and neurostimulator if screening is successful. Those with a temporary lead require simultaneous implantation of the pulse generator and the quadripolar lead, most commonly as a tined lead procedure. Those with a foramen electrode already in place for screening will undergo removal of the percutaneous extension before placement of the pulse generator (so-called "two-stage implant" [184]). Bilateral placement of foramen electrodes remains the exception, based either on improved outcome of bilateral stimulation during the screening phase [185] or on conceptual considerations [186]. The pulse generator is placed subcutaneously in the abdominal wall or gluteal area. The pulse generator is activated and stimulation parameters are set early after surgery by telemetry. The pulse generator can be deactivated by the patients with a small, handheld device commonly referred to as a "patient programmer."

2. PATIENT SELECTION AND INDICATIONS

Today, a variety of causes leading to faecal incontinence can be treated with SNS. During the initial SNS experience, only patients presenting with deficient function but no morphologic defect of the striated anal sphincter and levator ani were eligible for treatment. [182, 187, 188], However, because of the high predictive value of the test-stimulation, investigators took a more pragmatic, trial and error approach to subsequent patient selection. Patients are now selected for SNS based upon PNE results rather than conceptual considerations of the potential mechanism of action. Test stimulation is indicated, not by an underlying physiologic condition, but by the existence of an anal sphincter with reduced or absent voluntary squeeze function and existing reflex activity, indicating an intact nerve-muscle connection (confirmed by intact anocutaneous reflex activity or by muscular response to pudendal stimulation with the St. Mark's electrode) [187].

At present, the test stimulation is the only reliable modality to select patients who will likely benefit from permanent therapeutic stimulation. Two studies focused on potential predictors of success of SNS: In a study by Gourcerol et al [189], age was the only variable related with success of temporary stimulation. In patients with a permanent implant, neurologic disorders, delay of the left bulbocavernosus reflex and a prolonged or absent bulbocavernous reflex were more frequent in patients with successful outcome. In another cohort analysis, the need for repeated temporary procedures was associated with failure during the screening in univariate and multivariate analysis [190]. A low threshold to obtain motor response during temporary lead placement

was revealed to be associated with improved outcome only in univariate, but not in multivariate, analysis. Evidence of anal sphincter injury was related to a greater risk of failure during temporary testing, but not with permanent implant.

Contraindications to SNS include pathologic conditions of the sacrum preventing adequate electrode placement (such as spina bifida), skin disease at the area of implantation, anal sphincter damage requiring a sphincter substitute (e.g. artificial bowel sphincter, dynamic graciloplasty), trauma sequelae with micturition disorders or low bladder capacity, pregnancy, bleeding complications, psychological instability, low mental capacity, and the presence of a cardiac pacemaker or implantable defibrillator.

3. MECHANISM OF ACTION

The mechanism of action of SNS remains uncertain. Clinical outcome of SNS has been seen to correlate with results of anorectal physiology studies, but the effect of chronic stimulation varies greatly among published reports [187, 188]. Data are in part contradictory and inconclusive and sometimes not reproducible. The effect appears to be somatomotoric [191-198], somatosensoric [191], based on changes in the autonomic nervous system [191, 193, 199], and not limited to the continence organ per se, but also affecting the central nervous system [200]. Qualitative changes in anal and rectal motility, reduction of spontaneous rectal motility complexes [201, 202], and spontaneous anal sphincter relaxation [201] have been recorded during SNS. An effect on the mucosal neurochemistry during SNS has also been shown with elevation of Substance P and TRPV1 levels [203]. The relevance of each of this effects has not been proven in specific pathophysiological conditions. The mechanism of action is most likely multifactorial and different depending on the underlying condition. **[LEVEL OF EVIDENCE: 4]**

4. OUTCOME

The results of permanent SNS following the initial and pragmatic, trial-and-error, patient selection process are shown in **Table 5** [186, 191-194, 204-220]. Most studies have represented patients with very heterogeneous pathophysiologic conditions. Most commonly, clinical outcome is reported as an improvement in incontinent episodes or days with incontinence during the period of observation, changes in Cleveland Clinic incontinence score and in quality of life. The studies vary with regard to design and number of patients, but there is general agreement regarding the two-step stimulation for selection for permanent implant.

Matzel et al [208] published a multicenter prospective trial of SNS in 37 patients, 34 of whom underwent a permanent neurostimulator implant. Not only were the frequency of incontinence episodes and the CCIS score improved significantly, but also the ability to postpone defecation. These effects were attained immediately.

In most studies, quantitative measures are used to describe the clinical benefit, such as days with incontinent episodes/period of observation, absolute numbers of incontinent episodes/period of observation, ability to postpone defecation (in minutes), and percentage of improvement. Even though published reports differ with regard to patient population, a general pattern of outcome can be observed: when compared with baseline status, the clinical outcome is significantly improved. **[LEVEL OF EVIDENCE: 2]**

Melenhorst et al [194] published the largest single center study, with 100 patients undergoing permanent SNS. Late failure occurred in 21 patients as defined by a relapse of symptoms to less than 50% improvement over baseline, implementation of another therapy for faecal incontinence, or patient dissatisfaction. The mean time for definitive failure was 13.6 months (range 3–42.4). There was no evidence of technical failure as lead migration or lead breakage. Leroi et al [210] reported a double-blind, cross-over multicenter study in 34 patients with faecal incontinence treated with SNS. Three months after implantation, patients were randomized in a double-blind manner to on- or off-stimulation for a 2-month period, with reversal of the activation mode after 1 month. Of these, 24 of 27 randomized patients completed the 2-month trial. A significant decrease in median frequency of faecal incontinence episodes was noted during the on-stimulation period compared with the off-stimulation period. No significant change was observed between on and off stimulation for frequency of urgency episodes, delay in postponing defecation, or median number of bowel movements per week (10.2 and 11.1 for on and off, respectively). There was a trend towards greater improvement in the Cleveland Clinic Incontinence Score during on stimulation compared with off stimulation (8.5 vs 10.5; ns). A total of 24 patients (89%) considered that they had improved during the on period compared with 17 (63%) during the off period.

A report by Rosen et al [191] highlights the effect of SNS in a cohort of patients, 75% of whom suffered from faecal incontinence of neurologic origin. Frequency of incontinence episodes/week was reduced from 6 to 2 at 15 months follow-up.

Recently, some small case series and individual case reports have demonstrated the therapeutic effect of SNS in groups of patients presenting with distinct conditions and well defined anorectal physiology findings, e.g. muscular dystrophy [221], a history of rectal resection for cancer [216], neurologic dysfunction including spinal disc prolapse [217], status after rectal prolapse repair [218], after rectal resection and neoadjuvant chemoradiation [187], and with internal and external sphincter disruption due to Crohn's disease [219] . **[LEVEL OF EVIDENCE: 4]**

Table 5. Outcome of Sacral Nerve Stimulation

Authors (ref)	Year	Number of patients	Follow-up (months)	Incontinent episodes per week		Incontinence-score (CCIS)	
				Before SNS (baseline)	After SNS (last FU)	Before SNS (baseline)	After SNS (last FU)
Malouf et al [204]	2000	5	16*	ns	ns	16	2
Rosen et al [191]	2001	16	15*	6	2	ns	ns
Ganio et al [192]	2001	16	15,5	5,8	0	ns	ns
Rippetti et al [205]	2002	4	24	12	2	12,2	9,8
Matzel et al [206]	2003	16	32.5	ns	ns	16	2
Altomare et al [207]	2004	14	14*	7	0.5	15	2
Matzel et al [208]	2004	34	24*	16,4	2,0	ns	ns
Jarrett el al. [193]	2004	46	12*	7	1	14	6
Rasmussen et al [209]	2004	34	6	ns	ns	18	7
Leroi et al [210]	2005	34	7*	3,5*	0,5*	16*	10*
Kenefick et al [211]	2006	19	24*	12	0	ns	ns
Holzer et al [212]	2007	29	35*	2,3	0,67	ns	ns
Hetzer et al [213]	2007	37	13	ns	ns	14	5
Tan et al [214]	2007	53	12	9,5	3,1	16	1,2
Melenhorst et al [194]	2007	100	25,5	10,4	1,5	ns	ns
Melenhorst et al [215]	2008	A†=20	29,2	8,9	4,2	ns	ns
	2008	B††=20	22,6	8,3	1,4	ns	ns
DISTINCT CONDITIONS							
Jarrett et al [216]	2005	2	12	9,8	1	ns	ns
Jarrett et al [217]	2005	12	12*	9,2	2,4	ns	ns
Jarrett et al [218]	2005	4	12	12.2	2	ns	ns
Ratto et al [186]	2005	4	19.5	12	2,5	16,3	4,5
Vitton et al [219]	2008	5	14*	ns	ns	15*	6*
Jarrett et al [220]	2008	8	26,5*	5,5*	1,5*	15*	9,5*

* median, otherwise all data presented at mean

† A: after sphincter repair, †† B: with sphincter gap 17-33% of circumference, without repair.

An increasing body of evidence indicates that SNS may also be a treatment option for patients with sphincter defects, unrepaired or after attempted anatomic reconstruction. The presence of an internal anal sphincter defect on endoanal sonography is reportedly unrelated to the success of permanent SNS [190]. Three of five patients with ultrasound evidence of sphincter disruption measuring 25 % – 33% of the circumference benefited from chronic SNS [222]. In 20 patients with unrepaired obstetric trauma, SNS resulted in significant improvement of the Cleveland Clinic score (CCS; from 16 to 3) in 19, and of the numbers of incontinent episodes per week (from 10 – 1) with a minimum follow-up of 4 years [223]. In patients with an unrepaired external or internal anal sphincter or both, the frequency of incontinent episodes per week decreased from 1.3 to 0.3 and the CCS improved (from 15 to 3.5) with a follow-up of 12-97 months [224]. Melenhorst et al. showed that the primary use of SNS in patients with a sphincter gap 17-33% of the circumference appeared to result in an outcome similar to its use after failed sphincter repair [215].

SNS in 6 of 8 patients presenting with faecal incontinence related to obstetric full thickness anal sphincter lesions ranging from > 30 - 150 degree resulted at a median follow-up of 26.5 months in improved frequency of incontinent episodes per week from 5.5 to 1.5 clinical function [220], improved ability to postpone bowel emptying and improved ASCRS quality of life scores. A further cohort study [225] reports on the effect of permanent SNS in 53 patients presenting with either an intact external anal sphincter (N= 32 [37.5% after sphincter repair]) or an external anal sphincter lesion (N=21 [81% after prior sphincter repair]) of ≤ 90° (N=11) or 90-120° (N=10). Improvement of symptoms and quality of life was achieved in all groups. Outcome after 12 months was statistically not significantly different between those patients with an intact sphincter complex and those without. Chan & Tjandra [226] reviewed 53 consecutive patients who underwent SNS for faecal incontinence. There was no significant difference in outcomes between those with and without an external sphincter defect. **[LEVEL OF EVIDENCE: 3]**

In a randomized controlled trial Tjandra et al [225] compared the effect of sacral neurostimulation for severe faecal incontinence with supervised optimal medical therapy that comprised pelvic floor exercises, bulking agents, and dietary manipulation. Permanent SNS in 53 patients was significantly better than conservative treatment in 60 patients: Cleveland Clinic Continence Score 1.2 vs. 14.1; incontinent episodes/ week: 3.1 vs 9.4, days with incontinence/week; 1 vs. 9.4, lifestyle: 3.31 vs. 2,31, coping/behavior: 2.68 vs. 1,86, depression/self-perception: 3.25 vs. 2.64, embarrassment: 2.76 vs. 1.78. **[LEVEL OF EVIDENCE: 2]**

5. QUALITY OF LIFE

As with indications, outcome assessment has also evolved and aspects of quality of life were added to the evaluation of outcome (Cleveland Clinic Continence Scoring System, SF36 and FIQL Score.) The therapeutic impact of SNS is most evident when a disease-specific quality-of-life instruments ASCRS FIQL scale is applied.

In the multicenter clinical trial by Matzel el al [208], ASCRS FIQL was significantly increased in all 4 scales, SF-36 scores improved in 7 of 8 scales, the greatest being social functioning and mental health; but only social functioning reached statistical significance. A similar result was published by Leroi et al [210] using the French version of the ASCRS QOL (FIQL): at the final follow-up visit improvements in lifestyle, coping and behavior, depression and self-perception and embarrassment were significantly improved. Hetzer et al [213] demonstrated a significant improvement of the median Gastrointestinal Quality of Life Index score with permanent from a baseline score of 96 (range 47–128) to 107 (range: 36–128) at 6 months post-implantation. **[LEVEL OF EVIDENCE: 2]**

6. COST BENEFIT / ROLE IN THE TREATMENT ALGORITHM

Permanent SNS is expensive. Hetzer et al [227] conducted a comparative cost analysis of SNS with conservative treatment, anterior sphincteroplasty, dynamic graciloplasty and creation of a stoma in 34 consecutive patients. The 5-year cumulative costs for SNS is € 19333, compared with € 35965 for stoma with annual costs of € 5339, and € 34953 for dynamic graciloplasty with annual costs of € 1659. The equivalent cost for conservative treatment was € 3895. The overall median real cost for an anterior sphincteroplasty was €5327. **[LEVEL OF EVIDENCE: 4]**

7. SAFETY

SNS is a safe procedure. The rate of complications is relatively low [187, 188]. In only approximately 5% of the patients has discontinuation of treatment with device removal been necessary because of loss of effect, deterioration of symptoms, pain lead dislocation, or infection. When infection has necessitated removal, re-implantation at a later date has been successful [204]. **[LEVEL OF EVIDENCE: 3]**

VIII. POSTERIOR TIBIAL NERVE STIMULATION

For peripheral stimulation the tibial nerve is temporarily stimulated with surface or needle electrodes at the level of the malleolus. Scattered preliminary data from short term studies with temporary posterior tibial nerve

stimulation [228] indicate a therapeutic effect in idiopathic faecal incontinence [229] and incontinence due to partial spinal trauma [230] on frequency of incontinence episodes, incontinence scores and QoL. Larger patient studies are awaited. **[LEVEL OF EVIDENCE: 4]**

IX. INJECTABLE BIOMATERIALS

Injection of bulk-enhancing agents into the anal canal to treat faecal incontinence was a natural consideration after its successful use in urinary incontinence. The use of injectable agents to increase urethral resistance at the level of the bladder neck has had variable success; however the benefit of performing an outpatient procedure without anesthesia has resulted in its continued use [231]. The ideal agent for injection should be biocompatible, non-allergenic, non-immunogenic, easy to inject and not migrate within the tissues. No agent currently has all these properties. Agents that have a diameter of 80 μm are felt to be less prone to migration; however larger agents require a larger bore needle to inject, which put them at a higher risk for leakage from the injection site. The results of injectable biomaterials are shown in **Table 6** [232-242].

The history of using injectable agents for faecal incontinence began in 1993 when Shafik [232] treated 11 patients (7 of whom had internal sphincterotomy and 4 idiopathic incontinence) with injections of polytetrafluoroethylene paste into the anal submucosa. After an 18-24 month follow-up, 64% reported complete cure and 36% had partial improvement. Shafik [233] subsequently treated 14 patients with autologous fat injections. All patients became continent after repeat injections. There were no complications using either agent. However, subsequent reports of autologous fat injection have resulted in serious complications including death, stroke and pulmonary embolism and thus at the present time is not used for faecal incontinence [243].

Other agents used for injection thus far include micro balloons, glutaraldehyde cross-linked synthetic bovine dermal collagen (Contigen), PTQ implants (Bioplastique), Pyrolytic carbon-coated zirconium oxide beads (Durasphere), dextranomer- hyaluronic acid co-polymer (Zuidex), cross-linked porcine dermal collagen (Permacol), and polyacrylamide hydrogel (Bulkamid). A small series of six patients injected with self-detaching cross-linked silicone micro balloons with a biocompatible filler material demonstrated fairly good results with Browning-Parks incontinence scores for the group decreasing from 16 to 5 (range 0-20) [234]. However, sterilization issues have prevented the ongoing use of this product.

The first reported injections of glutaraldehyde cross-

Table 6. Injectable biomaterials

Authors (ref)	Year	Agent	Number of patients	Injection technique	Number of sites	Volume	Success
Shafik [232]	1993	Polytetrafluoroethylene paste	11	Transanal	2	0.5ml	64%
Shafik [233]	1995	Autologous fat	14	Transanal	2	50-60ml	100%
Kumar et al [235]	1998	Glutaraldehyde cross-linked synthetic bovine dermal collagen	17	Transanal	1-3	Up to 2ml	65%
Malouf et al [237]	2001	PTQ implants	10		1-4	5-11.5ml	60% initial, 20% long-term
Feretis et al [234]	2001	Microballoons with biocompatible hydrogel	6	Transanal	3-5	0.9ml in balloon	Incontinence score 16 to 5
Davis et al [240]	2003	Carbon-coated zirconium oxide beads	18	Transanal	1-4	1.28ml	83% improved
Tjandra et al [238]	2004	PTQ implants	82	Intrasphincteric with and without ultrasound	4	2.5ml	Incontinence score with US: 14.5 to 3 Without US: 14.5 to 11
Stojkovic et al [236]	2006	Glutaraldehyde cross-linked synthetic bovine dermal collagen	73	Transanal	3	1.7ml	63%
de la Portilla et al [239]	2008	PTQ implants	20	Transsphincteric	3	2.5 ml	Incontinence score 13.5 to 4.5
Altomare et al [241]	2008	Carbon-coated zirconium oxide beads	33	Transsphincteric	4	8.8ml	Incontinence score 12 to 8
Maeda et al [242]	2008	Cross-linked porcine dermal collagen	5	Transsphincteric	3	9ml	Incontinence score 15 to 12.5
		Polyacrylamide hydrogel	5	Transsphincteric	3	15ml	Incontinence score 15 to 12.5

* US: endoanal ultrasound guidance

linked collagen for faecal incontinence included 17 patients [235]. Following injection, with a mean follow-up of 8 months, 11 patients showed marked symptomatic improvement. A much larger series was recently reported by Stojkovic et al [236]. Patients were injected with 1.7 ml of collagen transanally into the submucosa in three separate areas just proximal to the anal canal. Of the 73 patients, 63% reported an improvement in their incontinence. The 49 patients with idiopathic incontinence (no sphincter defect and no pudendal neuropathy) had a significant decrease in their Cleveland Clinic Florida Incontinence Score (CCFIS). The disadvantages of using synthetic collagen are its potential to be allergenic and degradation over time. Furthermore, its success in urinary incontinence has been limited [231].

Injectable silicone biomaterial, also previously known as macroplastique and bioplastique, has been used extensively for urinary incontinence. It consists of polydimethylsiloxane particles suspended in a bioexcretable carrier hydrogel of polyvinylpyrrolidone. Two pilot studies in 2001 and 2002 [237, 244] led to increased use of this product in Europe and it has been renamed PTQ implants (PTP implants in Australia). Malouf et al [237] studied 10 patients with passive incontinence injected circumferentially or at a single site with Bioplastique. At six weeks, 6 of 10 patients showed either marked improvement or complete cessation of leakage. However, after six months, only 2 of 7 had maintained marked improvement. Complications included anal pain and ulceration at the injection site.

Tjandra et al [238] randomized 82 patients with severe faecal incontinence to receive PTP implants either with or without endoanal ultrasound guidance for injection. All patients had a significant improvement in their Cleveland Clinic Continence scores (range 0-20). The ultrasound guided group had a decrease in score from 14.5 to 3 and the non-guided group had a decrease from 14.5 to 11 at 12 months. Six patients, two from the ultrasound guided group complained of pain at the injection sites. There were no other complications. The ultrasound guided group was also found to have a more significant improvement in resting pressures and quality of life scores. The same group injected PTQ in 7 patients with passive incontinence after hemorrhoidectomy and found significant improvement of Cleveland Clinic Continence scores and quality of life scores in all patients [245].

Only one report exists of long-term results for injectable agents and this was using Bioplastique. Maeda et al [246] reported the 5 year outcome of 6 patients injected with Bioplastique in 1999. The median St. Mark's incontinence score was essentially unchanged from 11 to 13 (range 9-20) and one patient had undergone a colostomy. However, four of the remaining five patients reported subjective improvement in their incontinence and quality of life scores.

The largest series of patients injected with silicone biomaterial included 20 patients with passive faecal incontinence of liquid or solid stool who had failed conventional therapy [239]. Ten patients had disruption of the internal anal sphincter; nine had degeneration of the sphincter. Patients were injected trans-sphincterically using an 18-gauge needle in the skin 2 cm from the anal margin and a finger in the anal canal to direct the injection above the dentate line in the submucosal plane. Three areas were injected with 2.5 ml of product. The Cleveland Clinic Continence score (0-20) decreased significantly from 13.5 to 4.5 at one month and slowly increased to 9.4 at two years, which was a still a significant improvement from the baseline score. Quality of life scores also improved, but there was no effect on resting and squeeze pressures measured at baseline and 3 months after injection. Of note, 70% of patients experienced pruritus ani and one patient developed an infection at an injection site. Post procedure endoanal ultrasounds found no evidence of migration of the product in 19 patients.

Pyrolytic carbon-coated zirconium oxide beads (Durasphere) are non-reactive and are not biodegradable. However, they are known to migrate within the tissues and require a large bore needle to inject the substance. Davis et al [240] assessed the short and long-term efficacy in 18 patients with an internal anal sphincter defect refractory to conservative management. It was injected in the submucosal plane at the site of the defect until adequate anal sphincter symmetry was restored. At 12 months, incontinence scores and patient satisfaction scores were significantly improved. Fifteen of eighteen patients reported improvement in their incontinence. An abstract presented by Weiss et al [247] demonstrated improvement in ten patients who were only followed for 3 months. Altomare et al [241] recently published a study of 33 patients with minor or medium severity faecal incontinence (Cleveland Clinic Continence score $\leq$ 14 and/or American Medical Systems score $\leq$ 89) were injected with a mean of 8.8 ml (range 2-19 ml) of Durasphere into the submucosa at the level of the dentate line using an 18-gauge needle. After a mean follow-up of 21 months, the incontinence severity scores for the group decreased significantly but the faecal incontinence specific quality of life did not change. Resting and squeeze pressures were also increased 12 months after injection. Adverse events included anal pain in two patients, asymptomatic leakage of material in one patient, and distal migration of product in two patients.

Dextranomer-hyaluronic acid co-polymer (Zuidex) has been used to treat urinary incontinence [248] and a randomized placebo controlled trial is underway to assess its efficacy for faecal incontinence. Dextranomer microspheres are suspended in non-animal stabilized hyaluronic acid. There are no published reports of its use in faecal incontinence to date.

Another study of injectable agents for faecal incontinence involves two other products, cross-linked porcine dermal collagen (Permacol), and polyacrylamide hydrogel (Bulkamid) [242]. Ten patients with passive faecal incontinence to liquid or solid stool who had failed conventional treatments received either of the two products. Injection was performed transsphincterically after injecting the skin 2 cm from the anal margin. The median volume to achieve closure of the anal canal under direct vision was 9 ml for Bulkamid and 15 ml for Permacol. There was a decrease in the St Mark's incontinence score at 6 weeks for both groups and only a sustained decrease in the score for the Bulkamid group at 6 months. As this was a pilot study, there was inadequate power to assess the different in these treatments for faecal incontinence.

In summary, the data for injectable biomaterials comprises several small case series that in general show short term efficacy. [LEVEL OF EVIDENCE: 3]

X. COLOSTOMY

A permanent colostomy is usually formed as a last resort for severe faecal incontinence when all other interventions have failed. Because colostomy is generally regarded as a failure of treatment, its effectiveness, perioperative complications, and impact on the quality of life have never been properly evaluated except for patients with functional bowel disorders after spinal cord injury [249, 250]. For a specific role of colostomy for these patients, please refer to the specific chapter. Not only for patients with spinal cord injury, but also for the general population with severe faecal incontinence, colostomy is a frequently successful management strategy that restores dignity and allows them to regain social function.

No systematic reviews, randomized controlled trials or non-randomized cohort studies have been reported regarding colostomy for faecal incontinence, and only one case control study [251], two case series [252, 253], and one systematic review [146] were identified. **[LEVEL OF EVIDENCE: 4]**

Colquhoun et al [251] conducted a cross-sectional postal survey, comparing quality of life between 71 patients with faecal incontinence and 39 with a colostomy created for rectal cancer, complicated colonic diverticular disease or faecal incontinence. Analysis of the Short Form 36 General Quality of Life Assessment revealed significantly higher social function score in the colostomy group than in the faecal incontinence group (0 vs. -0.6, p=0.022). An age- and gender-adjusted regression analysis of the Faecal Incontinence Quality of Life score revealed significantly higher scores in the coping (2.7 vs. 2.0, p=0.005), embarrassment (2.7 vs. 2.2, p=0.014), and lifestyle scales (3.2 vs. 2.7, p=0.14) in the colostomy group compared to the faecal incontinence group. The authors concluded that a colostomy is a good option for patients who suffer from severe faecal incontinence and offers a definitive cure with improved quality of life.

Tan et al [146] performed a systematic review specifically comparing the cost-effectiveness between end stoma (ES), artificial anal sphincter (AAS) and dynamic graciloplasty (DG). The quality-adjusted life years (QALYs) and the incremental cost-effectiveness ratio (ICER) were compared between the three procedures, by obtaining the probability estimates for patients with faecal incontinence from published data supplemented by expert opinion. The end stoma was the most cost-effective therapy at 5 years, with a QALY gain of 3.45 for 16,280 GB£ and an ICER of £4,719/QALY, compared to AAS (4.38 for £23,569; £5,387/QALY) and DG (4.00 for £25,035; £6,257/QALY). After 10 years, AAS became the most cost-effective surgical intervention, with a QALY gain of 8.384 for £32,397 and an ICER of £3,864/QALY, compared to ES (6.9 for £27,910; £4,046/QALY) and DG (7.678 for £35,165; £4,580/QALY). The results of this study, however, must be interpreted with great caution, because it is not an interventional study but a systematic review with a rather complicated methodology and a variety of possible biases.

Norton et al [252] examined patients' view of a colostomy by conducting a questionnaire study of patients who had a colostomy created to manage their faecal incontinence. Sixty-nine people (58 women) responded. When patients were asked to rate their ability to live with their stoma now on a scale of 0-10, the median score was 8 (range 0 – 10). The majority (83%) felt that the stoma, within the past month, restricted their life "a little" or "not at all". Eighty-four percent answered that they would "probably" or "definitely" choose to have the stoma again. When they were asked the question "compared to when you were incontinent, how much change has having a stoma made to your overall quality of life?" on the scale of -5 (much worse) to +5 (much better), the median rating was +4.5 (range -5 to +5). The authors concluded that health care professionals should discuss the option of a stoma with incontinent patients because of the overwhelmingly positive outcomes.

An end sigmoid colostomy without proctectomy is usually recommended as a procedure of choice for patients who elect colostomy for the management of their refractory faecal incontinence. Creating such a colostomy, however, does not always solve all the problems of patients with faecal incontinence. Catena et al [253] reported a retrospective chart review of 44 patients (35 women) who underwent elective end sigmoid colostomy for faecal incontinence of various

etiologies. After colostomy formation 19 patients (43%) were asymptomatic, while the other 25 experienced such problems with their rectal stump as diversion colitis and mucus leakage. Of the 25 patients, 12 (27% of the total) underwent a secondary proctectomy due to the rectal stump problems sufficient to warrant the operation. Histological examination revealed diversion colitis in 6 patients. The factor associated with proctectomy was age, with younger patients being more likely to require rectal excision. The authors concluded that data are insufficient to recommend primary proctectomy in patients with severe faecal incontinence warranting permanent end sigmoid colostomy.

XI. ANTEGRADE CONTINENCE ENEMA

The concept of irrigation is to ensure emptying of the colon and/or rectum to prevent seepage of stool. It has been used for patients with neurogenic bowel dysfunction and those with symptoms of incontinence [254, 255].

Antegrade irrigation involves operative construction of an appendicostomy, cecostomy, or sigmoidostomy which will serve as a continent conduit for colonic enemas [254, 256-258]. The operation can also be done laparoscopically. There is a reported 65 to 78% subjective improvement in patients, but some studies have included patients with concurrent difficulty with defecation [259-261]. **[LEVEL OF EVIDENCE: 3]** The disadvantages of the procedure have been documented; the most common has been wound infection in up to 45% of patients [259]. This seems to be reduced by creating a so-called 'neo-appendicostomy' with a part of ileum [262, 263].

B. SURGERY FOR PEDIATRIC FAECAL INCONTINENCE

Faecal incontinence is common in children who have anorectal malformations, Hirschsprung's disease and spinal problems. Despite advances in technique for anatomic corrective surgery, many patients continue to suffer from persistent incontinence. This guideline mainly focuses on anorectal malformations and corrective surgeries along with their results and subsequent management in case of persistent incontinence. The latter is also applicable for treatment of patients suffering from incontinence after surgery for Hirschsprung's disease and those with spinal problems. Other surgical interventions used less frequently were also reviewed.

I. ANORECTAL MALFORMATIONS

Anorectal malformations occur one in 3000-5000 live births. Although the severity of malformation varies, it is invariably associated with defecatory problems including incontinence. The surgical advances have been most prominent in last few decades, particularly with the advent of posterior sagittal approach. **[LEVEL OF EVIDENCE: 3]** This technique has enabled surgeons to visualize the anatomy under direct vision and perform corrective surgeries more accurately [264, 265]. In brief, a mid-sagittal incision is performed and the sphincter mechanism is completely divided in the midline. The rectum is separated from the genitourinary tract and moved down to the perineum. The most challenging aspect of the operation is the separation of the rectum from the vaginal or urinary tract, which effectively requires creating two walls out of one septum without damaging each structure. This approach can also used for reoperation in anorectal malformations [266] and can also be applied for reconstruction of severe perineal trauma [267].

For both male and female babies, urethral-perineal fistula is the simplest fistula to correct. These require the so-called 'minimal posterior sagittal approach,' which enlarges the stenotic orifice and relocates the rectal orifice posteriorly within the limits of the sphincter complex. For males with recto-urethral-bulbar fistula or recto-urethral-prostatic fistula and females with recto-vestibular fistula or cloaca with short (less than 3 cm) common channel, posterior sagittal approach is the main operation performed. For males with higher fistulas such as recto-bladder neck fistula and other complex and unusual defects and females with cloaca with long (greater than 3 cm) common channel and complex defects, the posterior sagittal approach needs to be coupled with abdominal access which can be either laparoscopy or laparotomy.

Cloacal repair is the most challenging amongst the corrective surgeries for anorectal malformations. A recent operative advance in cloacal repair is a maneuver called total urogenital mobilization, whereby the rectum is separated from the vagina and both vagina and urethra are then mobilized together. The advantage of this technique is to avoid separating rectum, vagina and urethra completely, which is not feasible all the time and risks damaging these structures during the procedure. This technique avoids the risk of urethrovaginal fistula and vaginal stricture (previously reported as complications in 10% of cloacal repairs) and also gives enough mobilization to allow more than 50% of all cloacal repairs without opening the abdomen [268, 269].

Functional outcomes depend on the severity of the malformations. A review of more than 1000 anorectal malformation cases showed 100% of babies who had perineal fistula repair achieved continence. Approximately 55% of patients who had been operated

for recto-vestibular fistula had bowel control. Any malformations more complicated resulted in only up to 30% achieving continence. All patients who had recto-bladder neck fistula repair were incontinent. In cloacal repair the length of common channel shorter or longer than 3 cm appears to be the distinct prognostic factor in terms of functional outcome [270]. Overall it is estimated that nearly 40% will have voluntary bowel movement and no soiling, but some of them may still lose bowel control in case of severe diarrhea, and 25% of all repairs will result in total incontinence [271].

For the group of patients with persistent incontinence following the corrective surgery, the next aim will be to keep the colon clean to avoid unpleasant accidents and improve their quality of life. A good option is implementation of a bowel management program whereby the patient and family are instructed in the use of daily enema, manipulation of diet, and medication to remain clean [272]. This is also a good treatment for constipation, which is the most common difficulty after corrective surgery [273].

Although most young children accept their parents administering enemas, when they get older they want privacy and rectal enemas on a daily basis becomes an unpleasant routine. In such cases, continent appendicostomy is a feasible option, whereby a conduit for the administration of an antegrade continence enema (ACE) is created. First described by Malone [274], it has become an important option in pediatric surgery for functional bowel disorders.

According to the initial description by Malone, appendicostomy was created by dividing the appendix at its base and reimplanting by a reverse manner into the cecum, which was then exteriorized through the right lower quadrant. Malone later revised it and the reimplantation of appendix is no longer considered necessary [275]. Levitt et al introduced utilizing the appendix in situ and added cecal plication to prevent reflux of stool and exteriorizing through umbilicus fold rendering it less noticeable [276]. This appears to yield good long-term results [277], though a recent study has shown that cecal fixation and wrap may be unnecessary for appendicostomy (44 patients, consecutive) [278]. The benefit of a variation called orthotopic continent appendiceal stoma is not clear [279]. However, construction of appendicostomy with burial of the appendiceal tip appears to help avoid problems of exposed mucosa such as bleeding and mucus discharge. From this perspective, a few techniques have been suggested such as V-Y flap [275] and Y-appendicoplasty [280]. For patients without an appendix, a neoappendix could be formed from ileum or cecum [281, 282] (retrospective, 13 patients). Laparoscopic antegrade continence enema procedure has been reported to yield as good result as open procedure [283-285].

This procedure is not a cure to the problem but a more acceptable method for many children to engage in a bowel management program without the need for rectal enemas. Success rate is variable between 61-90% (31 patients, 3.25 yrs [286], 40 patients, < 2 years [287], 45 patients, retrospective [288], 21 patients [275], 62 patients, retrospective [289], < 4 years, 65 patients [290], with older children benefiting more [291]. Satisfaction with the treatment is reported to be as high as 93% [276, 292]. **[LEVEL OF EVIDENCE: 3]**

As with any operation, there are known complications associated with antegrade continence enema. Stomal stenosis is the most common complication and the use of the ACE stopper will prevent it in the short-term (retrospective, 14 patients) [293]. Leakage of stool from appendicostomy is another common complication. These complications cause 10% of patients to undergo revision of the appendicostomy [271]. Stoma prolapse, pressure sore, wound infection, anastomotic leak, stomal granulation, cecal-flap necrosis and cecal volvulus are less common complications reported after ACE [294-296].

II. OTHER CAUSES OF FAECAL INCONTINENCE

Some children with Hirschsprung's disease following pull-through operations and severe constipation may also present with symptoms of incontinence [297]. Patients with spinal problems often lack bowel control due to paralysis and absence of sensation; 50% of children with spina bifida suffer from incontinence [298]. The majority of these cases can be successfully managed with the above mentioned bowel management program including appendicostomy, although wheelchair-bound children with spinal neuropathy is a predictive factor for poorer outcome with ACE [287].

The mechanism of incontinence after an operation for Hirschsprung's disease, anorectal malformations and severe constipation is thought to be due to impaired bowel motility. Impaired bowel motility causes faecal impaction, which can lead to development of a dilated segment of bowel called 'megarectosigmoid'. This can subsequently lead to overflow incontinence due to incomplete evacuation [299]. Once the rectum is dilated it is refractory to conservative management, and resection of megarectum or megasigmoid has been associated with improvement [299-301]. A small minority of patients (5%) who fail these options may need colostomy [272].

III. OTHER SURGERIES

Sphincter augmentation by either palmaris longus transposition, gluteus muscle transposition, graciloplasty or levatorplasty has been used for children with faecal incontinence, albeit in small series[110, 302-309]. Dynamic graciloplasty has also been piloted and 50% of patients achieved complete continence, though the study only contained four patients [310]. **[LEVEL OF EVIDENCE: 4]**

C. CONCLUSIONS

Data regarding the surgical treatment of faecal incontinence are generally weak. Randomized, controlled studies are few, and practical considerations make the likelihood of such studies improbable. The quality of data reported in older studies was often poor. Problems included heterogeneous patient populations; variable definitions of “continence,” “incontinence,” “success,” and “failure”; non-standardized and non-validated continence scales; underreporting of validated symptom-specific quality of life measures; variable patient follow up and lack of independent assessment of continence outcomes. However, there has been a notable improvement in the quality of studies reported in the past decade.

The spectrum of surgery for faecal incontinence is broad and expanding. Interventions range from simple outpatient procedures to major reconstructive surgery. As the reported outcomes of these various operations are often similar, a sound general principle is to proceed first with the simplest and least invasive procedure. Major operations associated with more profound morbidity should be restricted to patients who have failed simpler measures.

1. Sphincter Repair (Grade B)

Sphincter repair is indicated for patients with acute traumatic sphincter disruption, such as following obstetrical injury, but many patients experience persisting symptoms.

2. Sphincteroplasty (Grade B)

Overlapping sphincteroplasty can be offered to patients with significant faecal incontinence and a documented sphincter defect. Most patients improve after sphincteroplasty, but outcomes deteriorate over time.

3. Postanal Repair (Grade C)

Postanal repair can be performed with modest success in carefully selected patients. However, this procedure is now rarely performed due to the advent of newer treatments.

4. Non-Stimulated Muscle Transposition (Grade C)

Non-stimulated muscle transposition repair can be performed with modest success in carefully selected patients, notably in children. However, this procedure is now rarely performed due to the advent of newer treatments.

5. Stimulated Muscle Transposition (Grade C)

Stimulated muscle transposition has been shown to have reasonable success but is associated with significant morbidity. It remains a useful technique in selected patients with significant perineal tissue loss or in those who have failed other treatments.

6. Artificial Anal Sphincter (Grade B)

Artificial anal sphincter has been shown to have reasonable success but is associated with significant morbidity. It remains a useful technique in carefully selected patients, particularly those who have failed other treatments.

7. Sacral Nerve Stimulation (Grade B)

SNS is an effective therapy for most patients with clinically significant incontinence who fail conservative management. The technique is safe, minimally invasive, and has the unique advantage of allowing a therapeutic trial prior to permanent stimulator implantation.

8. Posterior tibial nerve stimulation (Grade D)

Posterior tibial nerve stimulation is an investigational technique with few available data regarding efficacy and outcome.

9. Injectable Biomaterials (Grade C)

Most series of injectable biomaterials report reasonable success rates. However, the optimal injectable bulking agent and the technique for its insertion have not been established.

10. Colostomy (Grade C)

Formation of an end colostomy is a reasonable treatment option for patients with refractory faecal incontinence who are able to accept the associated alteration in body image. Colostomy provides restoration of a more normal lifestyle and improves quality of life. It also could be the most cost-effective in the short to medium term, compared to more complicated surgical procedures such as artificial anal sphincter and dynamic graciloplasty. Colostomy should not be regarded as a treatment failure but rather a reasonable treatment option for patients whose lives are restricted by faecal incontinence that is not amenable to other therapies. An end sigmoid colostomy alone, without proctectomy, is recommended. The minority of patients who develop significant symptoms from their retained rectal stump may eventually require proctectomy as a secondary procedure.

11. Antegrade Continence Enema (Grade C)

Antegrade continence enema is a useful technique to ameliorate faecal incontinence refractory to more conventional therapies. Patients must accept placement of a small stoma and be willing to adhere to a regular irrigation program.

12. Surgery for Pediatric Faecal Incontinence (Grade C)

Anorectal malformations should undergo surgical repair, most commonly by a posterior sagittal repair. An antegrade continence enema procedure can be considered for children with persistent or refractory faecal incontinence.

REFERENCES

1. Malouf AJ, Norton CS, Engel AF, Nicholls RJ, Kamm MA. Long-term results of overlapping anterior anal-sphincter repair for obstetric trauma. Lancet 2000;355(9200):260-5.
2. Halverson AL, Hull TL. Long-term outcome of overlapping anal sphincter repair. Dis Colon Rectum 2002;45(3):345-8.
3. Parks AG. Anorectal incontinence. Proceedings of the Royal Society of Medicine 1975;68(11):681-690.
4. Brown SR, Nelson RL. Surgery for faecal incontinence in adults. Cochrane Database Syst Rev 2007(2):CD001757.
5. Fitzpatrick M, Cassidy M, O'Connell PR, O'Herlihy C. Experience with an obstetric perineal clinic. Eur J Obstet Gynecol Reprod Biol 2002;100(2):199-203.
6. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. N Engl J Med 1993;329(26):1905-1911.
7. Donnelly V, Fynes M, Campbell D, Johnson H, O'Connell PR, O'Herlihy C. Obstetric events leading to anal sphincter damage. Obstet Gynecol 1998;92(6):955-61.
8. Oberwalder M, Connor J, Wexner SD. Meta-analysis to determine the incidence of obstetric anal sphincter damage. Br J Surg 2003;90(11):1333-7.
9. Williams AB, Bartram CI, Halligan S, Spencer JA, Nicholls RJ, Kmiot WA. Anal sphincter damage after vaginal delivery using three-dimensional endosonography. Obstet Gynecol 2001;97(5 Pt 1):770-5.
10. Zetterstrom JP, Lopez A, Anzen B, Dolk A, Norman M, Mellgren A. Anal incontinence after vaginal delivery: a prospective study in primiparous women. Br J Obstet Gynaecol 1999;106(4):324-30.
11. Fitzpatrick M, McQuillan K, O'Herlihy C. Influence of persistent occiput posterior position on delivery outcome. Obstet Gynecol 2001;98(6):1027-31.
12. Harkin R, Fitzpatrick M, O'Connell PR, O'Herlihy C. Anal sphincter disruption at vaginal delivery: is recurrence predictable? Eur J Obstet Gynecol Reprod Biol 2003;109(2):149-52.
13. Eogan M, Daly L, O'Connell PR, O'Herlihy C. Does the angle of episiotomy affect the incidence of anal sphincter injury? Bjog 2006;113(2):190-4.
14. Clemons JL, Towers GD, McClure GB, O'Boyle AL. Decreased anal sphincter lacerations associated with restrictive episiotomy use. Am J Obstet Gynecol 2005;192(5):1620-5.
15. Dudding TC, Vaizey CJ, Kamm MA. Obstetric anal sphincter injury: incidence, risk factors, and management. Ann Surg 2008;247(2):224-37.
16. Fitzpatrick M, Behan M, O'Connell PR, O'Herlihy C. A randomized clinical trial comparing primary overlap with approximation repair of third-degree obstetric tears. Am J Obstet Gynecol 2000;183(5):1220-4.
17. Fernando RJ, Sultan AH, Kettle C, Radley S, Jones P, O'Brien PM. Repair techniques for obstetric anal sphincter injuries: a randomized controlled trial. Obstet Gynecol 2006;107(6):1261-8.
18. Williams A, Adams EJ, Tincello DG, Alfirevic Z, Walkinshaw SA, Richmond DH. How to repair an anal sphincter injury after vaginal delivery: results of a randomised controlled trial. Bjog 2006;113(2):201-7.
19. Garcia V, Rogers RG, Kim SS, Hall RJ, Kammerer-Doak DN. Primary repair of obstetric anal sphincter laceration: a randomized trial of two surgical techniques. Am J Obstet Gynecol 2005;192(5):1697-701.
20. Fernando R, Sultan AH, Kettle C, Thakar R, Radley S. Methods of repair for obstetric anal sphincter injury. Cochrane Database Syst Rev 2006;3:CD002866.
21. Mahony R, Behan M, Daly L, Kirwan C, O'Herlihy C, O'Connell PR. Internal anal sphincter defect influences continence outcome following obstetric anal sphincter injury. Am J Obstet Gynecol 2007;196(3):217 e1-5.
22. Cook TA, Mortensen NJ. Is there a role for the colorectal team in the management of acute severe third-degree vaginal tears? Colorectal Dis 1999;1(5):263-266.
23. Nordenstam JF, Zetterstrom J, Lopez A, Johansson C, Anzen B, Parker S, Mellgren A. Operative technique and expertise influence outcome after primary repair for obstetric sphincter injury [abstract]. Dis Colon Rectum 2006;49(5):729.
24. Soerensen MM, Bek KM, Buntzen S, Hojberg KE, Laurberg S. Long-term outcome of delayed primary or early secondary reconstruction of the anal sphincter after obstetrical injury. Dis Colon Rectum 2008;51(3):312-7.
25. Mahony R, Behan M, O'Herlihy C, O'Connell PR. Randomized, clinical trial of bowel confinement vs. laxative use after primary repair of a third-degree obstetric anal sphincter tear. Dis Colon Rectum 2004;47(1):12-7.
26. Eogan M, Daly L, Behan M, O'Connell PR, O'Herlihy C. Randomised clinical trial of a laxative alone versus a laxative and a bulking agent after primary repair of obstetric anal sphincter injury. Bjog 2007;114(6):736-40.
27. Faltin DL, Boulvain M, Irion O, Bretones S, Stan C, Weil A. Diagnosis of anal sphincter tears by postpartum endosonography to predict fecal incontinence. Obstet Gynecol 2000;95(5):643-7.
28. Abramowitz L, Sobhani I, Ganansia R, Vuagnat A, Benifla JL, Darai E, Madelenat P, Mignon M. Are sphincter defects the cause of anal incontinence after vaginal delivery? Results of a prospective study. Dis Colon Rectum 2000;43(5):590-6; discussion 596-8.
29. MacArthur C, Glazener CM, Wilson PD, Herbison GP, Gee H, Lang GD, Lancashire R. Obstetric practice and faecal incontinence three months after delivery. Bjog 2001;108(7):678-83.
30. Fenner DE, Genberg B, Brahma P, Marek L, DeLancey JO. Fecal and urinary incontinence after vaginal delivery with anal sphincter disruption in an obstetrics unit in the United States. Am J Obstet Gynecol 2003;189(6):1543-9; discussion 1549-50.
31. Jorge JM, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum 1993;36(1):77-97.
32. Rothbarth J, Bemelman WA, Meijerink WJ, Stiggelbout AM, Zwinderman AH, Buyze-Westerweel ME, Delemarre JB. What is the impact of fecal incontinence on quality of life? Dis Colon Rectum 2001;44(1):67-71.
33. Fynes M, Donnelly VS, O'Connell PR, O'Herlihy C. Cesarean delivery and anal sphincter injury. Obstet Gynecol 1998;92(4 Pt 1):496-500.
34. Nelson RL, Westercamp M, Furner SE. A systematic review of the efficacy of cesarean section in the preservation of anal continence. Dis Colon Rectum 2006;49(10):1587-95.
35. Nygaard IE, Rao SS, Dawson JD. Anal incontinence after anal sphincter disruption: a 30-year retrospective cohort study. Obstet Gynecol 1997;89(6):896-901.
36. Faltin DL, Otero M, Petignat P, Sangalli MR, Floris LA, Boulvain M, Irion O. Women's health 18 years after rupture of the anal sphincter during childbirth: I. Fecal incontinence. Am J Obstet Gynecol 2006;194(5):1255-9.
37. Bollard RC, Gardiner A, Duthie GS, Lindow SW. Anal sphincter injury, fecal and urinary incontinence: a 34-year follow-up after forceps delivery. Dis Colon Rectum 2003;46(8):1083-8.

38. Eogan M, O'Brien C, Behan M, O'Connell PR, O'Herlihy C. Long-term effects of childbirth on fecal continence and pudendal nerve function [abstract]. Am J Obstet Gynecol 2003;189(6, Supplement 1):S72.
39. Mous M, Muller SA, de Leeuw JW. Long-term effects of anal sphincter rupture during vaginal delivery: faecal incontinence and sexual complaints. Bjog 2008;115(2):234-8.
40. Fornell EU, Matthiesen L, Sjodahl R, Berg G. Obstetric anal sphincter injury ten years after: subjective and objective long term effects. Bjog 2005;112(3):312-6.
41. Fleshman JW, Peters WR, Shemesh EI, Fry RD, Kodner IJ. Anal sphincter reconstruction: anterior overlapping muscle repair. Dis Colon Rectum 1991;34(9):739-43.
42. Engel AF, Kamm MA, Sultan AH, Bartram CI, Nicholls RJ. Anterior anal sphincter repair in patients with obstetric trauma. Br J Surg 1994;81(8):1231-4.
43. Londono-Schimmer EE, Garcia-Duperly R, Nicholls RJ, Ritchie JK, Hawley PR, Thomson JP. Overlapping anal sphincter repair for faecal incontinence due to sphincter trauma: five year follow-up functional results. Int J Colorectal Dis 1994;9(2):110-3.
44. Oliveira L, Pfeifer J, Wexner SD. Physiological and clinical outcome of anterior sphincteroplasty. Br J Surg 1996;83(4):502-5.
45. Gilliland R, Altomare DF, Moreira H, Jr., Oliveira L, Gilliland JE, Wexner SD. Pudendal neuropathy is predictive of failure following anterior overlapping sphincteroplasty. Dis Colon Rectum 1998;41(12):1516-22.
46. Young CJ, Mathur MN, Eyers AA, Solomon MJ. Successful overlapping anal sphincter repair: relationship to patient age, neuropathy, and colostomy formation. Dis Colon Rectum 1998;41(3):344-9.
47. Karoui S, Leroi AM, Koning E, Menard JF, Michot F, Denis P. Results of sphincteroplasty in 86 patients with anal incontinence. Dis Colon Rectum 2000;43(6):813-20.
48. Osterberg A, Edebol Eeg-Olofsson K, Graf W. Results of surgical treatment for faecal incontinence. Br J Surg 2000;87(11):1546-52.
49. Morren GL, Hallbook O, Nystrom PO, Baeten CG, Sjodahl R. Audit of anal-sphincter repair. Colorectal Dis 2001;3(1):17-22.
50. Tan M, O'Hanlon DM, Cassidy M, O'Connell PR. Advantages of a posterior fourchette incision in anal sphincter repair. Dis Colon Rectum 2001;44(11):1624-9.
51. Bravo Gutierrez A, Madoff RD, Lowry AC, Parker SC, Buie WD, Baxter NN. Long-term results of anterior sphincteroplasty. Dis Colon Rectum 2004;47(5):727-31; discussion 731-2.
52. Norderval S, Oian P, Revhaug A, Vonen B. Anal incontinence after obstetric sphincter tears: outcome of anatomic primary repairs. Dis Colon Rectum 2005;48(5):1055-61.
53. Zorcolo L, Covotta L, Bartolo DC. Outcome of anterior sphincter repair for obstetric injury: comparison of early and late results. Dis Colon Rectum 2005;48(3):524-31.
54. Trowbridge ER, Morgan D, Trowbridge MJ, Delancey JO, Fenner DE. Sexual function, quality of life, and severity of anal incontinence after anal sphincteroplasty. Am J Obstet Gynecol 2006;195(6):1753-7.
55. Barisic GI, Krivokapic ZV, Markovic VA, Popovic MA. Outcome of overlapping anal sphincter repair after 3 months and after a mean of 80 months. Int J Colorectal Dis 2006;21(1):52-6.
56. Madoff RD. Surgical treatment options for fecal incontinence. Gastroenterology 2004;126(1 Suppl 1):S48-54.
57. Rockwood TH, Church JM, Fleshman JW, Kane RL, Mavrantonis C, Thorson AG, Wexner SD, Bliss D, Lowry AC. Patient and surgeon ranking of the severity of symptoms associated with fecal incontinence: the fecal incontinence severity index. Dis Colon Rectum 1999;42(12):1525-32.
58. Vaizey CJ, Carapeti E, Cahill JA, Kamm MA. Prospective comparison of faecal incontinence grading systems. Gut 1999;44(1):77-80.
59. Maeda Y, Pares D, Norton C, Vaizey CJ, Kamm MA. Does the St. Mark's incontinence score reflect patients' perceptions? A review of 390 patients. Dis Colon Rectum 2008;51(4):436-42.
60. Mortele KJ, Fairhurst J. Dynamic MR defecography of the posterior compartment: Indications, techniques and MRI features. Eur J Radiol 2007;61(3):462-72.
61. Altringer WE, Saclarides TJ, Dominguez JM, Brubaker LT, Smith CS. Four-contrast defecography: pelvic "floor-oscopy". Dis Colon Rectum 1995;38(7):695-9.
62. Donnelly VS, O'Herlihy C, Campbell DM, O'Connell PR. Postpartum fecal incontinence is more common in women with irritable bowel syndrome. Dis Colon Rectum 1998;41(5):586-9.
63. Fitzpatrick M, O'Brien C, O'Connell P R, O'Herlihy C. Patterns of abnormal pudendal nerve function that are associated with postpartum fecal incontinence. Am J Obstet Gynecol 2003;189(3):730-5.
64. Fynes MM, Marshall K, Cassidy M, Behan M, Walsh D, O'Connell PR, O'Herlihy C. A prospective, randomized study comparing the effect of augmented biofeedback with sensory biofeedback alone on fecal incontinence after obstetric trauma. Dis Colon Rectum 1999;42(6):753-8; discussion 758-61.
65. Mahony RT, Malone PA, Nalty J, Behan M, O'Connell P R, O'Herlihy C. Randomized clinical trial of intra-anal electromyographic biofeedback physiotherapy with intra-anal electromyographic biofeedback augmented with electrical stimulation of the anal sphincter in the early treatment of postpartum fecal incontinence. Am J Obstet Gynecol 2004;191(3):885-90.
66. Norton C, Cody JD, Hosker G. Biofeedback and/or sphincter exercises for the treatment of faecal incontinence in adults. Cochrane Database Syst Rev 2006;3:CD002111.
67. Hull TL, Bartus C, Bast J, Floruta C, Lopez R. Multimedia article. Success of episioproctotomy for cloaca and rectovaginal fistula. Dis Colon Rectum 2007;50(1):97-101.
68. Kaiser AM. Multimedia article. Anovaginal reconstruction with bilateral x-flaps and sphincteroplasty for cloaca-like deformity after obstetrical injury. Dis Colon Rectum 2007;50(10):1707.
69. Parks AG, McPartlin JF. Late repair of injuries of the anal sphincter. Proc R Soc Med 1971;64(12):1187-9.
70. Tjandra JJ, Han WR, Goh J, Carey M, Dwyer P. Direct repair vs. overlapping sphincter repair: a randomized, controlled trial. Dis Colon Rectum 2003;46(7):937-42; discussion 942-3.
71. Pinedo G, Vaizey CJ, Nicholls RJ, Roach R, Halligan S, Kamm MA. Results of repeat anal sphincter repair. Br J Surg 1999;86(1):66-9.
72. Nielsen MB, Dammegaard L, Pedersen JF. Endosonographic assessment of the anal sphincter after surgical reconstruction. Dis Colon Rectum 1994;37(5):434-8.
73. Giordano P, Renzi A, Efron J, Gervaz P, Weiss EG, Nogueras JJ, Wexner SD. Previous sphincter repair does not affect the outcome of repeat repair. Dis Colon Rectum 2002;45(5):635-40.
74. Vaizey CJ, Norton C, Thornton MJ, Nicholls RJ, Kamm MA. Long-term results of repeat anterior anal sphincter repair. Dis Colon Rectum 2004;47(6):858-63.
75. Hull T. Invited editorial. Dis Colon Rectum 2004;47(5):731-732.
76. Evans C, Davis K, Kumar D. Overlapping anal sphincter repair and anterior levatorplasty: effect of patient's age and duration of follow-up. Int J Colorectal Dis 2006;21(8):795-801.

77. Gearhart S, Hull T, Floruta C, Schroeder T, Hammel J. Anal manometric parameters: predictors of outcome following anal sphincter repair? J Gastrointest Surg 2005;9(1):115-20.

78. Maslekar S, Gardiner AB, Duthie GS. Anterior anal sphincter repair for fecal incontinence: Good longterm results are possible. J Am Coll Surg 2007;204(1):40-6.

79. Bachoo P, Brazzelli M, Grant A. Surgery for faecal incontinence in adults. Cochrane Database Syst Rev 2000(2):CD001757.

80. Deen KI, Oya M, Ortiz J, Keighley MR. Randomized trial comparing three forms of pelvic floor repair for neuropathic faecal incontinence. Br J Surg 1993;80(6):794-8.

81. van Tets WF, Kuijpers JH. Pelvic floor procedures produce no consistent changes in anatomy or physiology. Dis Colon Rectum 1998;41(3):365-9.

82. Womack NR, Morrison JF, Williams NS. Prospective study of the effects of postanal repair in neurogenic faecal incontinence. Br J Surg 1988;75(1):48-52.

83. Orrom WJ, Miller R, Cornes H, Duthie G, Mortensen NJ, Bartolo DC. Comparison of anterior sphincteroplasty and postanal repair in the treatment of idiopathic fecal incontinence. Dis Colon Rectum 1991;34(4):305-10.

84. Browning GG, Parks AG. Postanal repair for neuropathic faecal incontinence: correlation of clinical result and anal canal pressures. Br J Surg 1983;70(2):101-4.

85. Scheuer M, Kuijpers HC, Jacobs PP. Postanal repair restores anatomy rather than function. Dis Colon Rectum 1989;32(11):960-3.

86. Yoshioka K, Keighley MR. Critical assessment of the quality of continence after postanal repair for faecal incontinence [published erratum appears in Br J Surg 1990 Mar;77(3):356]. Br J Surg 1989;76(10):1054-7.

87. Engel AF, van Baal SJ, Brummelkamp WH. Late results of postanal repair for idiopathic faecal incontinence. Eur J Surg 1994;160(11):637-40.

88. Jameson JS, Speakman CT, Darzi A, Chia YW, Henry MM. Audit of postanal repair in the treatment of fecal incontinence. Dis Colon Rectum 1994;37(4):369-72.

89. Setti Carraro P, Kamm MA, Nicholls RJ. Long-term results of postanal repair for neurogenic faecal incontinence. Br J Surg 1994;81(1):140-4.

90. Matsuoka H, Mavrantonis C, Wexner SD, Oliveira L, Gilliland R, Pikarsky A. Postanal repair for fecal incontinence—is it worthwhile? Dis Colon Rectum 2000;43(11):1561-7.

91. Abbas SM, Bissett IP, Neill ME, Parry BR. Long-term outcome of postanal repair in the treatment of faecal incontinence. ANZ J Surg 2005;75(9):783-6.

92. Keighley MR. Postanal repair for faecal incontinence. J R Soc Med 1984;77(4):285-8.

93. Ferguson EF, Jr. Puborectalis sphincteroplasty for anal incontinence. South Med J 1984;77(4):423-5.

94. van Vroonhoven TJ, Schouten WR. Postanal repair in the treatment of faecal incontinence. Neth J Surg 1984;36(6):160-2.

95. Henry MM, Simson JN. Results of postanal repair: a retrospective study. Br J Surg 1985;72(Suppl):S17-9.

96. Habr-Gama A, Alves PA, da Silva e Souza AH, Femenia Viera MJ, Brunetti-Netto C. Treatment of faecal incontinence by postanal repair. *Coloproctology 8*, 1986:244-246.

97. Rainey JB, Donaldson DR, Thomson JP. Postanal repair: which patients derive most benefit? J R Coll Surg Edinb 1990;35(2):101-5.

98. Scott AD, Henry MM, Phillips RK. Clinical assessment and anorectal manometry before postanal repair: failure to predict outcome. Br J Surg 1990;77(6):628-9.

99. Laurberg S, Swash M, Henry MM. Effect of postanal repair on progress of neurogenic damage to the pelvic floor. British Journal of Surgery 1990;77:519-522.

100. Rieger NA, Sarre RG, Saccone GT, Hunter A, Toouli J. Postanal repair for faecal incontinence: long-term follow-up. Aust N Z J Surg 1997;67(8):566-70.

101. Yoshioka K, Hyland G, Keighley MR. Physiological changes after postanal repair and parameters predicting outcome. Br J Surg 1988;75(12):1220-4.

102. Chetwood CH. Plastic operation for restoration of the sphicter ani with a report of a case. Med Rec 1902;61:529.

103. Schoemaker J. Un nouveau procédé opératoire pour la reconstitution du sphincter anal. Sem Med 1909;29:160.

104. Pickrell KL, Broadbent TR, Masters FW, Metzger JT. Construction of a rectal sphincter and restoration of anal continence by transplanting the gracilis muscle; a report of four cases in children. Annals of Surgery 1952;135(6):853-862.

105. Raffensperger J. The gracilis sling for fecal incontinence. J Pediatr Surg 1979;14(6):794-7.

106. Brandesky G, Holschneider AM. Operations for the improvement of faecal incontinence. Prog Pediatr Surg 1976;9:105-14.

107. Schwedler T, Erichsen K. [Gracilis-muscle transplant for the treatment of fecal incontinence (author's transl)]. Langenbecks Arch Chir 1975;339:451-7.

108. Holschneider AM, Hecker WC. Smooth muscle reverse plasty. A new method to treat anorectal incontinence in infants with high anal and rectal atresia. Results after gracilis plasty and free muscle transplantation. Prog Pediatr Surg 1984;17:131-45.

109. Corman ML. Gracilis muscle transposition for anal incontinence: late results. Br J Surg 1985;72 Suppl:S21-2.

110. Leguit P, Van Baal JG, Brummelkamp WH. Gracilis muscle transposition in the treatment of fecal incontinence. Long-term follow-up and evaluation of anal pressure recordings. Dis Colon Rectum 1985;28(1):1-4.

111. Scharli AF. Anorectal incontinence: diagnosis and treatment. J Pediatr Surg 1987;22(8):693-701.

112. Yoshioka K, Keighley MRB. Clinical and manometric assessment of gracilis muscle transplant for fecal incontinence. Diseases of the Colon & Rectum 1988;31(10):767-769.

113. Sielezneff I, Bauer S, Bulgare JC, Sarles JC. Gracilis muscle transposition in the treatment of faecal incontinence. Int J Colorectal Dis 1996;11(1):15-8.

114. Kotobi H, Forin V, Larroquet M, Khairouni A, Loc'h P, Grapin C, Audry G. [Pickrell intervention in children for anal incontinence secondary to anorectal malformation]. Ann Chir 2000;125(10):954-60.

115. Faucheron JL, Hannoun L, Thome C, Parc R. Is fecal continence improved by nonstimulated gracilis muscle transposition? Dis Colon Rectum 1994;37(10):979-83.

116. Kumar D, Hutchinson R, Grant E. Bilateral gracilis neosphincter construction for treatment of faecal incontinence. Br J Surg 1995;82(12):1645-7.

117. Prochiantz A, Gross P. Gluteal myoplasty for sphincter replacement: principles, results and prospects. J Pediatr Surg 1982;17(1):25-30.

118. Christiansen J, Hansen CR, Rasmussen O. Bilateral gluteus maximus transposition for anal incontinence. Br J Surg 1995;82(7):903-5.

119. Guelinckx PJ, Sinsel NK, Gruwez JA. Anal sphincter reconstruction with the gluteus maximus muscle: anatomic and physiologic considerations concerning conventional

and dynamic gluteoplasty. Plast Reconstr Surg 1996;98(2):293-302; discussion 303-4.

120. Devesa JM, Madrid JM, Gallego BR, Vicente E, Nuno J, Enriquez JM. Bilateral gluteoplasty for fecal incontinence. Dis Colon Rectum 1997;40(8):883-8.
121. Pearl RK, Prasad ML, Nelson RL, Orsay CP, Abcarian H. Bilateral gluteus maximus transposition for anal incontinence. Diseases of the Colon & Rectum 1991;34:478-481.
122. Yoshioka K, Ogunbiyi OA, Keighley MR. A pilot study of total pelvic floor repair or gluteus maximus transposition for postobstetric neuropathic fecal incontinence. Dis Colon Rectum 1999;42(2):252-7.
123. Hultman CS, Zenn MR, Agarwal T, Baker CC. Restoration of fecal continence after functional gluteoplasty: long-term results, technical refinements, and donor-site morbidity. Ann Plast Surg 2006;56(1):65-70; discussion 70-1.
124. George BD, Williams NS, Patel J, Swash M, Watkins ES. Physiological and histochemical adaptation of the electrically stimulated gracilis muscle to neoanal sphincter function. Br J Surg 1993;80(10):1342-6.
125. Baeten C, Spaans F, Fluks A. An implanted neuromuscular stimulator for fecal continence following previously implanted gracilis muscle: report of a case. Dis Colon Rectum 1988;31(2):134-7.
126. Baeten CG, Konsten J, Spaans F, Visser R, Habets AM, Bourgeois IM, Wagenmakers AJ, Soeters PB. Dynamic graciloplasty for treatment of faecal incontinence. Lancet 1991;338(8776):1163-5.
127. Williams NS, Patel J, George BD, Hallan RI, Watkins ES. Development of an electrically stimulated neoanal sphincter. Lancet 1991;338:1166-1169.
128. Konsten J, Baeten CGMI, Havenith MG, Soeters PB. Morphology of dynamic graciloplasty compared with the anal sphincter. Dis Colon Rectum 1993;36(6):559-563.
129. Salmons S, Vrbova G. The influence of activity on some contractile characteristics of mammalian fast and slow muscles. Journal of Physiology 1969;201:535-549.
130. Patel J, Shanahan D, Williams N, Sinnatamby C, George B, Watkins E. The anatomy of the anterior division of the obdurator nerve in relation to the electrically stimulated gracilis neoanal sphincter [abstract]. Clin Anat 1991;4(5):385.
131. Baeten GM, Geerdes BP, Adang EM, Heineman E, Konsten J, Engel GL, Kester ADM, Spaans F, Soeters PB. Anal dynamic graciloplasty in the treatment of intractable fecal incontinence. N Engl J Med 1995;332(24):1600-1605.
132. Geerdes BP, Heineman E, Konsten J, Soeters PB, Baeten CG. Dynamic graciloplasty. Complications and management. Dis Colon Rectum 1996;39(8):912-7.
133. Cavina E, Seccia M, Banti P, Zocco G. Anorectal reconstruction after abdominoperineal resection. Experience with double-wrap graciloplasty supported by low-frequency electrostimulation. Dis Colon Rectum 1998;41(8):1010-6.
134. Madoff RD, Rosen HR, Baeten CG, LaFontaine LJ, Cavina E, Devesa M, Rouanet P, Christiansen J, Faucheron JL, Isbister W, Kohler L, Guelinckx PJ, Pahlman L. Safety and efficacy of dynamic muscle plasty for anal incontinence: lessons from a prospective, multicenter trial. Gastroenterology 1999;116(3):549-56.
135. Mander BJ, Wexner SD, Williams NS, Bartolo DC, Lubowski DZ, Oresland T, Romano G, Keighley MR. Preliminary results of a multicentre trial of the electrically stimulated gracilis neoanal sphincter. Br J Surg 1999;86(12):1543-8.
136. Baeten CG, Bailey HR, Bakka A, Belliveau P, Berg E, Buie WD, Burnstein MJ, Christiansen J, Coller JA, Galandiuk S, LaFontaine LJ, Lange J, Madoff RD, Matzel KE, Pahlman L, Parc R, Reilly JC, Seccia M, Thorson AG, Vernava AM, 3rd, Wexner S. Safety and efficacy of dynamic graciloplasty for fecal incontinence: report of a prospective, multicenter trial. Dynamic Graciloplasty Therapy Study Group. Dis Colon Rectum 2000;43(6):743-51.
137. Matzel KE, Madoff RD, LaFontaine LJ, Baeten CG, Buie WD, Christiansen J, Wexner S. Complications of dynamic graciloplasty: incidence, management, and impact on outcome. Dis Colon Rectum 2001;44(10):1427-35.
138. Wexner SD, Baeten C, Bailey R, Bakka A, Belin B, Belliveau P, Berg E, Buie WD, Burnstein M, Christiansen J, Coller J, Galandiuk S, Lange J, Madoff R, Matzel KE, Pahlman L, Parc R, Reilly J, Seccia M, Thorson AG, Vernava AM, 3rd. Long-term efficacy of dynamic graciloplasty for fecal incontinence. Dis Colon Rectum 2002;45(6):809-18.
139. Rongen MJ, Uludag O, El Naggar K, Geerdes BP, Konsten J, Baeten CG. Long-term follow-up of dynamic graciloplasty for fecal incontinence. Dis Colon Rectum 2003;46(6):716-21.
140. Penninckx F. Belgian experience with dynamic graciloplasty for faecal incontinence. Br J Surg 2004;91(7):872-8.
141. Tillin T, Gannon K, Feldman RA, Williams NS. Third-party prospective evaluation of patient outcomes after dynamic graciloplasty. Br J Surg 2006;93(11):1402-10.
142. Rosen HR, Novi G, Zoech G, Feil W, Urbarz C, Schiessel R. Restoration of anal sphincter function by single-stage dynamic graciloplasty with a modified (split sling) technique. Am J Surg 1998;175(3):187-93.
143. Sielezneff I, Malouf AJ, Bartolo DC, Pryde A, Douglas S. Dynamic graciloplasty in the treatment of patients with faecal incontinence. Br J Surg 1999;86(1):61-5.
144. Thornton MJ, Kennedy ML, Lubowski DZ, King DW. Long-term follow-up of dynamic graciloplasty for faecal incontinence. Colorectal Dis 2004;6(6):470-6.
145. Chapman AE, Geerdes B, Hewett P, Young J, Eyers T, Kiroff G, Maddern GJ. Systematic review of dynamic graciloplasty in the treatment of faecal incontinence. Br J Surg 2002;89(2):138-53.
146. Tan EK, Vaizey C, Cornish J, Darzi A, Tekkis PP. Surgical strategies for faecal incontinence—a decision analysis between dynamic graciloplasty, artificial bowel sphincter and end stoma. Colorectal Dis 2008;10(6):577-86.
147. Scott FB, Bradley WE, Timm GW. Treatment of urinary incontinence by an implantable prosthetic urinary sphincter. J Urol 1974;112(1):75-80.
148. Christiansen J, Lorentzen M. Implantation of artificial sphincter for anal incontinence. Lancet 1987;2(8553):244-5.
149. Christiansen J, Sparso B. Treatment of anal incontinence by an implantable prosthetic anal sphincter. Ann Surg 1992;215(4):383-6.
150. O'Brien PE, Dixon JB, Skinner S, Laurie C, Khera A, Fonda D. A prospective, randomized, controlled clinical trial of placement of the artificial bowel sphincter (Acticon Neosphincter) for the control of fecal incontinence. Dis Colon Rectum 2004;47(11):1852-60.
151. Ortiz H, Armendariz P, DeMiguel M, Solana A, Alos R, Roig JV. Prospective study of artificial anal sphincter and dynamic graciloplasty for severe anal incontinence. Int J Colorectal Dis 2003;18(4):349-54.
152. Vaizey CJ, Kamm MA, Gold DM, Bartram CI, Halligan S, Nicholls RJ. Clinical, physiological, and radiological study of a new purpose- designed artificial bowel sphincter. Lancet 1998;352(9122):105-9.
153. Lehur PA, Roig JV, Duinslaeger M. Artificial anal sphincter: prospective clinical and manometric evaluation. Dis Colon Rectum 2000;43(8):1100-6.

154. Dodi G, Melega E, Masin A, Infantino A, Cavallari F, Lise M. Artificial bowel sphincter (ABS) for severe faecal incontinence: a clinical and manometric study. Colorectal Dis 2000;2:207-11.

155. Devesa JM, Rey A, Hervas PL, Halawa KS, Larranaga I, Svidler L, Abraira V, Muriel A. Artificial anal sphincter: complications and functional results of a large personal series. Dis Colon Rectum 2002;45(9):1154-63.

156. Wong WD, Congliosi SM, Spencer MP, Corman ML, Tan P, Opelka FG, Burnstein M, Nogueras JJ, Bailey HR, Devesa JM, Fry RD, Cagir B, Birnbaum E, Fleshman JW, Lawrence MA, Buie WD, Heine J, Edelstein PS, Gregorcyk S, Lehur PA, Michot F, Phang PT, Schoetz DJ, Potenti F, Tsai JY. The safety and efficacy of the artificial bowel sphincter for fecal incontinence: results from a multicenter cohort study. Dis Colon Rectum 2002;45(9):1139-53.

157. Ortiz H, Armendariz P, DeMiguel M, Ruiz MD, Alos R, Roig JV. Complications and functional outcome following artificial anal sphincter implantation. Br J Surg 2002;89(7):877-81.

158. Lehur PA, Zerbib F, Neunlist M, Glemain P, Bruley des Varannes S. Comparison of quality of life and anorectal function after artificial sphincter implantation. Dis Colon Rectum 2002;45(4):508-13.

159. Parker SC, Spencer MP, Madoff RD, Jensen LL, Wong WD, Rothenberger DA. Artificial bowel sphincter: long-term experience at a single institution. Dis Colon Rectum 2003;46(6):722-9.

160. Casal E, San Ildefonso A, Carracedo R, Facal C, Sanchez JA. Artificial bowel sphincter in severe anal incontinence. Colorectal Dis 2004;6(3):180-4.

161. Medicare Services Advisory Committee. Placement of artificial bowel sphincters in the management of faecal incontinence. Assessment report December 1999. Available at: http://www.health.gov.au/msac/pdfs/reports/msac1023.pdf. Accessed June 2, 2004.

162. Mundy L, Merlin TL, Maddern GJ, Hiller JE. Systematic review of safety and effectiveness of an artificial bowel sphincter for faecal incontinence. Br J Surg 2004;91(6):665-72.

163. Belyaev O, Muller C, Uhl W. Neosphincter surgery for fecal incontinence: a critical and unbiased review of the relevant literature. Surg Today 2006;36(4):295-303.

164. da Silva GM, Jorge JM, Belin B, Nogueras JJ, Weiss EG, Vernava AM, 3rd, Habr-Gama A, Wexner SD. New surgical options for fecal incontinence in patients with imperforate anus. Dis Colon Rectum 2004;47(2):204-209.

165. O'Brien PE, Skinner S. Restoring control: the Acticon Neosphincter artificial bowel sphincter in the treatment of anal incontinence. Dis Colon Rectum 2000;43(9):1213-6.

166. 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. Br J Surg 2001;88(11):1481-6.

167. Michot F, Tuech JJ, Lefebure B, Bridoux V, Denis P. A new implantation procedure of artificial sphincter for anal incontinence: the transvaginal approach. Dis Colon Rectum 2007;50(9):1401-4.

168. Malouf AJ, Vaizey CJ, Kamm MA, Nicholls RJ. Reassessing artificial bowel sphincters. Lancet 2000;355(9222):2219-20.

169. Michot F, Costaglioli B, Leroi AM, Denis P. Artificial anal sphincter in severe fecal incontinence: outcome of prospective experience with 37 patients in one institution. Ann Surg 2003;237(1):52-6.

170. La Torre F, Masoni L, Montori J, Ruggeri E, Montori A. The surgical treatment of fecal incontinence with artificial anal sphincter implant. Preliminary clinical report. Hepato-gastroenterology 2004;51(59):1358-61.

171. Altomare DF, Binda GA, Dodi G, La Torre F, Romano G, Rinaldi M, Melega E. Disappointing long-term results of the artificial anal sphincter for faecal incontinence. Br J Surg 2004;91(10):1352-3.

172. Colquhoun PH, Efron J, Wexner SD. Attainment of continence with J-pouch and artificial bowel sphincter for concomitant imperforate anus and familial adenomatous polyposis: report of a case. Dis Colon Rectum 2004;47 (4):538-41.

173. Benoist S, Panis Y, Michot F, Coffin B, Messing B, Valleur P. Artificial sphincter with colonic reservoir for severe anal incontinence because of imperforate anus and short-bowel syndrome: report of a case. Dis Colon Rectum 2005;48 (10):1978-82.

174. Clarke MD, Sagar PM, Lim M, Macklin C. Restoration of anal continence after ileal pouch anal procedure with partial excision of anal sphincter by means of an artificial bowel sphincter. Colorectal Dis 2005;7(5):527-8.

175. Romano G, La Torre F, Cutini G, Bianco F, Esposito P. Total anorectal reconstruction with an artificial bowel sphincter: Report of five cases with a minimum follow-up of 6 months. Colorectal Dis 2002;4(5):339-344.

176. Romano G, La Torre F, Cutini G, Bianco F, Esposito P, Montori A. Total anorectal reconstruction with the artificial bowel sphincter: report of eight cases. A quality-of-life assessment. Dis Colon Rectum 2003;46(6):730-4.

177. Marchal F, Doucet C, Lechaux D, Lasser P, Lehur PA. Secondary implantation of an artificial sphincter after abdominoperineal resection and pseudocontinent perineal colostomy for rectal cancer. Gastroenterol Clin Biol 2005;29(4):425-8.

178. Devesa JM, Lopez-Hervas P, Vicente R, Rey A, Die J, Fraile A. Total anorectal reconstruction: a novel technique. Tech Coloproctol 2005;9(2):149-52.

179. Hajivassiliou CA, Carter KB, Finlay IG. Biomechanical evaluation of an artificial anal sphincter prosthesis. J Med Eng Technol 1997;21(3-4):89-95.

180. Finlay IG, Richardson W, Hajivassiliou CA. Outcome after implantation of a novel prosthetic anal sphincter in humans. Br J Surg 2004;91(11):1485-92.

181. Matzel KE, Stadelmaier U, Hohenfellner M, Gall FP. Electrical stimulation of sacral spinal nerves for treatment of faecal incontinence. Lancet 1995;346(8983):1124-7.

182. Matzel KE, Schmidt RA, Tanagho EA. Neuroanatomy of the striated muscular anal continence mechanism. Implications for the use of neurostimulation. Dis Colon Rectum 1990;33(8):666-73.

183. Thon WF, Baskin LS, Jonas U, Tanagho EA, Schmidt RA. Neuromodulation of voiding dysfunction and pelvic pain. World Journal of Urology 1991;9:138-141.

184. Janknegt RA, Weil EH, Eerdmans PH. Improving neuromodulation technique for refractory voiding dysfunctions: two-stage implant. Urology 1997;49(3):358-62.

185. Matzel KE, Stadelmaier U, Bittorf B, Hohenfellner M, Hohenberger W. Bilateral sacral spinal nerve stimulation for fecal incontinence after low anterior rectum resection. Int J Colorectal Dis 2002;17(6):430-4.

186. Ratto C, Grillo E, Parello A, Petrolino M, Costamagna G, Doglietto GB. Sacral neuromodulation in treatment of fecal incontinence following anterior resection and chemoradiation for rectal cancer. Dis Colon Rectum 2005;48(5):1027-36.

187. Matzel KE, Stadelmaier U, Hohenberger W. Innovations in fecal incontinence: sacral nerve stimulation. Dis Colon Rectum 2004;47(10):1720-8.

188. Tjandra JJ, Lim JF, Matzel K. Sacral nerve stimulation: an emerging treatment for faecal incontinence. ANZ J Surg 2004;74(12):1098-106.

189. Gourcerol G, Gallas S, Michot F, Denis P, Leroi AM. Sacral nerve stimulation in fecal incontinence: are there factors associated with success? Dis Colon Rectum 2007;50(1):3-12.

190. Dudding TC, Pares D, Vaizey CJ, Kamm MA. Predictive factors for successful sacral nerve stimulation in the treatment of faecal incontinence: a 10-year cohort analysis. Colorectal Dis 2008;10(3):249-56.

191. Rosen HR, Urbarz C, Holzer B, Novi G, Schiessel R. Sacral nerve stimulation as a treatment for fecal incontinence. Gastroenterology 2001;121(3):536-41.

192. Ganio E, Ratto C, Masin A, Luc AR, Doglietto GB, Dodi G, Ripetti V, Arullani A, Frascio M, BertiRiboli E, Landolfi V, DelGenio A, Altomare DF, Memeo V, Bertapelle P, Carone R, Spinelli M, Zanollo A, Spreafico L, Giardiello G, de Seta F. Neuromodulation for fecal incontinence: outcome in 16 patients with definitive implant. The initial Italian Sacral Neurostimulation Group (GINS) experience. Dis Colon Rectum 2001;44(7):965-70.

193. Jarrett ME, Varma JS, Duthie GS, Nicholls RJ, Kamm MA. Sacral nerve stimulation for faecal incontinence in the UK. Br J Surg 2004;91(6):755-61.

194. Melenhorst J, Koch SM, Uludag O, van Gemert WG, Baeten CG. Sacral neuromodulation in patients with faecal incontinence: results of the first 100 permanent implantations. Colorectal Dis 2007;9(8):725-30.

195. Ganio E, Luc AR, Clerico G, Trompetto M. Sacral nerve stimulation for treatment of fecal incontinence: a novel approach for intractable fecal incontinence. Dis Colon Rectum 2001;44(5):619-29; discussion 629-31.

196. Kenefick NJ, Vaizey CJ, Cohen RC, Nicholls RJ, Kamm MA. Medium-term results of permanent sacral nerve stimulation for faecal incontinence. Br J Surg 2002;89(7):896-901.

197. Matzel KE, Stadelmaier U, Hohenfellner M, Hohenberger W. Chronic sacral spinal nerve stimulation for fecal incontinence: long- term results with foramen and cuff electrodes. Dis Colon Rectum 2001;44(1):59-66.

198. Leroi AM, Michot F, Grise P, Denis P. Effect of sacral nerve stimulation in patients with fecal and urinary incontinence. Dis Colon Rectum 2001;44(6):779-89.

199. Kenefick NJ, Emmanuel A, Nicholls RJ, Kamm MA. Effect of sacral nerve stimulation on autonomic nerve function. Br J Surg 2003;90(10):1256-60.

200. Vaizey CJ, Kamm MA, Turner IC, Nicholls RJ, Woloszko J. Effects of short term sacral nerve stimulation on anal and rectal function in patients with anal incontinence. Gut 1999;44(3):407-12.

201. Uludag O, Koch SM, van Gemert WG, Dejong CH, Baeten CG. Sacral neuromodulation in patients with fecal incontinence: a single-center study. Dis Colon Rectum 2004;47(8):1350-7.

202. Sheldon R, Kiff ES, Clarke A, Harris ML, Hamdy S. Sacral nerve stimulation reduces corticoanal excitability in patients with faecal incontinence. Br J Surg 2005;92(11):1423-31.

203. Gooneratne ML, Facer P, Knowles CH, Chan CL, Lunniss PJ, Scott SM, Anand P, Williams NS. Normalization of substance P levels in rectal mucosa of patients with faecal incontinence treated successfully by sacral nerve stimulation. Br J Surg 2008;95(4):477-83.

204. Malouf AJ, Vaizey CJ, Nicholls RJ, Kamm MA. Permanent sacral nerve stimulation for fecal incontinence. Ann Surg 2000;232(1):143-8.

205. Ripetti V, Caputo D, Ausania F, Esposito E, Bruni R, Arullani A. Sacral nerve neuromodulation improves physical, psychological and social quality of life in patients with fecal incontinence. Tech Coloproctol 2002;6(3):147-52.

206. Matzel KE, Bittorf B, Stadelmaier U, Hohenberger W. [Sacral nerve stimulation in the treatment of faecal incontinence]. Chirurg 2003;74(1):26-32.

207. Altomare DF, Rinaldi M, Petrolino M, Monitillo V, Sallustio P, Veglia A, De Fazio M, Guglielmi A, Memeo V. Permanent sacral nerve modulation for fecal incontinence and associated urinary disturbances. Int J Colorectal Dis 2004;19(3):203-9.

208. Matzel KE, Kamm MA, Stosser M, Baeten CG, Christiansen J, Madoff R, Mellgren A, Nicholls RJ, Rius J, Rosen H. Sacral spinal nerve stimulation for faecal incontinence: multicentre study. Lancet 2004;363(9417):1270-6.

209. Rasmussen OO, Buntzen S, Sorensen M, Laurberg S, Christiansen J. Sacral nerve stimulation in fecal incontinence. Dis Colon Rectum 2004;47(7):1158-62; discussion 1162-3.

210. Leroi AM, Parc Y, Lehur PA, Mion F, Barth X, Rullier E, Bresler L, Portier G, Michot F. Efficacy of sacral nerve stimulation for fecal incontinence: results of a multicenter double-blind crossover study. Ann Surg 2005;242(5):662-9.

211. Kenefick NJ. Sacral nerve neuromodulation for the treatment of lower bowel motility disorders. Ann R Coll Surg Engl 2006;88(7):617-23.

212. Holzer B, Rosen HR, Novi G, Ausch C, Holbling N, Schiessel R. Sacral nerve stimulation for neurogenic faecal incontinence. Br J Surg 2007;94(6):749-53.

213. Hetzer FH, Hahnloser D, Clavien PA, Demartines N. Quality of life and morbidity after permanent sacral nerve stimulation for fecal incontinence. Arch Surg 2007;142(1):8-13.

214. Tan JJ, Chan M, Tjandra JJ. Evolving therapy for fecal incontinence. Dis Colon Rectum 2007;50(11):1950-67.

215. Melenhorst J, Koch SM, Uludag O, van Gemert WG, Baeten CG. Is a morphologically intact anal sphincter necessary for success with sacral nerve modulation in patients with faecal incontinence? Colorectal Dis 2008;10(3):257-62.

216. Jarrett ME, Matzel KE, Stosser M, Christiansen J, Rosen H, Kamm MA. Sacral nerve stimulation for faecal incontinence following a rectosigmoid resection for colorectal cancer. Int J Colorectal Dis 2005;20(5):446-51.

217. Jarrett ME, Matzel KE, Christiansen J, Baeten CG, Rosen H, Bittorf B, Stosser M, Madoff R, Kamm MA. Sacral nerve stimulation for faecal incontinence in patients with previous partial spinal injury including disc prolapse. Br J Surg 2005;92(6):734-9.

218. Jarrett ME, Matzel KE, Stosser M, Baeten CG, Kamm MA. Sacral nerve stimulation for fecal incontinence following surgery for rectal prolapse repair: a multicenter study. Dis Colon Rectum 2005;48(6):1243-8.

219. Vitton V, Gigout J, Grimaud JC, Bouvier M, Desjeux A, Orsoni P. Sacral nerve stimulation can improve continence in patients with Crohn's disease with internal and external anal sphincter disruption. Dis Colon Rectum 2008;51(6):924-7.

220. Jarrett ME, Dudding TC, Nicholls RJ, Vaizey CJ, Cohen CR, Kamm MA. Sacral nerve stimulation for fecal incontinence related to obstetric anal sphincter damage. Dis Colon Rectum 2008;51(5):531-7.

221. Buntzen S, Rasmussen OO, Ryhammer AM, Sorensen M, Laurberg S, Christiansen J. Sacral nerve stimulation for treatment of fecal incontinence in a patient with muscular dystrophy: report of a case. Dis Colon Rectum 2004;47(8):1409-11.

222. Conaghan P, Farouk R. Sacral nerve stimulation can be successful in patients with ultrasound evidence of external anal sphincter disruption. Dis Colon Rectum 2005;48(8):1610-4.

223. Maslekar SK, Gardiner A, Duthie GS. Sacral nerve stimulation as primary treatment for faecal incontinence secondary to obstetric anal sphincter damage: medium and long-term results [abstract]. Dis Colon Rectum 2006;49(5):730. Abstract 38.

224. Ratto C. Sacral nerve stimulation in fecal incontinence due to anal sphincter lesions. Paper presented at: European Society of Coloproctology 2nd Annual Scientific Meeting; September 27, 2007; Malta.

225. Tjandra JJ, Chan MK, Yeh CH, Murray-Green C. Sacral nerve stimulation is more effective than optimal medical therapy for severe fecal incontinence: a randomized, controlled study. Dis Colon Rectum 2008;51(5):494-502.

226. Chan MK, Tjandra JJ. Sacral nerve stimulation for fecal incontinence: external anal sphincter defect vs. intact anal sphincter. Dis Colon Rectum 2008;51(7):1015-24; discussion 1024-5.

227. Hetzer FH, Bieler A, Hahnloser D, Lohlein F, Clavien PA, Demartines N. Outcome and cost analysis of sacral nerve stimulation for faecal incontinence. Br J Surg 2006;93(11):1411-7.

228. Shafik A, Ahmed I, El-Sibai O, Mostafa RM. Percutaneous peripheral neuromodulation in the treatment of fecal incontinence. Eur Surg Res 2003;35(2):103-7.

229. Queralto M, Portier G, Cabarrot PH, Bonnaud G, Chotard JP, Nadrigny M, Lazorthes F. Preliminary results of peripheral transcutaneous neuromodulation in the treatment of idiopathic fecal incontinence. Int J Colorectal Dis 2006;21(7):670-2.

230. Mentes BB, Yuksel O, Aydin A, Tezcaner T, Leventoglu A, Aytac B. Posterior tibial nerve stimulation for faecal incontinence after partial spinal injury: preliminary report. Tech Coloproctol 2007;11(2):115-9.

231. Keegan PE, Atiemo K, Cody J, McClinton S, Pickard R. Periurethral injection therapy for urinary incontinence in women. Cochrane Database Syst Rev 2007(3):CD003881.

232. Shafik A. Polytetrafluoroethylene injection for the treatment of partial fecal incontinence. Int Surg 1993;78(2):159-61.

233. Shafik A. Perianal injection of autologous fat for treatment of sphincteric incontinence. Dis Colon Rectum 1995;38(6):583-7.

234. Feretis C, Benakis P, Dailianas A, Dimopoulos C, Mavrantonis C, Stamou KM, Manouras A, Apostolidis N, Androulakis G. Implantation of microballoons in the management of fecal incontinence. Dis Colon Rectum 2001;44(11):1605-9.

235. Kumar D, Benson MJ, Bland JE. Glutaraldehyde cross-linked collagen in the treatment of faecal incontinence. Br J Surg 1998;85(7):978-9.

236. Stojkovic SG, Lim M, Burke D, Finan PJ, Sagar PM. Intra-anal collagen injection for the treatment of faecal incontinence. Br J Surg 2006;93(12):1514-8.

237. Malouf AJ, Vaizey CJ, Norton CS, Kamm MA. Internal anal sphincter augmentation for fecal incontinence using injectable silicone biomaterial. Dis Colon Rectum 2001;44(4):595-600.

238. Tjandra JJ, Lim JF, Hiscock R, Rajendra P. Injectable silicone biomaterial for fecal incontinence caused by internal anal sphincter dysfunction is effective. Dis Colon Rectum 2004;47(12):2138-46.

239. de la Portilla F, Fernandez A, Leon E, Rada R, Cisneros N, Maldonado VH, Vega J, Espinosa E. Evaluation of the use of PTQ implants for the treatment of incontinent patients due to internal anal sphincter dysfunction. Colorectal Dis 2008;10(1):89-94.

240. Davis K, Kumar D, Poloniecki J. Preliminary evaluation of an injectable anal sphincter bulking agent (Durasphere) in the management of faecal incontinence. Aliment Pharmacol Ther 2003;18(2):237-43.

241. Altomare DF, La Torre F, Rinaldi M, Binda GA, Pescatori M. Carbon-coated microbeads anal injection in outpatient treatment of minor fecal incontinence. Dis Colon Rectum 2008;51(4):432-5.

242. Maeda Y, Vaizey CJ, Kamm MA. Pilot study of two new injectable bulking agents for the treatment of faecal incontinence. Colorectal Dis 2008;10(3):268-72.

243. Vaizey CJ, Kamm MA. Injectable bulking agents for treating faecal incontinence. Br J Surg 2005;92(5):521-7.

244. Kenefick NJ, Vaizey CJ, Malouf AJ, Norton CS, Marshall M, Kamm MA. Injectable silicone biomaterial for faecal incontinence due to internal anal sphincter dysfunction. Gut 2002;51(2):225-8.

245. Chan MK, Tjandra JJ. Injectable silicone biomaterial (PTQ) to treat fecal incontinence after hemorrhoidectomy. Dis Colon Rectum 2006;49(4):433-9.

246. Maeda Y, Vaizey CJ, Kamm MA. Long-term results of perianal silicone injection for faecal incontinence. Colorectal Dis 2007;9(4):357-61.

247. Weiss E, Efron J, Nogueras J, Wexner S. Submucosal injection of carbon-coated beads is a successful and safe office-based treatment of fecal incontinence [abstract]. Dis Colon Rectum 2002;45(4):A46.

248. Stenberg AM, Larsson G, Johnson P. Urethral injection for stress urinary incontinence: long-term results with dextranomer/hyaluronic acid copolymer. Int Urogynecol J Pelvic Floor Dysfunct 2003;14(5):335-8; discussion 338.

249. Branagan G, Tromans A, Finnis D. Effect of stoma formation on bowel care and quality of life in patients with spinal cord injury. Spinal Cord 2003;41(12):680-3.

250. Randell N, Lynch AC, Anthony A, Dobbs BR, Roake JA, Frizelle FA. Does a colostomy alter quality of life in patients with spinal cord injury? A controlled study. Spinal Cord 2001;39(5):279-82.

251. Colquhoun P, Kaiser R, Jr., Efron J, Weiss EG, Nogueras JJ, Vernava AM, 3rd, Wexner SD. Is the quality of life better in patients with colostomy than patients with fecal incontience? World J Surg 2006;30(10):1925-8.

252. Norton C, Burch J, Kamm MA. Patients' views of a colostomy for fecal incontinence. Dis Colon Rectum 2005;48(5):1062-9.

253. Catena F, Wilkinson K, Phillips RK. Untreatable faecal incontinence: colostomy or colostomy and proctectomy? Colorectal Dis 2002;4(1):48-50.

254. Krogh K, Laurberg S. Malone antegrade continence enema for faecal incontinence and constipation in adults. Br J Surg 1998;85(7):974-7.

255. Teichman JM, Harris JM, Currie DM, Barber DB. Malone antegrade continence enema for adults with neurogenic bowel disease. J Urol 1998;160(4):1278-81.

256. Kiely EM, Ade-Ajayi N, Wheeler RA. Caecal flap conduit for antegrade continence enemas. Br J Surg 1994;81(8):1215.

257. Marsh PJ, Kiff ES. Ileocaecostomy: an alternative surgical procedure for antegrade colonic enema. Br J Surg 1996;83(4):507-8.

258. Gerharz EW, Vik V, Webb G, Leaver R, Shah PJ, Woodhouse CR. The value of the MACE (Malone antegrade colonic enema) procedure in adult patients. J Am Coll Surg 1997;185(6):544-7.

259. Hirst GR, Arumugam PJ, Watkins AJ, Mackey P, Morgan AR, Carr ND, Beynon J. Antegrade continence enema in the treatment of obstructed defaecation with or without faecal incontinence. Tech Coloproctol 2005;9(3):217-21.

260. Lefevre JH, Parc Y, Giraudo G, Bell S, Parc R, Tiret E. Outcome of antegrade continence enema procedures for faecal incontinence in adults. Br J Surg 2006;93(10):1265-9.

261. Poirier M, Abcarian H, Nelson R. Malone antegrade continent enema: an alternative to resection in severe defecation disorders. Dis Colon Rectum 2007;50(1):22-8.

262. Portier G, Ghouti L, Kirzin S, Chauffour M, Lazorthes F. Malone antegrade colonic irrigation: ileal neoappendicostomy is the preferred procedure in adults. Int J Colorectal Dis 2006;21(5):458-60.

263. Christensen P, Buntzen S, Krogh K, Laurberg S. Ileal neoappendicostomy for antegrade colonic irrigation. Br J Surg 2001;88(12):1637-8.

264. deVries PA, Pena A. Posterior sagittal anorectoplasty. J Pediatr Surg 1982;17(5):638-43.

265. Pena A, Devries PA. Posterior sagittal anorectoplasty: important technical considerations and new applications. J Pediatr Surg 1982;17(6):796-811.

266. Brain AJ, Kiely EM. Posterior sagittal anorectoplasty for reoperation in children with anorectal malformations. Br J Surg 1989;76(1):57-9.

267. Applebaum H, Atkinson JB. The posterior sagittal approach for reconstruction of severe rectovaginal injuries. J Pediatr Surg 1991;26(7):856-7.

268. Pena A. The surgical management of persistent cloaca: results in 54 patients treated with a posterior sagittal approach. J Pediatr Surg 1989;24(6):590-8.

269. Pena A. Total urogenital mobilization—an easier way to repair cloacas. J Pediatr Surg 1997;32(2):263-7; discussion 267-8.

270. Pena A, Levitt MA, Hong A, Midulla P. Surgical management of cloacal malformations: a review of 339 patients. J Pediatr Surg 2004;39(3):470-9; discussion 470-9.

271. Pena A, Hong A. Advances in the management of anorectal malformations. Am J Surg 2000;180(5):370-6.

272. Pena A, Guardino K, Tovilla JM, Levitt MA, Rodriguez G, Torres R. Bowel management for fecal incontinence in patients with anorectal malformations. J Pediatr Surg 1998;33(1):133-7.

273. Rintala R, Lindahl H, Marttinen E, Sariola H. Constipation is a major functional complication after internal sphincter-saving posterior sagittal anorectoplasty for high and intermediate anorectal malformations. J Pediatr Surg 1993;28(8):1054-8.

274. Malone PS, Ransley PG, Kiely EM. Preliminary report: The antegrade colonic enema. Lancet 1990;336:1217-8.

275. Griffiths DM, Malone PS. The Malone antegrade continence enema. J Pediatr Surg 1995;30(1):68-71.

276. Levitt MA, Soffer SZ, Pena A. Continent appendicostomy in the bowel management of fecally incontinent children. J Pediatr Surg 1997;32(11):1630-3.

277. Herndon CD, Rink RC, Cain MP, Lerner M, Kaefer M, Yerkes E, Casale AJ. In situ Malone antegrade continence enema in 127 patients: a 6-year experience. J Urol 2004;172(4 Pt 2):1689-91.

278. Koivusalo A, Pakarinen M, Rintala RJ. Are cecal wrap and fixation necessary for antegrade colonic enema appendicostomy? J Pediatr Surg 2006;41(2):323-6.

279. Goepel M, Sperling H, Stohrer M, Otto T, Rubben H. Management of neurogenic fecal incontinence in myelodysplastic children by a modified continent appendiceal stoma and antegrade colonic enema. Urology 1997;49(5):758-61.

280. Tam PK. Y-appendicoplasty: a technique to minimize stomal complications in antegrade continence enema. J Pediatr Surg 1999;34(11):1733-5.

281. Squire R, Kiely EM, Carr B, Ransley PG, Duffy PG. The clinical application of the Malone antegrade colonic enema. J Pediatr Surg 1993;28(8):1012-5.

282. Yerkes EB, Rink RC, Cain MP, Casale AJ. Use of a Monti channel for administration of antegrade continence enemas. J Urol 2002;168(4 Pt 2):1883-5; discussion 1885.

283. Webb HW, Barraza MA, Crump JM. Laparoscopic appendicostomy for management of fecal incontinence. J Pediatr Surg 1997;32(3):457-8.

284. Lynch AC, Beasley SW, Robertson RW, Morreau PN. Comparison of results of laparoscopic and open antegrade continence enema procedures. Pediatr Surg Int 1999;15(5-6):343-6.

285. Nanigian DK, Kurzrock EA. Intermediate-term outcome of the simplified laparoscopic antegrade continence enema procedure: less is better. J Urol 2008;179(1):299-303.

286. Curry JI, Osborne A, Malone PS. How to achieve a successful Malone antegrade continence enema. J Pediatr Surg 1998;33(1):138-41.

287. Shankar KR, Losty PD, Kenny SE, Booth JM, Turnock RR, Lamont GL, Rintala RJ, Lloyd DA. Functional results following the antegrade continence enema procedure. Br J Surg 1998;85(7):980-2.

288. Tackett LD, Minevich E, Benedict JF, Wacksman J, Sheldon CA. Appendiceal versus ileal segment for antegrade continence enema. J Urol 2002;167(2 Pt 1):683-6.

289. Dey R, Ferguson C, Kenny SE, Shankar KR, Coldicutt P, Baillie CT, Lamont GL, Lloyd DA, Losty PD, Turnock RR. After the honeymoon—medium-term outcome of antegrade continence enema procedure. J Pediatr Surg 2003;38(1):65-8; discussion 65-8.

290. Yerkes EB, Cain MP, King S, Brei T, Kaefer M, Casale AJ, Rink RC. The Malone antegrade continence enema procedure: quality of life and family perspective. J Urol 2003;169(1):320-3.

291. Wilcox DT, Kiely EM. The Malone (antegrade colonic enema) procedure: early experience. J Pediatr Surg 1998;33(2):204-6.

292. Kiely EM, Ade-Ajayi N, Wheeler R. Antegrade continence enemas in the management of intractable faecal incontinence. J R Soc Med 1995;88(2):103P-104P.

293. Lopez PJ, Ashrafian H, Clarke SA, Johnson H, Kiely EM. Early experience with the antegrade colonic enema stopper to reduce stomal stenosis. J Pediatr Surg 2007;42(3):522-4.

294. Hensle TW, Reiley EA, Chang DT. The Malone antegrade continence enema procedure in the management of patients with spina bifida. J Am Coll Surg 1998;186(6):669-74.

295. Driver CP, Barrow C, Fishwick J, Gough DC, Bianchi A, Dickson AP. The Malone antegrade colonic enema procedure: outcome and lessons of 6 years' experience. Pediatr Surg Int 1998;13(5-6):370-2.

296. Kokoska ER, Herndon CD, Carney DE, Lerner M, Grosfeld JL, Rink RC, West Kw KW. Cecal volvulus: a report of two cases occurring after the antegrade colonic enema procedure. J Pediatr Surg 2004;39(6):916-9; discussion 916-9.

297. Catto-Smith AG, Coffey CM, Nolan TM, Hutson JM. Fecal incontinence after the surgical treatment of Hirschsprung disease. J Pediatr 1995;127(6):954-7.

298. Malone PS, Wheeler RA, Williams JE. Continence in patients with spina bifida: long term results. Arch Dis Child 1994;70(2):107-10.

299. Pena A, el Behery M. Megasigmoid: a source of pseudoincontinence in children with repaired anorectal malformations. J Pediatr Surg 1993;28(2):199-203.

300. Lee SL, DuBois JJ, Montes-Garces RG, Inglis K, Biediger W. Surgical management of chronic unremitting constipation and fecal incontinence associated with megarectum: A preliminary report. J Pediatr Surg 2002;37(1):76-9.

301. Hallows MR, Lander AD, Corkery JJ. Anterior resection for megarectosigmoid in congenital anorectal malformations. J Pediatr Surg 2002;37(10):1464-6.

302. Hakelius L, Olsen L. Free autogenous muscle transplantation in children. Long-term results. Eur J Pediatr Surg 1991;1(6):353-7.

303. Skef Z, Radhakrishnan J, Reyes HM. Anorectal continence following sphincter reconstruction utilizing the gluteus maximus muscle: a case report. J Pediatr Surg 1983;18(6):779-81.

304. Chen YL, Zhang XH. Reconstruction of rectal sphincter by transposition of gluteus muscle for fetal incontinence. J Pediatr Surg 1987;22(1):62-4.

305. Meehan JJ, Hardin WD, Jr., Georgeson KE. Gluteus maximus augmentation for the treatment of fecal incontinence. J Pediatr Surg 1997;32(7):1045-7; discussion 1047-8.

306. Sonnino RE, Reinberg O, Bensoussan AL, Laberge JM, Blanchard H. Gracilis muscle transposition for anal incontinence in children: long-term follow-up. J Pediatr Surg 1991;26(10):1219-23.

307. Han SJ, Park HJ, Kim CB, Hwang EH. Long-term follow-up of gracilis muscle transposition in children. Yonsei Med J 1995;36(4):372-7.

308. Puri P, Nixon HH. Levatorplasty: A secondary operation for fecal incontinence following primary operation for anorectal agenesis. J Pediatr Surg 1976;11(1):77-82.

309. Kottmeier PK, Velcek FT, Klotz DH, Coren CV, Hansbrough F, Price AP. Results of levatorplasty for anal incontinence. J Pediatr Surg 1986;21(7):647-50.

310. Ruckauer KD. Dynamic graciloplasty in children with fecal incontinence: a preliminary report. J Pediatr Surg 2001;36(7):1036-9.