

Successful Treatment of Chronic Anal Fissure Utilizing Sacral Nerve Stimulation

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ABSTRACT

Objective: This is a report of a novel treatment approach for chronic anal fissure using minimally invasive sacral nerve stimulation (SNS).

Measurements: The patient underwent uneventful placement of 1 temporary 8-electrode lead (Medtronic Inc, Minneapolis, Minnesota) for sacral nerve root stimulation.

Results: The patient experienced instantaneous relief of perineal pain after start of SNS and steady healing of the fissure by the end of the second week.

Conclusion: SNS offers an alternative treatment option of chronic anal fissure for patients who choose not to use more invasive surgical interventions.

INTRODUCTION

Anal fissure is a common and painful disorder that causes significant morbidity. Fissure in ano or anal fissure is a linear tear in the squamous epithelium of the anus below the dentate line. This condition affects all age groups, but it is particularly seen in young and otherwise healthy adults. The classic symptoms are anal pain during or after defecation accompanied by the passage of bright red blood per anus.

The pathogenesis of chronic anal fissure is poorly understood, but it is believed to result from the combination of internal anal sphincter hypertonicity, relative ischemia, and anal canal trauma. The most consistent finding in typical fissures is spasm of the internal anal sphincter, which is so severe that the pain caused by fissure is thought to be due to ischemia.¹ Relief of

the spasm has been associated with relief of pain and healing of the fissure. Only a small proportion (<10%) of chronic anal fissures heal without interventions to reduce anal pressure.²

Historically, the most common approach for relieving the spasm is surgical intervention. Operative techniques commonly used to treat anal fissure include anal stretch, open lateral sphincterectomy, closed lateral sphincterectomy, posterior midline sphincterotomy, and, to a lesser extent, dermal flap coverage of the fissure. The lateral internal sphincterectomy is widely accepted as a highly reliable, effective surgical treatment of chronic anal fissure, with cure rates exceeding 90%.³⁻⁶ Morbidity from surgical procedures has been reported, including fecal incontinence. In recent years, botulinum toxin injection has emerged as an alternative to surgery in the management of chronic anal fissure, but this therapy is far less effective than surgery.⁷ Botulinum toxin injection is effective in medium-term with cure rate of 85% at 11 months but with high rate of late recurrence approaching 50% at 22 months after treatment.⁸ Surgical interventions and botulinum toxin injections are associated with risk of impaired bowel continence in 2.17% - 30% of patients.^{3,5,6,9}

The significant, traumatizing complication of incontinence has motivated a search for pharmacologic ways to create a temporary sphincterotomy, which would decrease sphincter pressure and allow the fissure to heal. There are hopeful reports about use of local application of nitric oxide donors and calcium channel blockers.^{10,11} Pharmacologic anal sphincter relaxants promote fissure healing, but their positive effect is transient and risk of fissure recurrence persists.¹² Topical glyceryl trinitrate successfully heals 70% of fissures by the end of the 8th week with recurrence of symptoms in 27% of patients within 2 years.¹³ Temporarily lower anal pressures achieved with these treatments appear to return to pre-treatment levels within 3 months of nitrates being discontinued after the fissure has healed.¹⁴ The principal

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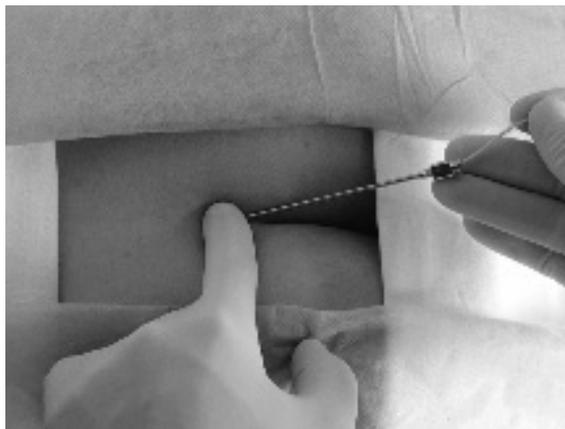


Figure 1. Percutaneous placement of one 8-electrode standard Octad Lead (Medtronic Inc., Minneapolis, Minn) through sacral hiatus under local anesthesia with C-arm guidance.

side effect of topical nitric oxide donors is headache, which causes up to 20% of patients to discontinue therapy.¹⁵ Topical application of calcium channel blockers, like nifedipine, lead to healing of the fissures in 60% of patients at 8 weeks¹⁶ but late recurrence rate was high. Calcium channel inhibitors also cause headache and may be associated with flushing and symptomatic hypotension.¹⁷ There were several reports published describing use of sacral nerve stimulation (SNS) for management of pelvic and anal pain and for modification of function of abdominal and pelvic organs.¹⁸⁻²⁰

CASE REPORT

A 20-year-old woman presented with a 2-year history of perineal pain, pruritus ani, and bright red bleeding per anus after defecation. She was diagnosed with chronic anal fissure and in the past had conservative therapy that included increased fluid and fiber intake, sitz baths, and topical anesthetics. Later, she used topical glyceryl trinitrate ointment 0.2% twice daily for 8 weeks with no improvement in symptoms but unfortunate side effect of headache. She underwent injection of 25 units of botulinum toxin into the internal anal sphincter followed by clinical evidence of fissure healing at 8 weeks. Unfortunately, after injection of the botulinum toxin, the patient developed incontinence of flatus, which lasted over 6 months. Eighteen months after injection of botulinum toxin, the patient had recurrence of the anal fissure. Colonoscopy was performed to rule out association of fissure with another diagnosis (eg, Crohn's disease, carcinoma). She refused surgical interventions and sought alternative treatment options for her intractable perineal pain in an interventional pain clinic.

The patient rated her pain as 7 out of 10 on the

visual analog scale during defecation and reported perineal pain for several hours after bowel movements. On physical examination, the anal fissure was seen at the posterior midline with horizontal fibers of the internal sphincter muscle in the base of mucosal defect. The mean anal resting pressure was 70 cm H₂O (normal 88 +/- 34 cm H₂O²¹). The patient declined the offer of ganglion impar block because of the fear of recurrence of incontinence of flatus like she experienced after botulinum toxin. She also was counseled regarding use of medications, but she chose to proceed with SNS. No psychological evaluation was performed prior to SNS procedure.

The patient underwent placement of 1 temporary 8-electrode lead for SNS. One 8-electrode standard Octad Lead (Medtronic Inc., Minneapolis, Minn) was placed in the epidural space percutaneously through the sacral hiatus (Figure 1) with final lead electrodes positioned between S1 and S4 (Figure 2). The procedure was done with C-arm guidance in an ambulatory surgical center with local anesthesia only. She was discharged home that day, after education regarding the use of the external programmer for temporary stimulation. The stimulation was conducted continuously, for 24 hours per day. Stimulator parameters were programmed for amplitude upper limit of 10.5 volts, pulse width 210 microseconds, and frequency 30 Hz. The patient's actual amplitude use ranged from 0.8 to 1.2 volts. The lead was removed after 2 weeks of continuous stimulation. On the first day of stimulation, the patient reported greater than 50% improvement in pain immediately after turning on SNS. On the 10th day of treatment with SNS, the patient reported 100% pain relief. The mean anal resting pressure decreased to 60 cm H₂O from 70 cm H₂O on the first day of treatment and to 52 cm H₂O at the end of second week when SNS ended. The patient's chronic anal fissure healed in 2 weeks with continuous temporary SNS. The patient had no complications related to the procedure. No recurrence of anal fissure was reported at a 20-month follow-up.

DISCUSSION

In the treatment of chronic anal fissure, surgical interventions and injections of botulinum toxin are effective therapies with high cure rate, but unfortunately are associated with subsequent incontinence. For the past 15 years, newer ways have been sought to decrease the anal sphincter pressure and thereby reduce mucosal ischemia, allowing the chronic anal fissure to heal. It is important to explore all alternative treatments in the management of chronic anal fissure in order to possibly

identify treatments that may carry less risk of serious complications, decrease cost, and shorten the time of recovery.

SNS has been used since the 1980s as a treatment for dysfunction of pelvic and abdominal organs and pelvic pain. This technique has been used successfully to treat patients with fecal incontinence, voiding dysfunction, irritable bowel syndrome, and pelvic pain.^{18-20,22,23} The mechanism of action of SNS is not clear and continues to evolve. The inhibition of pain transmission may be explained by the “gate-control theory” introduced by Melzack and Wall in 1965.²⁴ This theory postulates that activation of large-diameter afferent fibers via application of an externally applied electric field “closes the gate” to pain transmission. The stimulation of large myelinated A-beta fibers, which have the lowest threshold to stimulation, results in inhibition of reception of smaller unmyelinated C-fiber-evoked painful information at the substantia gelatinosa of the dorsal horn.²⁵ SNS also may block the pain by activation of descending antinociceptive pathways,²⁶⁻²⁷ inhibitory effects on the central sympathetic system, segmental inhibition through coarse fiber activation and brain stem loops, spinal inhibition by decreasing gamma-aminobutyric acid levels in the dorsal horn, and a thalamocortical mechanism masking the nociceptive input.²⁸ The neuromodulating effects of electrical stimulation also may alter local blood flow, cause release of endorphins, improve neural metabolism, affect neurotransmitters and axonal conduction, and block cell membrane depolarization.²⁹ The effectiveness of SNS may be explained by potential alteration of several peripheral and central neural mechanisms.

SNS provides a safe, effective, and convenient treatment option for patients suffering from chronic anal fissure and intractable perineal pain. This technique may have advantages over pharmacologic therapy evidenced by the apparent high effectiveness in the healing of anal fissure in this patient treated with SNS. This technique may have advantages over surgical treatment or botulinum toxin injections, which can be complicated by incontinence, an outcome that patients find unacceptable. There are no side effects created by SNS as there are with many medications. Complications related to SNS therapy may include patient discomfort at the subcutaneous tunnel in which electrodes run³⁰ or superficial wound infection in 3% - 17% of patients.³⁰⁻³¹ Patient care of temporary lead involves only keeping the external dressing clean and dry and maintaining the external cord and generator for ongoing therapy during the time the therapy is being used. The therapy is completely

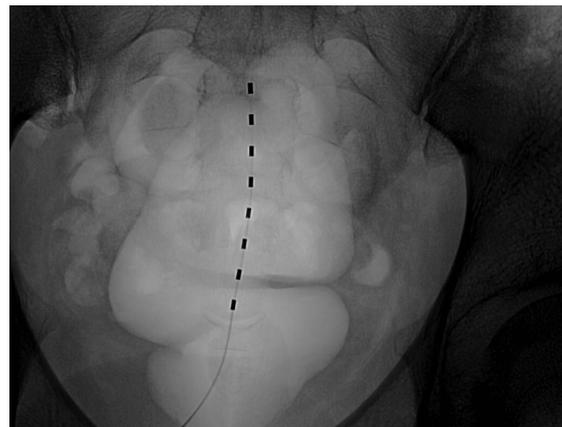


Figure 2. Final lead position in the epidural space between S1 and S4 with lead tip at S1.

reversible when it is no longer needed after the healing of the fissure. The external programmer for temporary stimulation gives the patient full control of the level of stimulation perceived based on severity of pain.

SUMMARY

Temporary SNS using a caudal approach appears to be an effective option in the treatment of chronic anal fissures. This therapy may allow patients rapid healing compared to pharmacological treatment, along with avoidance of debilitating complications often related to surgical procedures or botulinum toxin injections such as bowel incontinence, which seriously affects the patient’s quality of life. This technique may be an option for patients who defer surgical treatment, individuals who developed side effects to medical therapy, or patients with recurrence of fissure. In the future, the prospective studies comparing SNS to other therapies can be used to elucidate the mechanism of action and confirm long-term advantages of this promising and minimally invasive treatment.

REFERENCES

- Schouten WR, Briel JW, Auwerda JJ. Relationship between anal pressure and anodermal blood flow: the vascular pathogenesis of anal fissures. *Dis Colon Rectum*. 1994;37:664-669.
- Jonas M, Scholefield JH. Anal fissure. *Gastroenterol Clin North Am*. 2001;30:167-181.
- Nyam DC, Pemberton JH. Long-term results of lateral internal sphincterotomy for chronic anal fissure with particular reference to incidence of fecal incontinence. *Dis Colon Rectum*. 1999;42:1306-1310.
- Garcia-Aguilar J, Belmonte C, Wong WD, Lowry AC, Madoff RD. Open vs. closed sphincterotomy for chronic anal fissure: long-term results. *Dis Colon Rectum*. 1996;39:440-443.
- Hyman N. Incontinence after lateral internal sphincterotomy: a prospective study and quality of life assessment. *Dis Colon Rectum*. 2004;47:35-38.

6. Elsebae M. A study of fecal incontinence in patients with chronic anal fissure: prospective, randomized, controlled trial of the extent of internal anal sphincter division during lateral sphincterotomy. *World J Surg.* 2007;31:2052-2057.
7. Nelson R. A systemic review of medical therapy for anal fissure. *Dis Colon Rectum.* 2004;47:422-431.
8. Baraza W, Boereboom C, Shorthouse A, Brown S. The long-term efficacy of fissurectomy and botulinum toxin injection for chronic anal fissure in females. *Dis Colon Rectum.* 2008;51:239-243.
9. Lysy J, Israely E, Levy S, Rozentzweig G, Strauss-Liviatan N, Goldin E. Long-term results of "chemical sphincterotomy" for chronic anal fissure: a prospective study. *Dis Colon Rectum.* 2006;49:858-864.
10. Lund JN, Scholefield JH. Glyceril trinitrate is an effective treatment for anal fissure. *Dis Colon Rectum.* 1997;40:468-470.
11. Perrotti P, Bove A, Antropoli C, et al. Topical nifedipine with lidocaine ointment vs. active control for treatment of chronic anal fissure: results of a prospective, randomized, double-blind study. *Dis Colon Rectum.* 2002;45:1468-1475.
12. Evans J, Luck A, Hewett P. Glyceril trinitrate vs. lateral sphincterotomy for chronic anal fissure: prospective, randomized trial. *Dis Colon Rectum.* 2001;44:93-97.
13. Lund JN, Scholefield JH. Follow-up of patients with chronic anal fissure treated with topical glyceryl trinitrate [letter]. *Lancet.* 1998;352:1681.
14. Lund JN, Parsons JL, Scholefield JH. Spasm of the internal anal sphincter in anal fissure—cause or effect? *Gastroenterology.* 1996;110:A711.
15. Zubery BF, Rajput MR, Abro H, et al. A randomized trial of glyceryl trinitrate ointment and nitroglycerin patch in healing of anal fissures. *Int J Colorectal Dis.* 2000;15:243-245.
16. Cook T, et al. Oral nifedipine is an effective treatment for chronic anal fissures. *Colorectal Dis.* 1999;1:55.
17. Kocher HM, Steward M, Leather AJ, et al. Randomized clinical trial assessing the side-effects of glyceryl trinitrate and diltiazem hydrochloride in the treatment of chronic anal fissure. *Br J Surg.* 2002;89:413-417.
18. Comiter CV. Sacral neuromodulation for the symptomatic treatment of refractory interstitial cystitis: a prospective study. *J Urol.* 2003;169:1369-1373.
19. Siegel S, Paszkiewicz E, Kirkpatrick C, Hinkel B, Oleson K. Sacral nerve stimulation in patients with chronic intractable pelvic pain. *J Urol.* 2001;166:1742-1745.
20. Dudding TC, Vaizey CJ, Jerrett ME, Cohen RG, Kamm MA. Permanent sacral nerve stimulation for treatment of functional anorectal pain: report of a case. *Dis Colon Rectum.* 2007;50:1-4.
21. Arabi Y, Alexander-Williams J, Keighley MR. Anal pressures in hemorrhoids and anal fissure. *Am J Surg.* 1977;134:608-610.
22. Jerrett ME, Matzel KE, Christiansen J, et al. Sacral nerve stimulation for faecal incontinence in patients with previous partial spinal cord injury including disc prolapsed. *Br J Surg.* 2005;92:734-739.
23. Whitmore KE, Payne CK, Diokno AC, Lukban JC. Sacral neuromodulation in patients with interstitial cystitis: a multicenter clinical trial. *Int Urogynecol J.* 2003;14:303-309.
24. Melzack RA, Wall PD. Pain mechanisms: a new theory. *Science.* 1965;150:971-979.
25. Hanai F. Effect of electrical stimulation of peripheral nerves on neuropathic pain. *Spine.* 2000;25:1886-1892.
26. Stillier CO, Linderth F, O'Connor W, et al. Repeated spinal cord stimulation decreases the extracellular level of gamma-aminobutyric acid in periaqueductal grey matter of freely moving rats. *Brain Res.* 1995;699:231-241.
27. Linderth B, Gaxelius B, Franck J, et al. Dorsal column stimulation induced release of serotonin and substance P in the cat dorsal horn. *Neurosurgery.* 1992;31:289-296.
28. Kemler MA, et al. Pain relief in complex regional pain syndrome due to spinal cord stimulation does not depend on vasodilatation. *Anesthesiology.* 2000;92:1653-1660.
29. Novak CB, Mackinnon SE. Outcome following implantation of a peripheral nerve stimulator in patients with chronic nerve pain. *Plast Reconstr Surg.* 2000;105:1967-1972.
30. Tjandra JJ, Lim JF, Matzel K. Sacral nerve stimulation: an emerging treatment for faecal incontinence. *ANZ J Surg.* 2004;74:1098-1106.
31. Falletto E, et al. Is sacral nerve stimulation an effective treatment for chronic idiopathic anal pain? *Dis Colon Rectum.* 2009;52:456-462.

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