# **DORSAL COLUMN STIMULATION FOR LUMBAR SPINAL STENOSIS**

## Gilbert S. Chandler, III, MD<sup>,</sup> Bruce Nixon, MD, L. Todd Stewart, MD and Jennifer Love

Surgical decompression has been considered the gold standard for the symptomatic spinal stenotic patient. Thirty thousand decompressive procedures are performed annually and this number is expected to increase as the American population ages. Options are limited for the stenotic patient classified as a "poor surgical risk". Furthermore review of the literature indicates mixed results even in optimal populations. Nonsurgical approaches including epidural steroids and percutaneous adhesiolysis have not been completely evaluated.

Spinal cord stimulation has a long safe efficacious history in the treatment of neuropathic extremity pain but has never been evaluated in the treatment of spinal stenosis.

This retrospective cohort of 55 patients receiving spinal cord

stimulation was selected from a total of 72 patients presenting with spinal stenosis over a 4 year period. Twenty-one underwent subsequent permanent implantation with success rate of 67% at 1.5 years. Twelve elected to not receive implant despite " successful trial". 22 had "failed trial". Verbal pain scores, narcotic intake, and function were monitored.

Spinal cord stimulation is a promising nondestructive alternative in the treatment of symptomatic spinal stenosis. Mild-moderate stenosis, predominate leg pain, and "positive" exercise treadmill appear to be positive predictors. Prospective trials with rigorous statistical designs are needed.

*Keywords:* Spinal stenosis, Decompressive surgery, Dorsal column stimulation

Lumbar spinal stenosis (LSS) is a common entity in the elderly and is likely to become more prevalent with the graying of the U.S. population. In fact, it has been estimated that over 30,000 surgical procedures are performed annually for degenerative spinal stenosis (1).

Clinically LSS is characterized by the onset of neurogenic claudication. The pathogenesis remains controversial and may be related to vascular insufficiency or mechanical compression (2). Typically this pain is initiated by standing and/or walking. The onset of leg pain can be variable and may be described as an "achiness" or heaviness in the calves (3). Relief is usually obtained by forward flexion "Simeon stance" and assuming the seated position (4). In addition, walking uphill tends to delay radicular symptoms, while walking downhill tends to extend the spine, thus hastening leg pain (5). This positional pain syndrome, although rarely leading to "paralysis", can lead to significant disability as the disease progresses. MRI, CT, and CT/myelogram are all utilized, but cannot predict the pain severity or functional status of the patient

(6-9). Classic radiographic abnormalities are a sagittal diameter of less than 11 mm, spinal canal area of less than 1.45 cm and a lateral recess depth of less than 3 mm (10, 11). Obliterated fatty tissues and hypertrophic soft tissues may also be noted by MRI scan (12). EMG abnormalities are common, but may be less specific with more than one involved level (13, 14).

Furthermore, the surgical approach typically results in significant pain reduction and lifestyle restoration (15, 16). Fluoroscopically-guided selective nerve root injection may also be helpful in identifying the symptomatic level in cases of multilevel disease (17). Decompressive surgery has emerged as the benchmark of treatment when conservative approaches have failed or major neural deficits or evolving neural damage is present (18). The maturation of this surgical decompression has raised several questions regarding improved outcome. Outcome predictors include health self-perception, low cardiovascular co-morbidity (19), multiple laminectomy (16), and predominant back pain component (20). In addition, the cost effectiveness of instrumented fusion yields poor incremental gains in outcome versus non-instrumented fusion (21). This becomes particularly important when evaluating the patient with multilevel spondylotic pathology. A literature review also reveals persistent back pain after surgical decompression, and repeat operation and significant deterioration in walking capacity (22). Turner et al (23) in a large meta-analysis concluded that a good-excellent outcome is achieved in only 64% of stenotic patients. These observations initially led this group to consider a non-destructive procedure as a precursor/alternative to decompressive laminectomy in a select group of stenotic patients with co-morbid cardiac and pulmonary disease. Initially, these patients were felt to be a significant perioperative risk secondary to their co-morbid conditions and diffuse spinal pathology.

Spinal cord stimulation (SCS) has a long history of efficacy regarding painful radiculopathy in the "failed back syndrome" (24-26). The capability to perform a percutaneous lead placement further allowed assessment prior to consideration of permanent implant. This "trial" can be performed with low morbidity and emulates the permanent procedure (27). SCS therapy carries a favorable cost-effectiveness profile in patients with persistent pain and failed back surgery syndrome (28, 29). A 3-5 day trial with the percutaneous leads placement as an outpatient is a routine at our station. Efficacy is monitored by the patient's pain report, analgesic use, and activity level. If the intensity of pain reported is improved by at least 50%, analgesic use is decreased

From Departments of Pain Management and Neurosurgery Specialty Clinics of Georgia, Gainesville, GA. Address correspondence: Gilbert S. Chandler, III, MD, Specialty Clinics of Georgia, PC, Department of Pain Management, 1240 Jesse Jewell Parkway, Suite #300, Gainesville, GA 30501. E-mail: sewell8/@aol.com.

Funding: Sponsored in part by Medtronic, Inc

and the patient describes an increased activity level, then permanent implantation is offered. We utilize a three week follow-up after the trial to eliminate or "wash-out" placebo impact. During this visit all data is reviewed and the decision whether to offer a permanent implant is made. After 2.5 years into data collection, an exercise treadmill was incorporated 1-2 days pre-and-post trial lead placement to quantify impact of SCS. Further "a disinterested third party observer" (treadmill operator), observation is added. This adds a reproducible, and a quantifiable means of assessing the SCS's functional impact on the physiologic detriment imposed by spinal stenosis (8, 30). Despite an approximately 30-year history of SCS, continued refinement of patient selection, and advances in implantable hardware, this series represents the first-reported specific role of SCS for lumbar spinal stenosis.

#### **M**ETHODS

Seventy-two consecutive patients referred to our multi-specialty clinic for management of spinal stenosis after failure of conservative care. Each participant underwent evaluation by interventional pain management and neurosurgery. All had been diagnosed as having spinal stenosis with CT, CT/myelogram, or MRI. This retrospective study examined the outcome of SCS to all patients referred to our facility for the management of LSS over a four year period. Chart review, office visit, and phone follow-up were performed by the principal investigators. Patients were advised to return as needed after the initial post-op visit. Routine follow-up visits were not scheduled, therefore data was collected by office visits, phone contact and chart review. Seventy-two consecutive patients had LSS confirmed by CT scan (with or without myelogram) or MRI. All patients received fluoroscopicguided transforaminal steroid injections with highly variable degrees of benefit. Statistical analysis of response to transforaminal injections was not performed. Once therapeutic benefit was no longer demonstrated SCS was offered to those who met inclusion criteria. Inclusion criteria included; consent to treatment and some component of leg pain. Exclusion criteria was; failure to consent, pure axial back pain and anticoagulant therapy. After thorough discussion of the rational, risks, and expected benefits trial leads were placed. This was accomplished in the operating room with strict sterile conditions and fluoroscopic guidance. All were placed with local anesthesia and minimal sedation. A single lead "Quad Plus" was utilized in all cases, analgesic coverage was achieved with placement in the vertebral mid-Thoracic-9 vicinity for most cases. Patients were discharged that day and followed up 4 -5 days later. At that time, temporary lead was removed and SCS efficacy was evaluated by the physician. Repeat evaluation was then performed 14 - 21 days later in the "successful trials" to diminish any placebo and, if still deemed beneficial referral to neurosurgeon for permanent implant was made. Permanent implants were then placed in the operating room with monitor anesthesia care anesthesia as 23-hour admissions. The "Resume" Medtronic paddle lead was inserted with a mini-laminotomy in all cases. Post-operative follow up was at one week by the neurosurgeon or as needed by the pain management team. Patient followup and chart review examined characteristics such as patient age, radiographic characteristics, subjective descriptors and response to selective nerve blockade. Subjective pain relief, functional improvement, and changes in medications were reviewed at trial completion, and 1.5 years. Any complications were recorded. Pain relief of greater than 50 % and/or improvement of functional status with decrease in medications was considered significant.

Extensive statistical analysis was not performed secondary to retrospective design, small sample population, and lack of suitable control.

### RESULTS

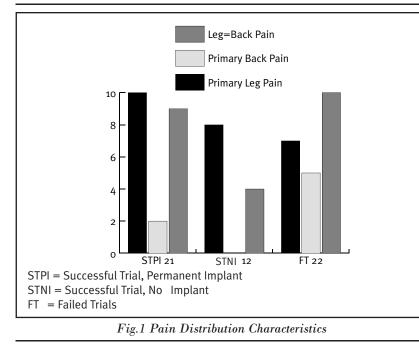
Fifty-five patients with the diagnosis of LSS received a spinal cord stimulation trial from December 1997 to December 2001. . The average age was 77 years, and the range was 58 - 88. All had predominately leg pain (neurogenic claudication), and none had axial pain only. The degree of spinal stenosis was not quantified on all patients. The degree of impairment was variable ranging from mild to wheel chair dependency. Pre-op medications ranged from NSAIDS only to methadone with the majority on some form of schedule II or III narcotics. Visual analogue scores (VAS) were tracked - ranging from 4-10, average 8. Psychological evaluations were not performed as overt psychopathology nor obvious secondary gain issues were

evident. No complications were noted. Twenty-one were deemed "successful" (successful trial-permanent implant or STPI) thus receiving implants. Twentytwo were labeled "failed trial - no implant" (FTNI). Twelve had positive trials, but did not progress to permanent implant status (successful trial-no implant, STNI). Analgesia, medication intake, and change in activities of daily living (ADL's) were considered in the stimulator evaluation. A reduction of pain by 50% or greater was considered significant. This combined with an improvement of ADL's usually warranted permanent implant status even if medications were unchanged. Later the exercise treadmill was also utilized to measure the impact of stimulation on ADL's, specifically neurogenic claudication. There were no statistically significant differences in age between the three groups (average age 73). The STPI group demonstrated improved analgesia (20/21), improved ADL's (13/14) and reduced medication requirements (17/21) at trial completion. 14/21 experienced persistent analgesia (67%) at 1.5 vears. This was defined as; greater than 50% subjective relief and/or decrease in medication or improvement in function. Two were lost to follow-up and five were classified as failures. One of the "failures" deemed a nonsurgical candidate had undergone permanent implantation without a trial secondary to inability to place a percutaneous lead.

The STNI group (n = 12) also had significant analgesia, and improved ADL's with stimulation. Reasons for implant refusal were as follows: three expired prior to implant, spouse death (one), one could not recall trial, three "changed their mind", one underwent hip surgery, and three labeled the stimulation as dysesthetic.

### DISCUSSION

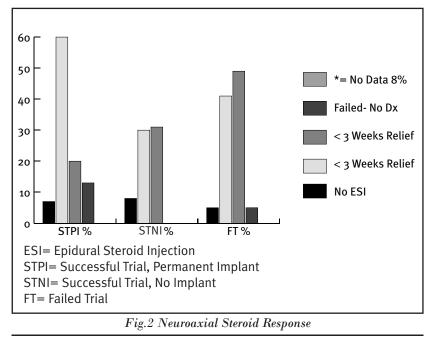
Spinal stenosis is a progressive disease and is not static. Furthermore, this progression is affirmed in the literature by the need for additional operations and a deteriorating surgical result (22). Johnsson et al (31) noted that in surgical patients 60% improved, while 25% deteriorated compared to the conservatively treated patients, 30% improved and 60% remained unchanged. Johnsson et al (32) in examining the natural course of spinal stenosis concluded that in 70% symptoms were unchanged, worse in 15%, and improved in 15% over an approximate 4 year



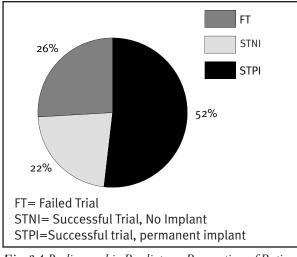
period. Initially our decision to utilize SCS for this clinical entity was restricted to patients whom were declared "poor" surgical risk by their primary physician. However, as our experience grew, the inclusion criteria began to expand as an alternative to decompressive surgery. This study may be criticized for its retrospective nature, but this was the logical starting point because there are no large stenosis-specific studies. There was no attempt to perform psychologic evaluation as a precursor to stimulator trial, possibly allowing psychological co-variables to increase the failure rate. Multiple variables were examined in an attempt to predict outcome including age, pain location, subjective pain descriptors, response to fluoro-guided, injections, stenosis severity (radiologic) and treadmill performance. Pain location was divided into three categories; predominately leg or back, or back equal leg pain. In our series, radicular pain appeared to be a positive predictor comprising the majority of the successful trials (Fig. 1). All trials utilized a single 4channel lead, which is consistent with the historical "best" stimulator candidate (33-35). Future studies might consider the impact of "dual-lead" or multi-channel stimulation to obtain wider pain coverage and possibly reduce coverage decay as the disease progresses.

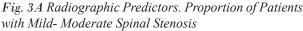
Several studies have investigated the role of epidural steroids in spinal stenosis with mixed results. Fukusaki et al (36) showed no benefit for neuro-claudication comparing interlaminar steroid versus placebo. However, Ciocon et al (37) demonstrated a significant reduction of pain with caudal epidural steroids. Furthermore Manchikanti et al (38) showed promise with percutaneous adhesiolysis in a cohort of 18. In this group between 1 and 10 adhesiolysis procedures were performed over 3 years. 56% received 4 injections and there appeared to be a cumulative benefit with each injection. However, significant therapeutic decay was still observed over time. We elected to proceed directly to spinal cord stimulation if minimal therapeutic benefit was demonstrated with transforaminal epidural steroid injections. This was based upon lack of literature-based efficacy for any non-surgical modalities including hypertonic saline adhesiolysis at the time of this study. Furthermore, the risk/ benefitratio of spinal cord stimulation was very appealing. In our experience of 55 patients, all have failed between one to three fluoro-guided transforaminal injections prior to consideration for SCS. Derby et al (39) have examined the predictive value of steroid response to surgical outcome and we extrapolated that would be similar for spinal stenosis. However, there was no predictive value in the steroid response for SCS outcome (Fig. 2). In fact, the "failed trial" had the best response to the transforaminal blocks. Perhaps in this group (prolonged steroid effect) multiple injections should be considered as per Manchikanti's protocol. In addition, this may imply more of a nociceptive component to the stenosis, thus predictive of a poor outcome with stimulation (40).

Numerous studies have examined the relationship between the degree of radiologic pathology, symptomology and outcome with variable results. Amundsen et al (41) failed to demonstrate any association between pain severity and degree of stenosis. Katz et al (42) concluded that



116





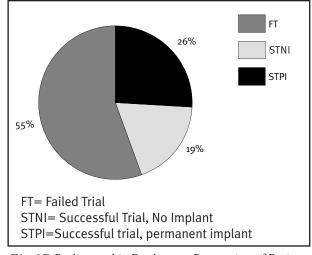


Fig. 3B Radiographic Predictors. Proportion of Patients with Severe Spinal Stenosis

radiographic variables are not associated with outcome in a prospective 2-year postoperative study of stenotic patients. Our data, on the other hand, suggests that patients with severe stenosis are more likely to have a "failed trial". Those with mild-to-moderate stenosis seem to experience a "positive trial" proceeding to full implant (Fig. 3A & 3B). The GTNI group had an almost equal number of severely and mild-moderate stenotic patients. This "ambivalent" group may indeed be Katz's group with poor health perception; therefore future trials should emphasize health perceptions and expectations, as he suggested (42).

The role of the exercise treadmill test (ETT) in assessing the impact of decompressive laminectomy has been well described (8) and was eventually incorporated into our evaluation. We evaluated the impact of SCS on walking distance (minutes), speed, heart rate, verbal pain scores every 30 sec, and time to maximum VPS. Positive ETT was defined as improved walking distance or decreased work load (heart rate decreased) and/or decreased verbal pain score. Although the ETT sample size was too small for bold conclusions, the ability to quantify walking distance, VAS scores, and hemodynamic variables pre- and post-SCS lead insertion was intriguing (Table 1). In fact, in most cases the ETT showed improvement of VAS scores and hemodynamic measures. Potentially, as our knowledge of ETT improves, it may become a very "sensitive" tool for SCS screening. Deen et al (8) suggests ETT can be effectively utilized preoperatively to defer surgery if an unexpectedly positive trial occurs. Treadmill testing may assist in distinguishing between generalized deconditioning (fatigue without leg pain) versus true neurogenic claudication (leg pain). The deconditioned group therefore may consider reconditioning prior to a SCS trial.

Subjective pain descriptors were also examined with respect to outcome. Pain

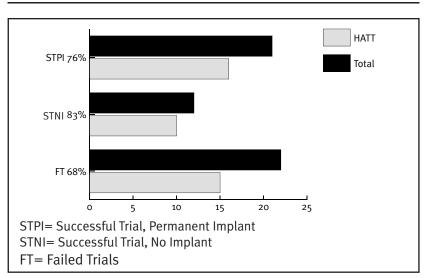
described as "all the time" or made worse "by everything" were felt to be harbingers of a psychological component and not characteristic of classic neurogenic claudication, thus precluding a successful trial. While psychological distress can impact pain perceptors its role in determining outcome is less clear (43). The combination of the primary author's 9 year experience with SCS and the obvious spine pathology is this cohort lulled us into the abandon-

Patient #1	Pre 2 1/2 Minutes	1.5 mph	VPS= 9	HR- 110
	Post 4 Minutes	2.5 mph	VPS= o	HR- 106
Patient #2	Pre 5 Minutes	1.5 mph	VPS= 8	HR- 138
	Post 10 Minutes	1.5 mph	VPS=7	HR- 137
Patient #3	Pre 30 Seconds	o mph	VPS= 3	HR- 68
	Post 1 1/2 Minutes	.5 mph	VPS= 4	HR- 106
Patient #4	Pre 10 Minutes	2.0 mph	VPS= 6	HR- 106
	Post 12 Minutes	2.0 mph	VPS= 2.5	HR- 98
Patient #5	Pre 2 Minutes	1.0 mph	VPS= 9	HR- 77
	Post 12 Minutes	1.2 mph	VPS= 2	HR- 74
Patient #6	Pre 8 1/2 Minutes	.9 mph	VPS= 10	HR- 110
	Post 9 Minutes	1.0 mph	VPS= 8	HR- 112
Patient #7	Pre 3 1/2 Minutes	.5 mph	VPS= 8	HR- 83
	Post 3 Minutes	.5 mph	VPS= 5	HR- 96
Patient #8	Pre 8 1/2 Minutes	1.0 mph	VPS= 8.5	HR- 80
	Post 1 1/2 Minutes	1.0 mph	VPS= 2	HR- 95
Patient #9	Pre 1 1/2 Minutes No Post	.5 mph	VPS= 8	HR- 75
Patient #10	Pre 2 Minutes	1.0 mph	VPS= 8	HR- 64
	Post 3 Minutes	1.0 mph	VPS= o	HR- 93
Patient #11	Pre 10 Minutes	3.0 mph	VPS= 2	HR- 120
	Post 10 Minutes	3.3 mph	VPS= 5	HR- 113

VPS= Verbal Pain Score HR= Heart Rate SCS= Spinal Cord Stimulator MPH= Miles Per Hour

Patient #12	Pre 3 Minutes Post 21/2 Minutes	2.0 mph 2.0 mph	VPS= 10 VPS= 6	HR- 83
Patient #13	Pre 16 Minutes	2.5 mph	VPS= 2	HR- 87
	Post 17 Minutes	2.5 mph	VPS= 2	HR- 123
Patient #14	Pre 6 Seconds	1.8 mph	VPS= 4	HR- 120
	Post 10 Minutes	<u>2.0 mph</u>	VPS= o	HR- 120
Patient #15	Pre 6 Minutes	1.5 mph	VPS= 8	HR- 140
	Post 5 1/2 Minutes	2.5 mph	VPS= 5	HR- 81
Failed Trial				
Patient #16	Pre 5 Steps		VPS= 10	HR- 94
	Post 37 Steps		VPS= 10	HR- 84

VPS= Verbal Pain Score HR= Heart Rate MPH= Miles Per Hour



Subjective Descriptors. Proportion of patients describing that Fig. 4 "everything hurts all the time" (HATT)

ment of our usual psychological screening. Interestingly, in isolating these subjective variables the STNI had the highest prevalence (Fig. 4). Future recommendations might be a limited psychological assessment (age-specific) that evaluates cognitive function along with standard psychological variables.

### CONCLUSION

In summary, while limited by its retrospective nature, this represents the largest reported number of cases of spinal stenosis undergoing SCS. Many questions arose in attempting to identify the "ideal" patient and similar to a traditional surgical approach success is multi-factorial. "Overall success rates for SCS trials in patients with spinal stenosis were 60% and for permanent SCS implants were 67%. Their success rates were similar

to previously published success rates for other SCS indications such as Failed Back Surgery Syndrome" (33). It is possible that if patients were screened for significant co-morbid disease, the success rates may have been higher. Earlier studies show a wide degree of "success" with SCS (44, 45). The "failed trials" are not factored in long-term follow up (46). This is common in evaluating procedures for chronic pain where a screening test is utilized and would affect the outcome for our study population. This study suggests SCS warrants future investigation especially when 7. compared to a non-reversible, destructive approach with its own wide range of reported success (23). Although our numbers are small, it appears that predominate "leg pain", mild-to-moderate stenosis and a positive treadmill test are the best predictors for a sustained benefit.

Future considerations would include cognitive and psychological testing, pre-trial "TENS" application (screening for technical overload), and utilization of multichannel leads. Ultimately, prospective trials are needed to determine the role of SCS in treating lumbar spinal stenosis.

# Author Affiliation:

### Gilbert S. Chandler, III, MD

Medical Director, Department of Pain Management, Specialty Clinics of Georgia, 1240 Jesse Jewell Parkway, Suite #300, Gainesville, GA 30501. E-mail:sewell87@aol.com.

# Bruce Nixon, MD

Department of Neurosurgery, Specialty Clinics of Georgia.1240 Jesse Jewell Parkway, Suite #300, Gainesville, GA 30501.

## L. Todd Stewart, MD

Department of Pain Management, Specialty Clinics of Georgia, 1240 Jesse Jewell Parkway, Suite #300, Gainesville, GA 30501. and

# Jennifer Love

Clinical Research Coordinator at the Specialty Clinics of Georgia, 1240 Jesse Jewell Parkway, Suite #300, Gainesville, GA 30501.

#### REFERENCES

5.

- Taylor VM, Deyo RA, Cherkin DC et al. Low 1. back pain hospitalization: Recent United States trends and regional variations. Spine 1994; 17:1207-1213.
- 2. Arnoldi CC, Brodsky AE, Cauchoix J et al. Lumbar spinal stenosis and nerve root entrapment syndromes: Definition and Classification. Clin Orthop 1976; 115:4-5.
- Borestein DG. Chronic low back pain. 3. Rheum Dis Clin North Am 1996; 22: 439-455.
- Simkin PA. Simeon stance: A sign of spinal 4. stenosis. Lancet 1982; 2: 652-653
  - Dai LY, Xu YK, Zhang WM et al. The effect of flexion-extension motion of the lumbar spine on the capacity of the spinal canal: An experimental study. Spine 1989; 14950: 523-525.
- 6. Nagler, W, Bodack MP. Management options in lumbar spinal stenosis. In Ernst E, Jayson MIV, Pope MH (eds). Advances in Idiopathic Low Back Pain, Blackwell-Mzy, Vienna, 1993, pp 292-297.
- Admundsen T, Weber H, Lilleas F et al. Lumbar spinal stenosis. Clinical and radiologic features. Spine 1995; 20: 1178-1186
- Deen HG, Zimmerman RS, Lyons MK et al. 8. Use of exercise treadmill to measure baseline functional status and surgical outcome in patients with severe lumbar spinal stenosis. Spine 1998; 23: 244-248.
  - Stucki G, Liang MH, Lipson SL et al. Con-

tribution of neuromuscular impairment to physical functional status in patients with lumbar spinal stenosis. *Rhuematol* 1994; 21:1338-1343.

- Onel D, Sari H, Donmez C. Lumbar spinal stenosis: Clinical/radiologic therapeutic evaluation in 125 patients: Conservative treatment or surgical intervention? *Spine* 1993; 18: 291-298.
- Ullrich CG, Binet EF, Sanecki MG et al. Quantitative assessment of the lumbar spinal canal by computed tomography. *Radiology* 1980; 134:137-143.
- Boden SD, Davis DO, Dina TS et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects: A prospective investigation. *J Bone Joint Surg (Am)* 1990; 72:403-408.
- Jacobson RE. Lumbar Stenosis. An electromyographic evaluation. *Clin Orthop* 1976; 115: 68-71.
- 14. Leyslion A, Kirwan EOG, Parry CB. Electrical studies in the diagnosis of compression of the lumbar root. *J Bone Joint Surg* (*Br*) 1981; 63:71-75.
- Leregui-Lauber T, Vader JP, Burnand T et al. Appropriateness of indications for surgery of lumbar disc hernia and spinal stenosis. *Spine* 1997; 22:203-209.
- Iguchi T, Kurihara A, Nakayama J et al. Minimum 10-year outcome of decompressive laminectomy for degenerative lumbar spinal stenosis. *Spine* 2000; 25:1754-1759.
- 17. Schillberg B, Nystrom B. Quality of life before and after microsurgical decompression in lumbar spinal stenosis. *J Spinal Disord* 2000; 13: 237-241.
- Sanderson PL, Wood PL. Surgery for lumbar spinal stenosis in old people. *J Bone Joint Surg (Br)* 1993; 75:393-397.
- 19. Katz JN, Stucki G, Lipson SJ et al. Predictors of surgical outcome in lumbar spinal stenosis. *Spine* 1999; 24:2229-2233.
- 20. Katz JN, Lipson SJ, Brick GW et al. *Spine* 1995; 20:1155-1160.
- 21. Kuntz KM, Snider RK, Weinstein JN et al. Cost-effectiveness of fusion with and without instrumentation for patients with degenerative spondylolisthesis and spinal stenosis. *Spine* 2000; 25:1132-1139.
- 22. Katz JN, Lipson SJ, Chang L et al. Sevento-10-year outcome of decompressive sur-

gery for degenerative lumbar spinal stenosis. *Spine* 1996; 21: 92-98.

- 23. Turner JA, Ersek M, Herron L et al. Surgery for lumbar spinal stenosis Attempted meta-analysis of the literature. *Spine* 1992; 17:1-6.
- 24. Kumar K, Toth C, Nath RK et al. Epidural spinal cord stimulation for treatment of chronic pain-some predictors of success. A 15-year experience. *Surg Neurol* 1998; 50:110-120.
- 25. Barolat G, Ketcik B, He J. Long-term outcome of spinal cord stimulation for chronic pain management. *Neuromodulation* 1998; 1:19-29.
- 26. Burchiel KJ, Anderson VC, Brown FD et al. Prospective, multi-center study of spinal cord stimulation for relief of chronic back and extremity pain. *Spine* 1996; 21:2786-2794.
- 27. North RB, Guarino AH. Spinal cord stimulation for failed back surgery syndrome: Technical Advances, patient selection and outcome. *Neuromodulation* 1999; 2: 171-178.
- 28. Bel S, Bauer BL. Dorsal column stimulation: cost to benefit analysis. *Acta Neurochir Suppl (Wein)* 1991; 52:121-123.
- Bell GK, Kidd D, North RB. Cost-effectiveness analysis of spinal cord stimulation in treatment of failed back surgery syndrome. *J Pain Symptom Manage* 1997; 13: 286-295.
- 30. Deen HG, Zimmerman RS, Lyons MK et al. Test-retest reproducibility of the treadmill examination in lumbar spinal stenosis. *Mayo Clin Proc* 2000; 75:1002-1007.
- Johnsson KE, Uden A, Rosen I. The effect of decompression on the natural course of spinal stenosis: A comparison of surgically treated and untreated patients. *Spine* 1991; 16:615-619.
- 32. Johnsson KE, Rosen I, Uden A. The natural course of lumbar spinal stenosis. *Clin Orthop* 1992; 279:82-86.
- North RB, Ewend MG, Lawton MT et al. Failed back surgery syndrome: Five-year follow-up after spinal cord stimulator implantation .A prospective, randomized study design. *Neurosurgery* 1991; 28:692-699.
- 34. North RB, Kidd DH, Zahurak M et al. Spinal cord stimulation for chronic, intracta-

ble pain: Two decades' experience. *Neurosurgery* 1993; 32:384-395.

- 35. Barolat G. A prospective multi-center study to assess the efficacy of spinal cord stimulation utilizing a multi-channel radiofrequency system for the treatment of intractable low back and lower extremity pain. *Neuromodulation* 1999; 2:179-183.
- Fukusaki M, Kobayashi I, Hara T et al. Symptoms of spinal stenosis do not improve after epidural steroid injection. *Clin* J Pain 1998;14:148 -151.
- Ciocon JO, Galindo-Clocon D, Amarnath et al. Caudal epidural blocks for elderly patients with lumbar canal stenosis. J Am Geriatric Soc 1994; 42:593-596.
- Manchikanti L, Pampati V, Fellows B et al. Effectiveness of percutaneous adhesiolysis with hypertonic saline neurolysis in refractory spinal stenosis. *Pain Physician* 2001; 4:366-373.
- Derby R, Kine G, Saal JA et al. Response to steroid and duration of radicular pain as predictors of surgical outcome. *Spine* 1992; 17: S176-S178.
- 40. Marchand S, Bushnell MC et al. The effects of dorsal column stimulation on measures of clinical and experimental pain in man. *Pain* 1991; 45:249-257.
- 41. Admundsen T, Weber H. et al. Lumbar spinal stenosis; clinical and radiologic features. *Spine* 1995; 20:1178-1186.
- Katz JN, Stucki G, Lipson SJ et al. Predictors of surgical outcome in degenerative lumbar spinal stenosis. *Spine* 1999; 24: 2229-2233.
- North RB, Kidd DH, Wimberly RL et al. Prognostic value of psychological testing in spinal cord stimulation patients: a prospective study. *Neurosurgery* 1996; 39: 301-311.
- Erickson DL. Percutaneous trial stimulation for patient selection for implantable stimulating devises. *J Neurosurg* 1975; 43: 440-444.
- Kolin M-T, Winkelmuller W. Chronic pain after multiple lumbar discectomies-significance of intermittent spinal cord stimulation. *Pain* 1991; 44:119-130.
- North RB, Guarino AH. Spinal cord stimulation for failed back surgery syndrome:technical advances, patient selection and outcome. J Neuromodulation 1999; 2: 171-178.