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ANAL SPHINCTER RECONSTRUCTION FOR INCONTINENCE DUE TO NON-OBSTETRIC SPHINCTER DAMAGE*
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ABSTRACT

Objective: **To assess the outcome after anal sphincteroplasty in patients with anal incontinence following non-obstetric anal sphincter damage.**

Design: ***A*** **prospective study carried out in an urban teaching hospital over five years (1994-1998).**

Patients and Methods: **Fourteen patients, median age of 30 years, all undergoing anal sphincter reconstruction. The procedure was performed under general or regional anaesthesia. Operative management was (i) excision of the scar tissue and apposition of the cut ends of the sphincter or (ii) a reefing technique.**

Results: **Sphincter damage was as a result of trauma in nine cases, haemorrhoidectomy in two cases, sepsis in two and failed recto-vaginal fistula repair in one case. In six patients with traumatic sphincter damage a colostomy was performed at original operation. The other eight presented with incontinence, four of whom required a diverting colostomy prior to repair. The median delay between sphincter injury and repair was six months (range 4-120). The posterior approach was used in eight patients, an anterior approach in five and both approaches in one. Non-absorbable suture material was used in six and absorbable material was used in eight patients. Twelve of the 14 patients had scar tissue at the site of damage requiring excision. Two post-haemorrhoidectomy patients underwent the reefing technique. Eleven patients (79%) were completely continent after initial repair. Two required further repair resulting in complete continence. One patient remained with mild incontinence.**

Conclusion: **Anal sphincter reconstruction for non-obstetric anal incontinence produced good short-term results.**

INTRODUCTION

Anal continence requires intact sensation and motor innervation as well as an anatomically intact sphincteric complex (1-3). Anal incontinence is generally the result of an acquired sphincter muscle damage and is defined as the loss of anal sphincter control and the consequent inability to defer the call to stool to a socially acceptable norm(1). Anatomic disruption of the sphincter mechanism is the common cause of anal incontinence, the major aetiological factor being obstetric trauma(1,4,5). Other causes are direct injury, sepsis and anorectal surgery(2-9). Anal incontinence occurs in 5% to 18% of patients(6,7) and occurs at any age, its incidence increases with age and has a female preponderance(2,7,10,11).

Whereas the results of overlapping anal sphincter repair following obstetric sphincter damage are well documented(1), there is paucity of information on sphincter reconstruction for non-obstetric sphincter damage using the overlapping technique. The objective of this study therefore was to assess outcome of anal sphincter reconstruction in patients with anal incontinence following non-obstetric sphincter damage.

MATERIALS AND METHODS

This is a prospective audit of patients undergoing anal sphincter repair following non-obstetric damage over a 5-year period (1994-1998). The aetiology, patients' demographic data, delay before surgery, the procedure performed and outcome were all documented. A proforma was completed and the information sequentially analysed.

There were 14 patients (nine males), with a median age of 30 (range 13-75) years. Table 1 lists the patients' profile and outcome of treatment. Sphincter damage was as a result of trauma (nine), haemorrhoidectomy (two), sepsis (two), and failed repair of recto-vaginal fistula (one). In nine patients with traumatic sphincter injury, damage was due to firearms in four patients, road traffic crashes in two, one patient was stabbed during sexual abuse, one was gored by a bull and one was assaulted with an umbrella. In six patients with traumatic sphincter damage a colostomy was performed at the original operation. The remaining eight presented with incontinence. Four required diverting colostomy prior to repair because of severe wound sepsis. Sphincter injury in these four patients was due to trauma in three and sepsis in one.

Technique: Sphincter repair was performed only after all perineal wounds had healed. Pre-operative assessment was

by digital examination to assess the integrity of the anal sphincter, both clinically and under anaesthesia. A covering colostomy for faecal diversion was only performed when there was severe contamination of the perineal wound precluding speedy recovery. All patients had mechanical cleansing of the large bowel prior to surgery and prophylactic antibiotics (metronidazole and a second-generation cephalosporin) at induction of anaesthesia. The procedure was performed under general or regional anaesthesia, depending on the patient's fitness for anaesthesia.

The surgical approach was anterior or posterior, depending on the site of damage to the sphincter muscle. A curvilinear incision about 1cm from the anal verge was used. The dissection was deepened to expose the external sphincter muscle and continued further laterally, to mobilise the external sphincter. Scar tissue was then excised, care being taken not to completely excise it but to leave sufficient scar tissue to retain the suture used for the repair. Repair was then performed using the apposition technique. The reefing technique was employed where there was no distinct defect on the sphincter. The scarred anal mucosa was then excised in a "V" fashion and the anal mucosa reconstructed with an absorbable suture. In this series non-absorbable suture material (Prolene®, Ethicon, Somerville USA) was used up to and including 1995 and absorbable suture material (Vicry®, Ethicon, Somerville USA) was used after 1995. Currently it is our policy to use absorbable material for anal sphincter repair. The subcutaneous tissues were apposed and the skin left open. Postoperatively a high fibre diet was commenced with bulk forming agents. Postoperative wound care consisted of a sitz bath after every bowel action and cleansing the wound with betadine until healing was complete.

The median delay between sphincter injury and repair was six months (range 4-120). One patient had a 10-year delay and the longest delay was 24 months in the remaining 13 patients. The posterior approach was used in eight patients and the anterior approach in five. One patient required both anterior and posterior approaches. There was identifiable scar tissue at the site of damage in 12 patients. They underwent scar excision followed by apposition repair. The two post-haemorrhoidectomy patients had no identifiable scar tissue but there was attenuation of the external sphincter and were managed by the posterior reefing technique. Non-absorbable suture material was used in six patients and absorbable material in the other eight. Four patients had suffered associated rectal injury, and one had an additional bladder neck injury. This patient underwent a urinary diversion prior to anal sphincter repair and colostomy closure. The other three patients had uncomplicated colostomy closure a month after sphincter reconstruction.

RESULTS

Median hospital stay was 9.5 days (range 3-20 days). Eleven patients became completely continent after initial repair. Two required further repair resulting in complete continence. The first patient had been gored by a bull and initial sphincter repair did not include the levator ani muscle. His subsequent repair included a levatorplasty. The reason for failure in the second patient with incontinence following recto-vaginal fistula repair was sepsis. One of the two patients with incontinence consequent upon haemorrhoidectomy still remained mildly incontinent after reconstruction. This patient has been followed up for five years because of unrelated problems. He remains satisfied with the outcome of sphincter reconstruction. Thus all patients achieved acceptable continence (100%) and 13 patients achieved complete continence (93%). All the 12 patients who underwent reconstruction by apposition achieved complete continence (100%). The 13 patients with complete continence were followed up for 1-3 months.

DISCUSSION

The absence of obstetric related sphincter injury in this series is difficult to explain but it is tempting to postulate that many patients have occult incontinence following vaginal delivery or that women with mild incontinence might regard it as a normal phenomenon following delivery and not report it. Leigh and Turnberg(12) provided evidence that patients are

Table 1

Outcome of anal sphincter reconstruction in 14 patients with non-obstetric anal sphincter damage

Patient No.	Age (year)	Cause	Colostomy	Delay (Months)	Pre-operative continence	Post-operative result
1	59	Sepsis	Yes	9	Incontinence of solids	Continent
2	15	Trauma	Yes	12	Incontinence of solids	Continent
3	13	Trauma	Yes	3	Incontinence of solids	Continent
4	63	Sepsis	No	120	Incontinence of solids	Continent
5	21	Trauma	Yes	6	Incontinence of solids	Continent
6	14	Trauma	Yes	12	Incontinence of solids	Continent
7	28	Trauma	Yes	6	Incontinence of solids	Continent
8	22	Post RVF-repair	No	24	Incontinence of solids	Continent*
9	40	Haemorrhoidectomy	No	24	Incontinence of solids	Soiling
10	75	Trauma	Yes	3	Incontinence of solids	Continent*
11	15	Trauma	Yes	5	Incontinence of solids	Continent
12	30	Trauma	Yes	6	Incontinence of solids	Continent
13	32	Haemorrhoidectomy	No	6	Incontinence of liquids	Continent
14	30	Trauma	Yes	6	Incontinence of solids	Continent

* After second repair

reluctant to report anal incontinence and indicated that this incontinence occurred in 51% of patients with diarrhoea. Only half of these patients included incontinence as an initial complaint. This is because the stigma is so considerable that many patients are unwilling to admit having incontinence, and physicians are often reluctant even to enquire about it (2,4). Anal incontinence is consequently probably under-reported.

Overlapping sphincteroplasty has been reported by many to be the operation of choice in incontinent patients with isolated anterior defects in the external anal sphincter muscle, particularly following obstetric trauma with variable success rates of 47-100% (1,7,8,11,13-16). The patients reported in the many series addressing the overlapping approach suffer from obstetric related anal incontinence, in whom the anterior sphincter is involved, resulting in severe incontinence because the puborectalis is normally deficient anteriorly in females(6). Pudendal nerve conduction can also become impaired after vaginal delivery with delayed development of anal incontinence. This has been attributed to progressive denervation of the anal sphincter muscles(17), with attenuation and sphincter muscle degeneration(17,18).

Direct sphincter repair by apposition has been criticised because it is associated with a failure rate of 20-40% (1,19-22). This failure is attributed to tearing of sutures and splaying of muscle ends(2,6,23). The splaying is a result of surgeons strictly adhering to the description of excising all scar tissue and apposing the muscle edges(4), which leaves only the muscle fibres to hold the sutures. A resurgence of direct sphincter repair has demonstrated good results provided a rim of fibrous tissue is left intact at the muscle ends to prevent the sutures from pulling through the muscle(2,6,20). We share this view and the short term success rate in patients who underwent the apposition technique in the present series was 100%, which compares favourably with the 58-86% reported in the literature(3,7,8,11,20-22).

Traumatic anorectal injuries commonly lead to posterior and lateral sphincter damage, although the anterior sphincter may also be involved(6). Despite the severity of trauma, the remaining sphincter usually actively contracts(4). Furthermore Gilliland *et al*(9) studied the correlation between the size of the external sphincter defect as determined by endo-anal ultrasound and the outcome after sphincteroplasty and demonstrated that defects of >20% of the circumference can be repaired successfully with results similar to patients with smaller defects. Indeed the patients undergoing the apposition repair in this study were young and fit with a sizeable and functional sphincter muscle and no attenuation and degeneration.

Reefing of the non-injured part of the sphincter has been carried out through the posterior or anterior approach with success rates of up to 94%(1,4,6,18,24). However despite good initial results with this technique loss of control of liquid stool and flatus has been shown to increase with increasing length of follow up(24,25). This is possibly because the procedure does not address the sphincter defect but merely tightens the sphincter muscle. Only two patients with anal incontinence following haemorrhoidectomy required reefing in the present series, resulting in complete continence in one patient and mild incontinence in the other. We concede that, had these two patients undergone endo-anal ultrasound, sphincter defects could have been identified.

Optimal conditions for successful sphincter repair include an isolated external anal sphincter defect, preservation of some scar tissue, bilateral intact pudendal nerves, normal rectal sensation, no previous attempted repair, and an asthenic young patient(1,8). Apart from the two post-haemorrhoidectomy patients, the patients in this study fulfilled most of the criteria. Although some authors have suggested that old age is associated with a poor outcome(1), others(7,11) have found age alone not to be a predictor of poor outcome. Simmang *et al*(26) performed anal sphincteroplasty in 14 women of mean age of 66 years with a 93% improvement in continence and they concluded that anal sphincter reconstruction could be performed safely without a higher incidence of complications in the elderly population. Unlike other authors who believe that results are dependent on preoperative severity, the study by Osterberg *et al*(14) demonstrated no correlation between outcome and the preoperative continence score.

The reasons for failure include wound infection, faulty technique, unrecognised second sphincter injury, surgeon expertise and co-existent problems such as irritable bowel syndrome. Two patients required repeat repair in this series, which was successfully completed, the reasons for failure being sepsis in one and inadequate repair in the other. The place of repeat repair following failure of sphincteroplasty was addressed by Pinedo *et al* (27) in 1999. In their study of 23 patients they were able to return to continence 15 (65%) of patients by re-repair. They concluded that repeat repair should be offered to patients with an intrinsically functioning external sphincter.

The use of defunctioning colostomy at the time of primary repair is controversial. Although it is used routinely in some studies(13,28) most authors do not recommend it and there is agreement that it should be reserved only for the most severely affected patients with sphincter and pelvic floor damage, the presence of a large sphincter defect, a previously failed repair or with contaminated wounds(3,6,16). Furthermore many authors(2,7,13,29) have shown that addition of a colostomy has no positive influence on the overall outcome of surgery, does not improve primary wound healing and it adds significant time and morbidity to the initial hospital admission. Our policy is to reserve covering colostomy for injuries with severe sepsis, unless the colostomy was performed at the initial laparotomy. The four patients who underwent colostomy

prior to repair had severe sepsis in the perineal wound. The four, whose wounds were clean, did not receive diversion colostomy.

There is no agreement regarding the feeding protocol following anal sphincter reconstruction. Most studies do not report on postoperative nutritional regimens. Some authors include the use of elemental diets, total parenteral nutrition, together with constipating agents with a urethral catheter in the postoperative period, and strict bed rest(3,8,30). Our policy is to commence feeding immediately after the procedure. Rosenberg and Kehlet(16) have confirmed the safety and effectiveness of this approach. So far there is no scientific rationale given in the literature for a conservative feeding regimen(16).

The criticisms of the present study are the small number of patients, the heterogeneity of patients with regards to aetiology of incontinence, the lack of physiological and endosonographic assessment as well limited follow-up. Despite this we believe that the study is significant in that it focuses on non-obstetric sphincter damage and emphasises the place of the apposition technique for anal sphincter reconstruction in these patients, producing good short-term results.

In conclusion we have shown that anal sphincter repair was effective in a large majority of patients with non-obstetric-related incontinence and was associated with acceptable continence in all. The apposition technique was effective in this cohort of patients with 100% short-term success in the group that underwent limited scar excision and the apposition technique.

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