# AGA Technical Review on the Diagnosis and Care of Patients With Anal Fissure

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Anal fissure is a disruption of the skin at the distal anal canal. Most anal fissures are located in the posterior midline, with 10% to 15% occurring anteriorly. "Off the midline" fissures must be viewed with suspicion for underlying pathology such as Crohn's disease, HIV/AIDS, tuberculosis, syphilis, or anal carcinoma. Early fissures have the appearance of a simple tear in the anoderm. With the passage of time, chronic fissures develop thickened skin margins, and fibers of the internal anal sphincter (IAS) become visible at the fissure's base. Many patients with chronic fissures develop a sentinel skin tag at the distal fissure margin and a hypertrophied anal papilla just proximal to the fissure within the anal canal.

Anal fissure is a common disorder, but its exact incidence is unknown. The condition may frequently be misdiagnosed as hemorrhoids by primary care providers. The clinical hallmark of anal fissure is pain during, and especially after, defecation. The pain may be short-lived with acute fissures, but may last hours or even become continuous in chronic cases. The pain is often severe enough for patients to dread or even attempt to avoid bowel movements altogether; some patients describe the pain as akin to passing razor blades or broken glass. Fissure patients also can experience rectal bleeding, usually consisting of small quantities of fresh red blood seen on the toilet tissue.

The standard algorithm for anal fissure therapy has traditionally consisted of a trial of fiber supplementation, sitz baths, and topical analgesics; if the pain is intolerable or if conservative care fails, surgery is performed (usually a lateral internal sphincterotomy). This approach has been modified in recent years, as a better understanding of fissure pathophysiology has provided a number of novel therapeutic options.

## Literature Review

Anal fissure literature is replete with retrospective case series; until recently, few randomized controlled studies were reported. Given these constraints, it was necessary to accept imperfectly designed or imperfectly conducted studies as part of the available fund of knowledge.

A MEDLINE search of the English language literature (1980–1999) was performed using the medical subject terms *anal fissure* cross-referenced with *etiology, treatment,* and *surgery*. From the reviewed literature, we also identified articles of particular importance published in earlier years.

In addition to study design, several key factors affect the quality of the anal fissure literature. Acute and chronic fissures must be differentiated by clear criteria, because acute fissures are easier to treat, whereas chronic fissures tend to be refractory. Follow-up must be adequate to assure relapse has not occurred. Healing must be assessed by physical examination, not merely by abatement of symptoms, because even in the presence of a persisting fissure, symptoms can abate. Assessment of the side effects of therapy, particularly diminished continence, must be systematic. A daily diary card filled out for a specified period of time is ideal for this purpose, but this relatively cumbersome technique has rarely been used in fissure studies. Patients must, at a minimum, be provided a systematic opportunity to report minor alterations in stool control to an independent observer; a retrospective review of a surgeon's office chart that does not mention deterioration of continence is far from adequate assurance that none has occurred.

## Etiology

The cause of anal fissure has been debated for many years. Trauma, usually because of passage of a large or hard stool, is believed to be a common initiating factor. Yet, some patients offer no such history; others simply experience a bout of diarrhea. More importantly from a practical view is the question of why many simple traumatic fissures proceed to heal while others become

*Abbreviations used in this paper:* BT, botulin toxin; GTN, glyceryl trinitrate; IAS, internal anal sphincter; ISDN, isosorbide dinitrate.


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chronic. The key insight into this problem came with the observation in the 1970s that resting anal pressure is elevated in fissure patients.[1,2] This observation, even if it did not fully explain the pathogenesis of fissure, at least provided an explanation for the accepted (and successful) surgical therapies of the time: anal dilatation and internal sphincterotomy, both of which decrease resting pressure. More recent studies using ambulatory manometry have confirmed the presence of sustained resting hypertonia, with abnormally few episodes of spontaneous IAS relaxation in chronic fissure patients.[3]

Why should high resting pressure be associated with fissure persistence? An ischemic cause, suggested by Gibbons and Read in 1986,[4] was supported by postmortem angiographic studies of the inferior rectal artery. Those studies demonstrated a relative paucity of vascularity in the posterior midline of the anal canal in 85% of the cadavers examined.[5] This finding alone hardly explains the cause of anal fissure; quite clearly, 85% of the general population does not have this disorder, and fissure prevalence has not been shown to increase in older patients or in those with known peripheral vascular disease. However, anodermal perfusion, at least in part, depends on arterioles that must cross the IAS.[5] High anal pressures could therefore diminish perfusion pressure to the anoderm and lead to ischemic ulceration; older patients would be protected from fissure by their diminished anal tone.

Schouten et al., using Doppler laser flowmetry to measure anodermal blood flow, found that in healthy individuals, the posterior midline had the lowest perfusion of all 4 quadrants.[6] They noted a significant inverse correlation between posterior midline anodermal blood flow and maximum resting anal pressure in a large cohort of patients that included (among others) fissure patients and normal controls. Anal fissure patients had the highest resting anal pressures and the lowest posterior commissure blood flow of any group, and anesthesia-related diminution of anal pressure was accompanied by improved posterior midline blood flow. Subsequent work demonstrated that lateral internal sphincterotomy normalized both sphincter hypertonia and anodermal blood flow in anal fissure patients.[7]

Although the association of anal fissure with sphincter hypertonia was not noted until the 1970s and its relationship to anodermal hypoperfusion until the 1990s, the mainstays of surgical therapy for fissure, anal dilatation, and internal sphincterotomy, both lead to decreased anal tone. Each of these procedures dates to the 19th century, well before their mechanism of action could be documented.[8] In contrast, the 1990s witnessed the development of several specific pharmacologic therapies for fissure, each of which is designed to correct sphincter hypertonia. The impetus for medical, rather than surgical, therapy emanates predominantly from the recognition that therapeutic weakening of the anal sphincter by stretching or cutting can lead to concomitant loss of function with impaired levels of continence. Moreover, improved understanding of anal sphincter neurophysiology has provided a rational approach to drug development for anal fissure therapy.

## Diagnosis

Anal fissure occurs most frequently in young adults and affects both sexes equally.[9] The great majority of fissures occur in the posterior midline, although anterior midline fissures are seen in 25% of affected women and 8% of affected men.[9] About 3% of patients have both anterior and posterior fissures.

The diagnosis of anal fissure is suspected from the patient's history and is confirmed by physical examination. Most fissures are visible by separating the buttocks with opposing traction of the thumbs. The presence of a sentinel skin tag should alert the examiner to the likely presence of a fissure, even when one is not readily visible. Once a typical fissure is seen, further endoscopic evaluation can be deferred until the patient's symptoms have resolved. Digital examination and rigid or flexible endoscopic evaluation are not appropriate if the patient has a markedly tender anus, because such steps are far more likely to inflict pain than establish a diagnosis. When the diagnosis in this setting is in doubt, examination under anesthesia is warranted, particularly to exclude the presence of an occult perianal abscess. Off-the-midline fissures, especially multiple, painless fissures and fissures that fail to heal, also require further evaluation, usually in the form of examination under anesthesia with biopsy and appropriate cultures.

## Treatment

### Medical Therapy

**Standard conservative care.** About half of all patients with fissure can expect healing with conservative care. In a retrospective review, Shub et al. reported that 44% of fissure patients healed with sitz baths, a psyllium fiber supplement, and emollient suppositories. In 27% of these "healed" patients, the fissure recurred over a 5-year follow-up period.[10] A retrospective review by Hananel and Gordon[9] reported remarkably similar figures of healing in 44.7% and recurrence in 18.6% of their patients; medical therapy comprised of bulk-forming agents and

warm sitz baths. Neither of these 2 reports addressed patient education and compliance issues.

Jensen[11] conducted a randomized trial of sitz baths with bran supplementation (10 g unprocessed bran twice daily) vs. 2% lignocaine ointment vs. 2% hydrocortisone cream in patients with first-time acute posterior midline fissures. After 3 weeks, fissure healing was observed in 87.5% of the sitz bath and bran group, 82.4% of the hydrocortisone group, and 60% of the lignocaine group. Although healing rates were not significantly different between the sitz bath and bran group and the hydrocortisone group at 3 weeks, healing was fastest in the sitz bath and bran group. In a separate randomized trial, the daily ingestion of 15 g of bran significantly reduced the relapse rate of recently healed acute fissures when compared with 7.5 g bran and placebo.[12]

Anal dilators, usually coated with a topical anesthetic preparation, have been advocated by some for fissure therapy. Two randomized trials comparing conservative care with or without an anal dilator showed no benefit with self-dilatation.[13,14]

**Pharmacological sphincter relaxants.** Interest in pharmacologic manipulation of the IAS was spurred by the recognition that sphincter hypertonia is associated with fissure persistence, and that surgical approaches to decreased tone may lead to permanent dysfunction. The IAS consists of smooth muscle whose tone is caused partially by intrinsic myogenic properties and partly to extrinsic neural influences.[15–17] Nitric oxide is the principal nonadrenergic, noncholinergic neurotransmitter in the IAS,[18,19] and its release results in IAS relaxation. IAS contraction is mediated by increased cytosolic calcium levels.[15,20] Calcium-channel blockers conversely reduce IAS tone. $\alpha_1$-Adrenergic stimulation leads to IAS contraction, whereas $\beta$-adrenergic and muscarinic cholinergic agonists lead to IAS relaxation.[15]

**Topical nitrates.** Specific pharmacologic therapy for anal fissure became feasible with the recognition of nitric oxide's central role in IAS relaxation. Exogenous nitrates release nitric oxide in vivo and have been used clinically as nitric oxide donors. Loder et al.[21] demonstrated that topical application of 0.2% glyceryl trinitrate (GTN) led to decreased resting anal pressure. This finding set the stage for a number of studies using various topical organic nitrate preparations to alleviate anal hypertonia and thereby improve anodermal blood flow.

Healing was reported in 77% of patients with anal fissure and 54% with anal ulcers after 8 weeks of therapy 4 times a day in an uncontrolled trial.[22] A number of GTN trials were reported,[23–25] culminating with a randomized, double-blind, placebo-controlled trial involving 80 patients with chronic anal fissure (Table 1).[26] Placebo patients were treated with white soft paraffin ointment and did not receive special counseling regarding sitz baths or fiber intake. Reduced maximal resting anal pressures and improved anodermal blood flow were found in GTN patients but not controls at the time of initial therapy. After 8 weeks, healing occurred in 68% of GTN patients and in only 8% of controls ($P < 0.0001$). In another placebo-controlled randomized trial, 46% of patients with chronic anal fissures treated with GTN healed vs. 16% of controls.[27]

Bacher et al.[28] conducted a randomized trial of 0.2% GTN vs. 2% lignocaine gel, each applied 3 times daily, in a mixed group of acute and chronic fissure patients. After 1 month, healing rates were higher with GTN in both the acute (91.6%, GTN vs. 50%, lignocaine) and chronic (62.5%, GTN vs. 20%, lignocaine) fissure groups. Carapeti et al.[29] conducted a double-blind randomized trial comparing 0.2% GTN vs. escalating GTN doses (0.2% increasing weekly by 0.1% to a maximum dose of 0.6%) vs. placebo. Healing rates after 8 weeks of treatment were significantly better in both GTN groups (65% with 0.2%; 70% with escalating doses) than in controls (32%).

Several organic nitrate preparations besides GTN have been assessed for fissure therapy. One percent isosorbide dinitrate (ISDN) ointment was tested in 34 chronic anal fissure patients; the ointment was applied every 3 hours while awake.[30] In this uncontrolled study, maximum anal pressure decreased and anodermal blood flow increased significantly at 3 and 6 weeks; 30 patients (88%) were healed at 12 weeks. ISDN spray (2.5 mg 3 times daily) was tested in 41 chronic fissure patients whose conservative medical therapy had failed.[31] A total of 34 patients (83%) experienced healing by 1 month; 6 patients relapsed within 11 months, but all 6 responded to a second course of therapy.

Despite the encouraging results reported with topical nitrates, a number of caveats remain. Standard conservative care was not used in some nitroglycerin trials,[26–29] so the healing rate was low in the control arms. For example, Lund and Scholefield[26] reported healing in only 8% of their control group. Long-term follow-up has been lacking in many studies; thus, the risk of late recurrence after topical nitrate therapy remains uncertain. Lund and Scholefield[32] conducted a telephone follow-up study of 44 patients who had been randomized to GTN therapy in previous trials. Of 41 contacted patients, 11 (27%) had at least 1 symptomatic relapse (median follow-up, 28 months); of the 11 patients, 6 had multiple recurrences

**Table 1.** Randomized Trials of Fissure Pharmacotherapy

| Author | Year | n | Treatment | Success (%) | Follow-up | Incontinence (%) | Recurrence (%) | Headache (%) |
|---|---|---|---|---|---|---|---|---|
| Bacher et al.[28] | 1997 | 35 | 0.2% GTN | 80 | 28 d | NS | NS | 20 |
| | | | 2% lidocaine | 40 | 28 d | NS | NS | 0 |
| Lund and Scholefield[25] | 1997 | 80 | 0.2% GTN | 68 | 4 mo | NS | 8 | 58 |
| | | | Placebo | 8 | 4 mo | NS | 0 | 18 |
| Maria et al.[53] | 1998 | 30 | Botulin toxin 20 U | 73 | 16 mo | 4[b] | 0 | NS |
| | | | Placebo | 13 | 16 mo | 0 | 0 | NS |
| Antropoli et al.[40] | 1999 | 283 | 0.2% nifedipine gel | 95 | 21 d | NS | NS | 0 |
| | | | 1% lidocaine/ 1% HC | 50 | 21 d | NS | NS | NS |
| Brisinda et al.[54] | 1999 | 50 | Botulin toxin 20 U | 96 | 15 mo | 0 | 0 | 0 |
| | | | 0.2% GTN | 60 | 15 mo | 0 | 0 | 20 |
| Carapeti et al.[29] | 1999 | 70 | 0.2% GTN | 65 | 9 mo | 13[c] | 33 | 65 |
| | | | 0.2%–0.6% GTN[a] | 70 | 9 mo | 13[c] | 25 | 78 |
| | | | Placebo | 32 | 9 mo | 0 | 43 | 27 |
| Kennedy et al.[27] | 1999 | 43 | 0.2% GTN | 46 | 29 mo | NS | NS | 29 |
| | | | Placebo | 16 | 29 mo | NS | NS | 5 |
| Altomare et al.[35] | 2000 | 119 | 0.2% GTN | 49 | 12 mo | NS | 19 | 34 |
| | | | Placebo | 52 | NS | NS | NS | 8 |
| Richard et al.[36] | 2000 | 82 | 0.25% GTN | 27 | 6 mo | 0 | 11 | 84 |
| | | | LIS | 92 | 6 mo | 0 | 0 | 5 |
| Zuberi et al.[94] | 2000 | 37 | 0.2% GTN | 67 | NS | 0 | NS | 72 |
| | | | 10 mg nitroglycerin patch | 63 | NS | 0 | NS | 63 |
| Jonas et al.[43] | 2001 | 50 | 2% diltiazem | 65 | 8 wk | NS | 4 | 0 |
| | | | Diltiazem 60 mg po bid | 38 | 8 wk | NS | 4 | 8 |
| Werre et al.[95] | 2001 | 37 | 1% isorbide dinitrate | 85 | 39 wk | NS | 12 | 45 |
| | | | Placebo | 35 | 39 wk | NS | 33 | 18 |

GTN, glyceryl trinitrate; NS, not stated; HC, hydrocortisone; po, by mouth; bid, twice daily.
[a]Increased weekly by 0.1%.
[b]Transient incontinence to flatus only.
[c]Flatus only, combined data for all GTN patients.

of symptoms, and 3 subsequently required sphincterotomy. Dorfman et al.[33] reported a similar 27% symptomatic relapse rate (median follow-up, 6 months). Carapeti et al.[29] noted relapse rates of 33% with 0.2% GTN, 25% with escalating-dose GTN, and 43% with placebo (mean follow-up, 9 months).

The reported rates and severity of side effects after topical nitrate therapy have varied considerably. Lund and Scholefield[26] reported treatment-associated headaches in 58% of GTN patients and 18% of controls; 1 GTN patient (3%) quit therapy because of this side effect. Other investigators have reported considerably higher morbidity. Dorfman et al.[33] noted GTN-associated side effects in 78% of patients, including headaches in 63% and light-headedness in 52%. Hyman and Cataldo[34] reported headaches or light-headedness in 88% of GTN patients. Kennedy et al.[27] noted headaches in 29% of GTN patients and 21% of controls. The severity of headaches apparently varied between these groups; 17% of GTN patients, but no control patients, quit therapy as a result. Altomare[35] reported headaches in 33.8% and orthostatic hypotension in 5.9% of GTN patients. Carapeti et al.[29] reported headaches in 72% of GTN patients vs. 27% of blinded placebo controls.

More recent studies have shown lower healing rates with GTN than were initially reported. In a retrospective review, Dorfman et al. found that only 67% of patients completed therapy and 55% had resolution of symptoms.[33] Hyman and Cataldo[34] found healing in only 1 (6%) of 17 chronic fissure patients; an additional 6 patients were symptom-free but not healed. A placebo-controlled double-blind trial found that the addition of 0.2% GTN to standard conservative care did not improve healing rates (49.2% with GTN vs. 51.7% with placebo).[35]

Results of a multicenter randomized controlled trial of GTN (0.25% 3 times daily) vs. LIS in 82 chronic anal fissure patients reported healing rates at 6 weeks and 6 months of 29.5% and 27.2% for GTN vs. 89.5% and 92.1% for LIS.[36] Eighty-four percent of GTN patients developed side effects, the vast majority of which were headache, vs. 30% in the LIS group. Twenty-one percent of GTN patients were unable to continue their treatment caused by headache or syncope, and 45% of GTN patients required LIS to heal their fissure. No alteration of continence was reported in the LIS group.

**Calcium-channel blockers.** Calcium-channel blockers have been the focus of considerable recent work on

pharmacologic fissure therapy. Chrysos et al.[37] studied the effect of sublingual nifedipine on anal sphincter pressure in acute anal fissure patients; resting anal pressure decreased, as did anal slow and ultra-slow wave activity. Cook et al.[38] administered oral nifedipine (20 mg twice daily) to healthy volunteers and to chronic anal fissure patients. Maximum resting anal pressure decreased by a third in both groups. Of 15 fissure patients, 9 were healed at 8 weeks. Carapeti et al.[39] found that oral diltiazem (60 mg twice daily) reduced anal pressure 17%.

Several trials have investigated the use of topical calcium-channel blockers in anal fissure therapy. Antropoli et al.[40] conducted a randomized double-blind trial of topical 0.2% nifedipine gel vs. topical 1% lidocaine and 1% hydrocortisone in 283 acute anal fissure patients. Maximum resting anal pressure decreased 30% in the nifedipine group and 95% of these patients healed, vs. 50% of controls. Carapeti et al.[39] performed a dose-ranging trial using diltiazem gel and found that 2% gel produced a maximal effect of 28% reduction in resting anal pressure. The effect lasted 3 to 5 hours after a single application. In a subsequent study, 10 of 15 chronic anal fissure patients treated with 2% diltiazem gel 3 times daily healed within 8 weeks; no patients reported headache or other side effects.[41] Surprisingly, anodermal blood flow did not change with treatment. Seventy-one consecutive chronic anal fissure patients were treated with 2% diltiazem gel for a median of 9 weeks.[42] Fifty-one patients (75%) healed, as did 8 of 17 patients who failed initial therapy after an additional 8-week course. Twenty-seven of 41 patients (66%) remained symptom-free at a median follow-up of 32 weeks; 6 of 7 patients with fissure recurrence responded to repeat therapy. Side effects included headache in 1 patient and dermatitis in 4.

Jonas et al.[43] performed a randomized controlled trial of oral (60 mg twice daily) and topical (2% gel twice daily) diltiazem in 50 chronic anal fissure patients. Healing rates were 38% in the oral group and 65% in the topical group ($P = 0.09$). Side effects occurred in 8 of the patients (33%) treated orally and in none of the patients treated topically.

Based on the relatively limited data available to date, topical anal fissure therapy with calcium-channel blockers appears to be roughly as effective as treatment with topical nitrates. Moreover, the side effect profile of topical calcium-channel blockers appears superior, specifically with respect to fewer reported headaches. However, long-term follow-up studies are lacking, and a randomized trial comparing topical nitrates with topical calcium-channel blockers has not yet been reported.

**Muscarinic agonists.** A dose-ranging study of topical bethanechol in 10 healthy volunteers found the maximal effect of 24% reduction in resting anal pressure occurred using 0.1% gel.[39] In a subsequent study, 9 of 15 chronic anal fissure patients (60%) healed after treatment with 0.1% bethanechol gel applied 3 times daily. Mean resting pressure decreased 16% with treatment, but no change in anodermal blood flow was detected.[41] No side effects were reported in either study.

**Adrenergic agonists and antagonists.** Pitt et al.[44] investigated the effect of $\alpha_1$-adrenoreceptor blockade using a single oral 20 mg dose of indoramin in 6 healthy volunteers and 7 chronic anal fissure patients. Resting anal pressure decreased to a similar extent (36% to 40%) in each group. However, a randomized controlled trial of indoramin vs. placebo was abandoned prematurely due to a low healing rate in the indoramin group.[45] $\beta_2$-Receptor stimulation using salbutamol resulted in reduced resting anal pressure in both healthy volunteers and anal fissure patients.[46]

**Botulin toxin.** Locally injected botulin toxin (BT) has been used as an alternative approach to sphincter relaxation in the treatment of anal fissure. This commercially available agent, which prevents neural transmission by preventing acetylcholine release from presynaptic nerve terminals, has been used to treat both skeletal,[47] as well as smooth muscle,[47–49] disorders, including achalasia.[48]

Jost and Schimrigk[50] first reported the use of BT for anal fissure in 1993. Jost[51] subsequently reported on a series of 100 patients treated with BT injection. In all, 78 patients became pain-free within 3 days, and healing rates at 3 and 6 months were 82% and 79%. During the first 6 months, 8 patients relapsed and 9 had transient incontinence (7 to flatus, 2 to stool). Retreatment led to healing in 63% of the patients whose initial BT injection failed and in 70% of those whose fissure recurred after 3 months.[52]

Maria et al.[53] conducted a double-blind, placebo-controlled trial of BT (vs. saline) in 30 chronic anal fissure patients. Resting anal pressure decreased by 27% in the treated patients, but was unchanged in the control group. Healing occurred in 53% and 73% of treated patients at 1 and 2 months, vs. 13% and 13% in the control group. Of 10 control patients who crossed over to BT injection after initial saline treatment failure, 7 healed. The 4 unhealed patients in the BT arm underwent retreatment and all healed within 2 months. Only 1 patient had transient incontinence to flatus after BT injection.

BT injection was compared with topical GTN (0.2% twice daily) in a randomized trial of 50 chronic anal fissure patients.[54] Resting anal pressure decreased in both groups, but did so to a greater extent in the BT group (29% with BT vs. 14% with GTN at 2 months, $P = 0.04$). Healing rates were 96% in the BT group and 60% in the GTN group ($P = 0.005$). Patients whose initial therapy failed (1 BT, 9 GTN) crossed over to the other arm; all healed with additional treatment. Although 20% of GTN patients had treatment-related headaches, no adverse effects were seen in the BT group. No incontinence was reported, and no relapses were seen at a mean follow-up of 15 months.

BT dosage and administration have varied between studies. Jost[51] injected 2.5 or 5 units of BT into the external anal sphincter on either side of the fissure (total dose 5 to 10 units). He suggested a retreatment dose of 5 units for recurrent fissures and 10 units if therapy failed.[52] Patients in the Italian studies received injections totaling 20 BT units into the IAS on either side of the fissure.[53,54] Maria et al.[55] compared chronic anal fissure patients initially treated with either 15 or 20 BT units injected into the IAS. After 2 months, healing occurred in 43% of the low-dose group and 68% of the high-dose group. Patients whose initial therapy failed and who accepted further BT treatment (at doses of 20 and 25 units) had healing rates of 60% and 100%. Minguez et al.[56] compared doses of 10, 15, and 21 BT units injected in equal aliquots "through the intersphincteric groove in the direction of the internal sphincter" on each side of the anal canal (10-unit group), with a third aliquot injected into the IAS immediately below the fissure in the 15-unit and 21-unit groups. Resting anal pressure decreased significantly only in the 15-unit and 21-unit groups, and the need for surgery was lowest in the 21-unit group (5% vs. 17% in the 15-unit and 19% in the 10-unit groups). Transient mild incontinence to gas and soiling were observed in 10% of patients, none of whom were in the 21-unit group.

## Surgery

**Anal dilatation.** Manual anal dilatation has been used to treat various anorectal disorders since the 19th century. The technique was reintroduced for anal fissure therapy in 1964,[57] with success rates of 87% to 100%.[57–64] Fissure recurrence is more variable, ranging from 0% to 56%.[57–64]

Anal dilatation has been widely criticized for causing poorly controlled or diffuse sphincter damage. Nielsen et al.[65] performed endoanal ultrasound in 20 patients who had undergone dilatation for fissure and found sphincter defects in 13 (65%). In that series, 12.5% of patients had "minor" incontinence; incontinence in other series ranged from 0% to 51%.[57–64]

Despite the excellent results reported in some series, anal dilatation literature must be read with caution. The exact technique varies between, and sometimes within, series. Important variables, such as the extent and duration of sphincter stretch or the type of anesthesia, are often neither specified nor standardized. In one exception to this rule, Sohn et al.[66] carefully standardized anal dilatation, using either an anal retractor or pneumatic balloon, under local anesthesia with sedation; 94% of patients healed. Many reports of anal dilatation are relatively old and details regarding continence assessment are frequently lacking, suggesting that reported incontinence rates may actually represent minimal figures.

**Sphincterotomy.** Eisenhammer[67,68] popularized internal anal sphincterotomy for the treatment of anal fissure during the 1950s. Although he initially advocated posterior midline sphincterotomy through the bed of the fissure, he subsequently recommended use of the lateral internal sphincterotomy (LIS), which he believed was associated with less functional impairment and better wound healing. In a retrospective review, Abcarian[69] and Hawley,[70] in a randomized trial, each concluded that LIS was the preferred operation. However, meta-analysis of the fissure surgery literature failed to demonstrate a difference between midline and lateral internal sphincterotomy.[71] One potential complication of midline sphincterotomy, particularly when performed in association with fissurectomy, is the development of a "keyhole deformity," a scarred groove that permits loss of flatus or seepage of stool. Because the anal orifice is shaped as an anteroposterior slit, LIS does not lead to the same type of anal deformity and functional impairment.

LIS results are depicted in Table 2. The vast majority of patients heal after LIS, and recurrence rates are low. In contrast to the uniformity of these results, the wide variability in postoperative incontinence rates is striking. These differences probably reflect the care and nature of follow-up more than differences in surgical skill or technique. Subtle alterations in continence may not be volunteered or recorded in a routine postoperative office visit. Note the studies listed in Table 1 that used standardized continence questionnaires reported dramatically higher levels of impairment[72–75] than those that did not.

The extent of sphincterotomy is an important determinant of outcome. Garcia-Granero et al.[76] used anal

**Table 2.** Results of Lateral Internal Sphincterotomy

| Author | Year | n | Success (%) | Recurrence (%) | Incontinence (%)[a] | Follow-up (type) | Follow-up (%) | Follow-up (months) |
|---|---|---|---|---|---|---|---|---|
| Abcarian[69] | 1980 | 150 | 100 | 1.3 | 0 | C | NS | NS |
| Keighley et al.[97] | 1981 | 71 | 100 | 25 | 2 | I, E | 89 | 12 |
| Ravikumar et al.[98] | 1982 | 60 | 97 | 0 | 5 | C | 100 | 24 minimum |
| Hsu et al.[99] | 1984 | 89 | 100 | 5.6 | 0 | C | NS | NS |
| Jensen et al.[61] | 1984 | 30 | 100 | 3 | 0 | Q, E | 100 | 18 median |
| Walker et al.[100] | 1985 | 306 | 100 | 0 | 15 | I | 33 | 52 mean |
| Gingold[101] | 1987 | 86 | 100 | 3.5 | 0 | C | NS | 24 median |
| Weaver et al.[62] | 1987 | 39 | 93 | 5.1 | 2.5 | I, E | 86 | 17 mean |
| Lewis et al.[81] | 1988 | 350 | 94 | 6 | 6 | I | 100 | 37 median |
| Zinkin[102] | 1988 | 151 | 94.7 | NS | NS | none | 0 | 0 |
| Khubchandani et al.[73] | 1989 | 717 | 97.7 | NS | 35.1 | Q | 52.9 | NS |
| Kortbeek et al.[82] | 1992 | 112 | 95.5 | NS | NS | I | NS | 1.5 |
| Pernikoff et al.[74] | 1994 | 500 | 99 | 2 | 16 | Q | 78 | 67 |
| Romano et al.[103] | 1994 | 44 | 100 | 0 | 9 | E | NS | 8 |
| Leong et al.[104] | 1995 | 20 | 100 | NS | 0 | I, E | NS | 6.5 median |
| Prohm et al.[105] | 1995 | 177 | 96 | 3.3 | 1.6 | E | NS | 1–1.5 |
| Usatoff et al.[75] | 1995 | 98 | 90 | 20 | 18 | Q | 80 | 41 mean |
| Garcia-Aguilar et al.[72] | 1996 | 864 | 96 | 11 | 37.8 | Q | 63.5 | 36 mean |
| Hananel et al.[106] | 1997 | 312 | 98.6 | 1.4[b] | – | C | 93.3 | NS |
| Littlejohn et al.[79] | 1997 | 352 | 99.7 | 1.4 | 1.4 | C | 81.5 | 9 mean |
| Nyam et al.[107] | 1999 | 585 | 96 | 8 | 15 | Q | 83 | 72 mean |

C, chart review; E, examination; I, interview; Q, questionnaire; NS, not stated.
[a]Includes soiling, incontinence to flatus, and incontinence to stool.
[b]Recurrence and persistence combined.
Modified from Fleshman et al.[96]

ultrasonography to demonstrate a high rate of incomplete sphincterotomy in fissure patients with symptomatic recurrences. Sultan et al.,[77] in a prospective ultrasound study, demonstrated a tendency for women, because of their anatomically shorter sphincter, to undergo more extensive sphincterotomy than men. This factor, along with underlying obstetrical sphincter injury, is associated with impaired continence. Postpartum fissures are associated with diminished anal pressures, a fact that militates against sphincterotomy in this setting.[78]

The optimal proximal extent of sphincterotomy has received little scientific attention. Most authors describe sphincterotomies extending to the dentate line, but this choice seems to have more to do with the convenience of this anatomic landmark than with the physiology of the anal canal. With the risk of minor incontinence now more apparent, some authorities recommend that the proximal extent of sphincterotomy match that of the fissure itself, an approach that cuts less muscle.[79]

LIS can be performed using either an open (exposing and dividing the IAS) or closed (dividing the IAS via a small stab wound) technique.[80] Both techniques are similarly effective with respect to fissure healing.[72,81,82] One paper suggests that closed sphincterotomy may be preferable because of its lower rate of continence impairment,[72] but this difference was not seen in a meta-analysis of surgery trials.[71]

Several randomized trials compared LIS with anal dilatation.[61,62,70,83] Weaver et al.[62] found no difference in either success or complication rates. Hawley[70] reported no recurrences after LIS vs. 28% after anal stretch; impaired continence was not seen in either group. Jensen et al.[61] reported recurrences in 29% of dilated patients vs. 3% of those treated with LIS. The dilated group also had a significantly higher rate of impaired control (39% vs. only 3% with LIS).[61] Differences between these studies in the dilated group results may be attributed to variability in operative technique.

## Special Situations

**Crohn's disease.** Perianal disease is a source of significant morbidity for patients with Crohn's disease, although reports of its incidence vary widely. Platell et al.[84] noted symptomatic anal pathology in 42.4% of Crohn's disease patients; 27.6% of these patients had anal fissures. Sangwan et al.[85] reported that 3.8% of Crohn's disease patients required surgery for symptomatic perianal disease, and 31.8% of these patients had anal fissures. Such fissures are sometimes asymptomatic.

Frequently, they are multiple or off the midline,[86] and they often coexist with other pathology.[85] Unlike typical fissures, those associated with Crohn's disease can also be locally aggressive, progressing to form deep ulcers with granulating bases and overhanging skin edges.

Surgeons have traditionally approached the anal canal with caution in Crohn's disease patients, fearing that an operation might precipitate complications leading to proctectomy. Furthermore, therapies requiring sphincter muscle divisions are correctly perceived as putting the patient at risk for incontinence, as these patients frequently have an underlying diarrheal state and are at significant risk for requiring additional anal surgery in the future. Despite these concerns, the degree of continence impairment after sphincterotomy for fissure has not been systematically studied in patients with Crohn's disease.

Most authorities advocate standard conservative treatment, combined with medical or surgical therapy directed at controlling diarrhea, as first line treatment for Crohn's fissures. Results of treatment with topical sphincter relaxants or BT have not been reported to date. If conservative care fails and the fissure remains symptomatic, the patient should be examined under anesthesia (to exclude occult-associated pathology) and a sphincterotomy or gentle dilatation should be performed. (Cavitating ulcers, in contrast, are debrided without a sphincterotomy; they are not caused by anal hypertonia and have frequently already eroded the underlying sphincter.)

Only small retrospective series of patients with Crohn's fissures who have undergone surgery have been reported. Wolkomir and Luchtefeld[87] reported uncomplicated wound healing in 22 of 25 such patients. Fleshner et al.[86] reviewed a series of 56 patients with Crohn's disease and anal fissures; 49% healed after medical therapy, 88% after anorectal surgery, and 29% after abdominal surgery. However, 26% of the medical therapy group went on to develop an abscess or fistula at the site of the fissure, suggesting local disease progression. Fleshner et al.[86] advocated closed lateral internal sphincterotomy for "judiciously" selected patients whose medical therapy failed. Results with respect to continence were not reported.

Limited numbers of patients who have undergone anal dilatation for Crohn's fissures have been reported. Allan and Keighley[88] described 7 such patients; 4 improved and 1 was rendered incontinent.

**HIV/AIDS.** HIV-associated anal fissures must be differentiated from HIV-associated anal ulcers. Fissures maintain their typical (non-HIV) appearance. However, HIV ulcers are broad-based and deep, they can occur anywhere along the length of the anal canal,[89,90] and sphincter tone tends to be low rather than high.[89] Early pessimistic reports of poor wound healing and high rates of incontinence after sphincterotomy for HIV-associated fissures may have been skewed by inclusion of HIV ulcers in the fissure group.[90] Furthermore, although poor wound healing has been frequently reported following anorectal surgery in HIV-positive patients,[91,92] there is little literature to date regarding surgery in the era of highly effective antiretroviral therapy. Little specific literature is available on HIV-associated fissures, and detailed reports of function after surgery are nonexistent. No data has been reported regarding use of topical sphincter relaxants or BT in HIV-positive patients.

Barrett et al.[93] reported their experience with perianal disease in 260 HIV-positive patients. Anal fissures were seen in 82 patients (32%). Specific results following 18 sphincterotomies were not reported. Viamonte et al.[89] reviewed the treatment of 33 HIV-positive fissure patients. Ten patients improved with conservative care, 10 were lost to follow-up, and 13 underwent LIS. Of the 13 LIS patients, 12 improved, but actual healing rates were not provided. No cases of postoperative infection or incontinence were reported.

## Conclusions

Anal fissure is a common, highly symptomatic disorder. Because its symptoms are so typical, its presence can often be inferred from the patient's history alone. Diagnosis is established by simple physical examination and does not require anal instrumentation.

Anal fissure is associated with elevated resting anal pressure, and therapy is directed at reducing anal tone. Standard conservative care leads to fissure healing in about half of all cases. Novel nonoperative options include use of topical sphincter relaxants and locally injected BT; early reports on both these therapies are promising, although the GTN literature has varied significantly in the reported rates of healing, relapse, and side effects. Topical agents such as calcium-channel blockers may be as effective as GTN but cause fewer side effects. Presently, neither appropriately diluted GTN nor topical calcium-channel blocker preparations are commercially available in the United States. One well-designed study suggests that BT therapy is superior to GTN, but the total patient numbers were small and other centers have not yet confirmed its results.

Surgery is highly successful in the management of anal fissure. In the United States, virtually all authorities advocate LIS as the operation of choice. This operation has been

associated with minor continence alterations in a minority of patients in series that have carefully scrutinized their functional results. Anal dilatation still has proponents, but it is poorly standardized, with a risk of sphincter damage and incontinence after excessive stretching.

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