Instrumented Open-Door Laminoplasty as Treatment for Cervical Myelopathy in 104 Patients

John R. Dimar II, MD, Kelly R. Bratcher, RN, CCRP, Dylan C. Brock, BS, Steven D. Glassman, MD, Mitchell J. Campbell, MD, and Leah Y. Carreon, MD, MSc

Abstract

Treatment of multilevel cervical myelopathy remains a challenge. We report on a large series of cervical myelopathy patients treated with instrumented open-door laminoplasty.

We retrospectively examined the medical records of 104 patients who had undergone instrumented open-door laminoplasty (titanium plate) for cervical myelopathy (minimum follow-up, 24 months). All patients had been myelopathic, 57 (54.8%) had stenosis, 39 (37.5%) had spondylosis, 66 (63.5%) reported gait disturbance, 18 (17.3%) had handwriting changes, 33 (31.7%) complained of deterioration of dexterity, 56 (53.8%) had grasp weakness, 7 (6.7%) had bowel and bladder complaints, 27 (26.0%) had a positive Hoffmann sign, 10 (9.6%) had sustained clonus, and 10 (9.6%) had a positive Babinski sign.

Mean preoperative-to-postoperative improvement in Nurick grade was 1.47. Complications included 4 nerve root injuries (3.8%), 1 of which (at C5) was permanent, and 1 transient neurologic deterioration (<1%), 1 incidental durotomy (<1%), and 5 wound infections (4.8%). Four patients required anterior revision for persistent symptoms.

Open-door laminoplasty with miniplate instrumentation is an effective, safe method for preventing progression of myelopathy with multilevel involvement while alleviating the need for multilevel fusion.

Dr. Dimar is Associate Professor, Department of Orthopaedic Surgery, University of Louisville School of Medicine, Louisville, Kentucky.
Ms. Bratcher is Senior Clinical Research Coordinator, and Mr. Brock is Research Assistant, Kenton D. Leatherman Spine Center, Louisville, Kentucky.
Dr. Glassman is Associate Professor, and Dr. Campbell is Assistant Clinical Professor, Department of Orthopaedic Surgery, University of Louisville School of Medicine, Louisville, Kentucky.
Dr. Carreon is Clinical Research Director, Kenton D. Leatherman Spine Center, Louisville, Kentucky.

Address correspondence to: Leah Y. Carreon, MD, MSc, Kenton D. Leatherman Spine Center, 210 E Gray St, Suite 900, Louisville, KY 40202 (tel, 502-584-7525; fax, 502-584-6851; e-mail, lcarreon@spinemds.com).

Am J Orthop. 2009;38(7):E123-E128.

Cervical myelopathy is caused by spinal canal narrowing leading to spinal cord dysfunction.[1,2] The most common etiologies are congenital (developmental) stenosis presenting as early as the third or fourth decade of life and acquired stenosis from age-related degenerative spondylosis.[3,4] Another common etiology is ossification of the posterior longitudinal ligament (OPLL), which produces diffuse and severe narrowing of the spinal canal. Spinal canal narrowing correlated significantly with an increased chance of neurologic injury after cervical trauma, particularly with a fracture-dislocation.[5] Conversely, the same study showed that a larger diameter spinal canal offered protection from a spinal cord injury.[5]

The classic presentation of cervical myelopathy is subtle loss of balance and coordination, decreased hand dexterity, weakness, numbness, and potential paralysis. Most patients with these problems remain unaware of them until they are advanced and are brought up by family members or a physician. Often the lack of significant pain and the ability to compensate for lost neurologic function result in a delay in diagnosis. Fortunately, over the past decade, the ability to accurately diagnose cervical spinal stenosis in the aging population has been facilitated by magnetic resonance imaging (MRI) and has resulted in more expedient treatment.

Although some authors have described nonoperative treatment, most patients manifest with frank myelopathy or significant worsening of neurologic function and are candidates for surgical intervention to arrest progression of the myelopathy.[6-9] The primary goal of surgery is to restore the anteroposterior diameter of the cervical spinal canal to relieve the spinal cord compression.[8,9] Traditionally, the cervical spinal canal diameter was restored with either an anterior procedure that removes portions of the anterior spinal column (discs, vertebral bodies, or both) or a posterior laminectomy with or without concurrent fusion.[9-14] More recently, laminoplasty techniques have become increasingly popular in treating multilevel cervical spinal stenosis.[15-19] Laminoplasty is essentially a multilevel osteotomy that restores the cervical canal diameter while preserving the dorsal roof and ligamentous support of the spinal canal.

In this article, we report 8-year results for 104 patients treated with an instrumented laminoplasty

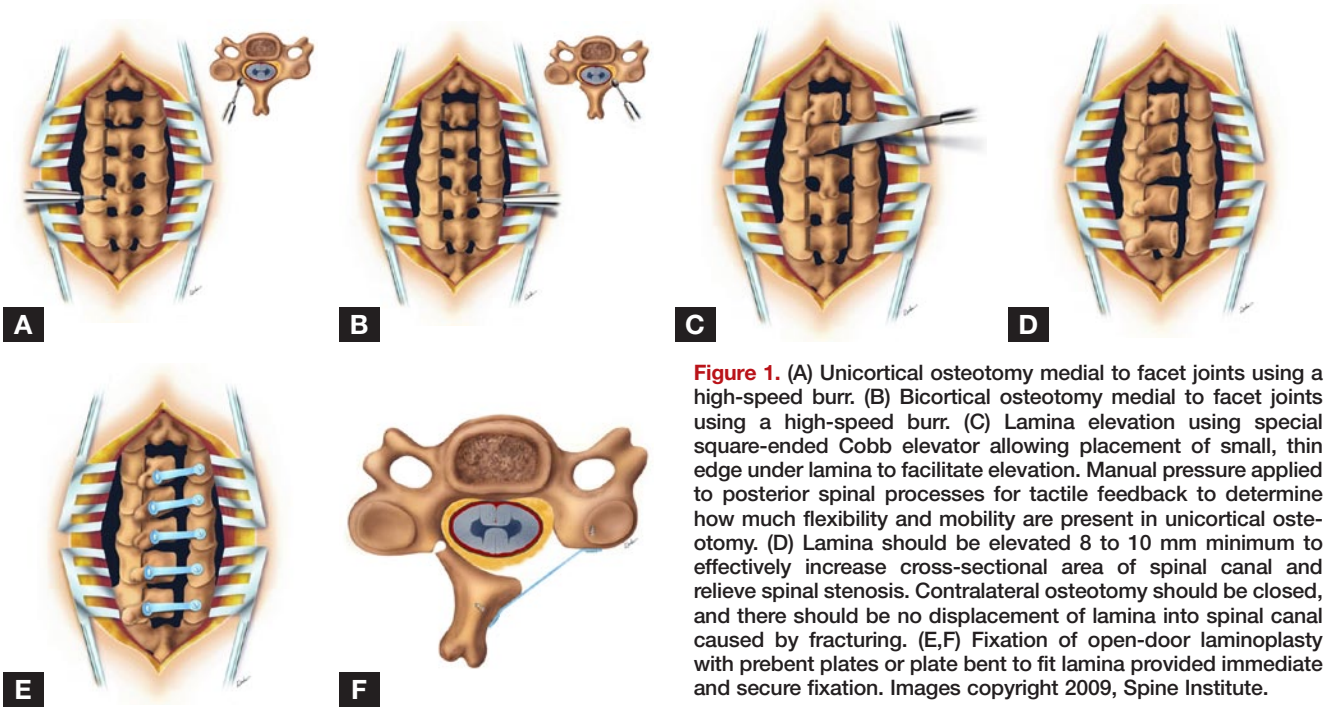

Figure 1. (A) Unicortical osteotomy medial to facet joints using a high-speed burr. (B) Bicortical osteotomy medial to facet joints using a high-speed burr. (C) Lamina elevation using special square-ended Cobb elevator allowing placement of small, thin edge under lamina to facilitate elevation. Manual pressure applied to posterior spinal processes for tactile feedback to determine how much flexibility and mobility are present in unicortical osteotomy. (D) Lamina should be elevated 8 to 10 mm minimum to effectively increase cross-sectional area of spinal canal and relieve spinal stenosis. Contralateral osteotomy should be closed, and there should be no displacement of lamina into spinal canal caused by fracturing. (E,F) Fixation of open-door laminoplasty with prebent plates or plate bent to fit lamina provided immediate and secure fixation. Images copyright 2009, Spine Institute.

technique at a spinal center. We also review the current literature.

Materials and Methods

We identified patients who had been treated for cervical myelopathy between 1998 and 2006 and excluded those who had been treated with anterior vertebrectomy, anterior interbody fusion, anterior strut grafts, or anterior instrumentation or who had a condition, such as cervical kyphosis, that required combined anterior and posterior procedures. We then examined the records of the 104 patients who had had multilevel cervical spinal myelopathy treated with multilevel laminoplasty using miniplates.

We collected standard demographic data: age, sex, symptom duration, comorbidities, work status, and physical findings, including neurologic deficits. Indications for surgery included failed conservative treatment for myelopathy, progressive motor and/or sensory deficit, loss of bowel or bladder function, myelomalacia, and severe radiculopathy. Nurick neurologic functional grading was done before and after surgery.

Surgical Planning and Technique

Preoperative planning was essential in determining the number of levels to be expanded and whether foraminotomies were indicated for relief of radicular symptomatology. In cases of no concurrent radiculopathy, the side opened was the surgeon's preference; in cases of unilateral radicular symptoms, the affected side was preferentially opened; in cases of bilateral radiculopathy and radiographically documented foraminal narrowing, foraminotomies were performed on both sides before rotation of the laminae.

Table I. Summary of History and Physical Examination Data

Preoperative Symptom	n	%
Handwriting changes	18	17.3
Problems holding cups	56	53.8
Change in dexterity	33	31.7
Gait disturbance	66	63.5
Bowel/bladder changes	7	6.7
Balance disturbances	0	0.0
Abnormal reflex		
Positive Hoffmann sign	27	26.0
Sustained clonus	10	9.6
Positive Babinski sign	10	9.6

Table II. Summary of Surgical Data

	n	%
Previous surgery	28	26.9
Indication for surgery		
Stenosis	57	54.8
Myelopathy	49	47.1
Spondylosis	39	37.5
Surgical levels	3.8 ± 0.41	
Right-sided opening	53	50.9
Left-sided opening	50	48.1
Bilateral opening	1	1.0
Operating room time (h:min)	2:44 ± 0:41	
Estimated blood loss (mL)	250.0 ± 82.1	
Postoperative complication		
Cord injury	0	0.0
Root injury	4	3.8
Neurologic deterioration	1	<1.0
Dural tear	1	<1.0
Infections	5	4.8
Nurick grade		
Preoperative	2.11 ± 0.85	
Postoperative	0.63 ± 0.69	
Improvement	1.47 ± 0.92	

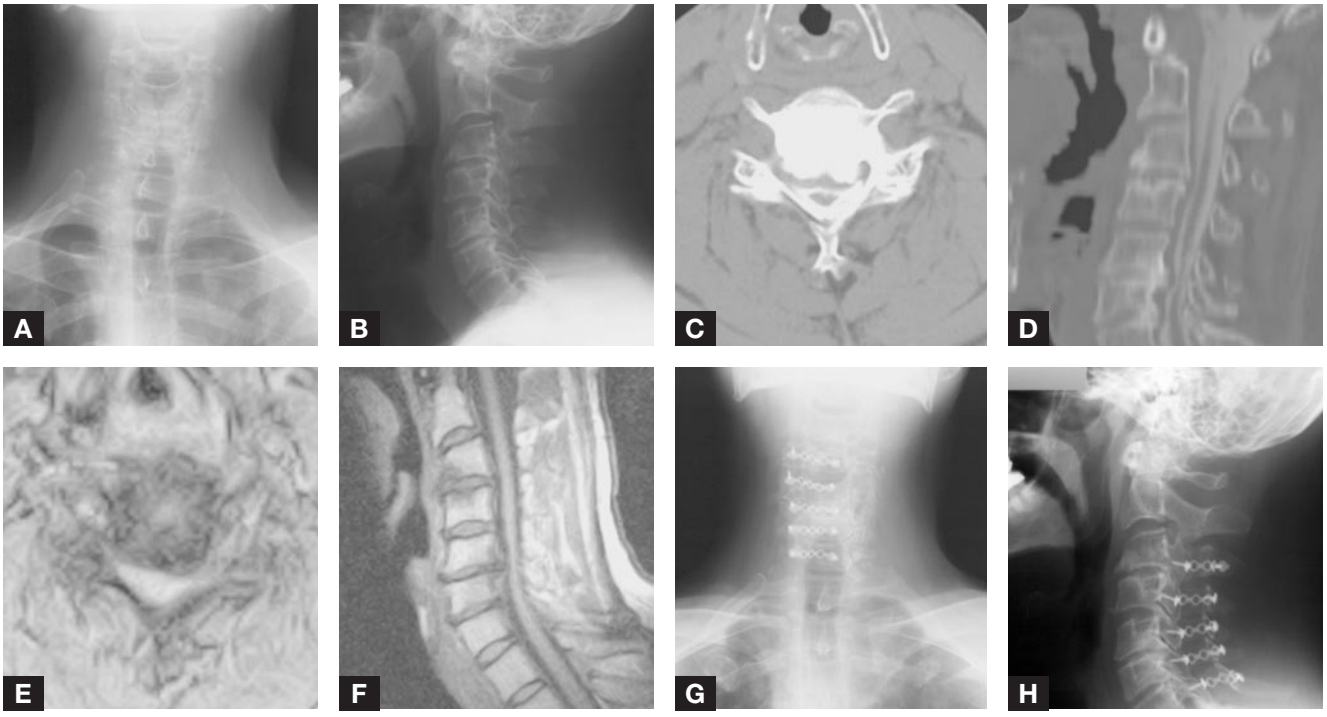

Figure 2. Fifty-nine-year-old man complained of neck pain and bilateral arm numbness and tingling, worse on left than on right. Positive Hoffmann and Babinski signs. Anteroposterior (A) and lateral (B) radiographs show spondylosis and degenerative changes in spine with maintenance of cervical lordosis. (C) Computed tomography (CT) shows narrowed spinal canal diameter. (D) Sagittal reconstruction of CT scan shows stenosis at several levels causing indentation of thecal sac. (E) Axial magnetic resonance imaging (MRI) shows narrowed spinal canal. (F) Sagittal MRI shows multilevel stenosis. Postoperative anteroposterior (G) and lateral (H) radiographs show instrumentation in place.

Most laminoplasties were from C3 to C7. Seldom did C7 and/or T1 stenosis require extension of the laminoplasty to this level. Although laminoplasty is technically feasible at C2, in the rare case of C2–C3 stenosis, use of the dome laminoplasty technique to adequately restore spinal canal diameter was preferred. Dome laminoplasty is simple and preserves the integrity of the C2 lamina and paraspinal muscle function.

The technique used in this study was described elsewhere[18] and is depicted in Figures 1A through 1F. Shoulder depression should be avoided, as the added traction on the brachial plexus is thought to possibly result in increased incidence of C5 nerve injury.

The laminar osteotomy was made using an AM-8 tip (Midas Rex, Medtronic, Memphis, Tenn), which is very safe to use around the dura. It is unicortical on the hinge side (ie, closing V-shape osteotomy) and bicortical on the opening side. The lamina was elevated 8 to 10 mm using a special square-ended Cobb elevator that allowed the placement of a small, thin edge under the lamina to facilitate elevation along with digital pressure on the posterior spinous process.

Secure fixation of the open-door laminoplasty was accomplished with either AO/ASIF (Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation) 2.5 maxillofacial miniplates (Synthes, West Chester, Pa) or Timesh plates (Medtronic Sofamor Danek, Memphis, Tenn). No bone or sutures were needed to hold the laminoplasty open, as the plates provided immediate and secure fixation. Early in the series, the patients were braced in an Aspen collar (Aspen Medical, Littleton, Co) for 6 weeks and then allowed gradual mobilization. It soon became apparent that many patients removed their collars within this period and rapidly regained motion with normal day-to-day activity. We now place patients in a soft collar for 3 weeks after surgery and allow gradual mobilization in flexion-extension, rotation, and side bending as tolerated. All patients were followed for 6 weeks and 3, 6, 12, and 24 months after surgery with serial radiographs and Nurick neurologic grading. Standard surgical data were recorded along with postoperative neurologic examination results.

Results

Of the 104 cases of myelopathy, 8 were caused by OPLL, 14 by congenital stenosis, and 82 by acquired degenerative stenosis (in some cases, the cause was a combination of congenital and acquired stenosis). There were 63 male and 41 female patients. Mean age was 59.2 years (SD, 11.6 years). There were 36 smokers (34.6%). Mean follow-up was 50.2 months (SD, 19.0 months), and minimum follow-up was 24 months. Twenty-eight patients (26.9%) had previous

cervical surgery. Mean symptom duration was 24 months (range, 2-84 months). Of the 85 patients who underwent MRI, 16 showed signal changes within the cord. Forty-four patients had a cervical myelogram followed by computed tomography (CT) scan. The primary indication for surgery was radiographically confirmed severe central cervical spinal stenosis in 104 (100%) of the patients combined with myelopathy. Many patients complained of upper extremity weakness, numbness, loss of dexterity (eg, handwriting degradation), and decreased grip strength. On physical examination, 66 patients (63.5%) exhibited gait disturbances, 56 (53.8%) had difficulty holding cups, 27 (26.0%) had a positive Hoffmann sign, 10 (9.6%) had sustained clonus, 10 (9.6%) had a positive Babinski sign, and 3 were so unsteady they were unable to ambulate (Table I).

All patients underwent instrumented open-door laminoplasty (as described earlier). Fifty-three patients had a right-sided opening, 50 had a left-sided opening, and 1 had a right-sided opening at C3 and C4 and a left-sided opening from C5 to C7. Seventy-one patients had concurrent foraminotomies. Mean surgical time was 2 hours 44 minutes (SD, 41 minutes), mean blood loss was 250 mL (SD, 82.1 mL), and mean number of levels was 3.8 (SD, 0.41). Postoperative complications included 4 nerve root injuries (3.8%), 1 of which (at C5) was permanent, and 1 transient neurologic deterioration (<1%), 1 incidental durotomy (<1%), and 5 wound infections (4.8%). There were no spinal cord contusions, epidural hematomas, or loss of fixation (Table II). Four patients required anterior cervical decompression and fusion a mean of 27 months after laminoplasty for persistent anterior cord compression and/or radiculopathy. The Nurick neurologic grade improved from 2.11 (SD, 0.85) before surgery to 0.63 (SD, 0.69) after surgery, for a mean improvement of 1.47 (SD, 0.92).

Discussion

Early anatomical studies established that cervical myelopathy results from the narrowing of the normal anteroposterior cervical spinal canal diameter of 17 to 18 mm to a critical threshold of less than 12 to 14 mm, resulting in spinal cord compression (Figure 2).[1] These studies also identified the primary causes of cervical stenosis resulting in myelopathy—the normal degenerative aging process, congenital narrowing aggravated by acute trauma, and bony malformations.[2] Subsequent epidemiologic reports described the natural history of symptomatic cervical stenosis as a stepwise deterioration, typified by periods of neurologic stability. In most untreated cases, the ultimate prognosis is poor. Some studies have shown that patients with severe cervical stenosis inevitably experience a progressive downhill course with their disease, eventually becoming functionally disabled, and with some losing their ability to ambulate.[6] Furthermore, nonoperative treatments for cervical spinal stenosis have usually demonstrated little efficacy in arresting the natural progression of neurologic deterioration.[7]

As opposed to the Asian population, in which OPLL is common, the North American population and our study population most often had congenital narrowing, degenerative spondylotic narrowing, or both causing cervical stenosis with myelopathy. The cause of congenital cervical stenosis is unknown, but its effects are considerable, as spinal canal narrowing may predispose a person to myelopathy at a very early age, particularly as degenerative changes develop within the cervical spine with aging. The etiology of acquired cervical spinal stenosis is thought to be natural aging changes within the spine that result in hypertrophy of the ligamentum flavum, uncovertebral joint hypertrophy, facet hypertrophy, and development of anterior spondylotic ridges, all of which contribute to the circumferential narrowing of the spinal canal with resultant spinal cord compression.[3,4] Other, less commonly encountered causes of cervical stenosis are OPLL, posttraumatic narrowing, tumors, and large acute herniated discs.

One of the first manifestations of congenital narrowing of the spinal canal may occur after a traumatic episode. A smaller diameter spinal canal correlates significantly with an increased chance of neurologic injury, whereas a large diameter canal provides more protection from neurologic injury.[5] These injuries also highlight the potential added contribution of excessive dynamic range of motion to development of a spinal cord injury when static narrowing of the spinal canal already exists.[3]

There are essentially 3 useful surgical treatments for cervical spinal stenosis—anterior decompression and fusion with or without instrumentation, posterior laminectomy with or without fusion and instrumentation, and laminoplasty techniques, which may be instrumented.[8] The conundrum is in determining which technique is best suited for a particular pathologic condition. These techniques also depend on the surgeon's training, experience, and familiarity with a specific procedure.[9]

For cervical stenosis with myelopathy involving only 1 or 2 vertebral bodies, the traditional treatment of choice has been anterior vertebrectomy and fusion with or without instrumentation.[10,11] The rationale for avoiding posterior procedures is their failure to address the anterior compression that results from the spinal cord being draped over the vertebral bodies and anterior osteophytes and the propensity for the kyphosis to worsen when the posterior stabilizing structures are violated. Likewise, there are numerous reports of favorable results with wide posterior decompression with concurrent fusion, particularly in straight and

lordotic spines.[12-14] However, more recent reports have shown that multilevel laminectomies have a higher incidence of complications and result in less improvement in neurologic function.[15]

Dissatisfaction with the results of multilevel corpectomies and extensile laminectomies with fusion for multilevel cervical stenosis led to the development of laminoplasty techniques to address multilevel disease.[16] These techniques were very successful in treating OPLL and subsequently have been used to treat cervical stenosis resulting from congenital and degenerative spondylosis.

Studies have reported better range of motion, less narcotic use, and lower prevalence of complications in patients who undergo laminoplasty than in patients who undergo multilevel anterior corpectomies and fusion.[17] However, there is no difference in operative time, length of hospital stay, amount of surgical blood loss, or degree of neurologic recovery (Nurick scores).[17] With further refinement, laminoplasty has also shown efficacy and a low complication rate when used to treat 1- and 2-level cervical stenosis, which traditionally has been treated with anterior fusion combined with instrumentation.[9]

The numerous laminoplasty techniques can be divided into 2 basic types—the midline splitting technique, in which the laminae are opened with a posterior spinous process splitting technique, and an osteotomy of the lamina, which allows rotation of the entire lamina away from the lateral mass.[18-20] Both techniques have demonstrated long-term success in increasing the spinal canal diameter and preventing worsening of the myelopathy.[21] Still, once the spinal canal is restored to a normal diameter, there is no guarantee that complete neurologic recovery will occur, particularly in patients with a long duration of symptomatology. MRI performed after laminoplasty has demonstrated that long-term preoperative spinal cord compression often leads to permanent spinal cord degeneration and atrophy. This damage is not repaired and leads to a permanent neurologic deficit.[19]

The Hirabayashi open-door laminoplasty technique was used in the cases in our series.[18,21] The technique was modified by adding titanium miniplates for rigid fixation to maintain the opening of the laminoplasty. Use of miniplates arose from frustration with other techniques for holding the osteotomy open—use of sutures or bone blocks, which are subject to potential failure or migration. The 1996 adoption of maxillofacial titanium plates (Synthes, West Chester, Pa) contoured to fit the open laminoplasty solved this dilemma by providing immediate stability with a simple technique. In 2001, a titanium plate system (Timesh; Medtronic Sofamor Danek, Memphis, Tenn) designed specifically for the open-door laminoplasty technique became commercially available.[16] Absence of fatigue failure or loosening of the plates in this large series of patients demonstrates the clear advantage of miniplate fixation. There are several potential reasons for this success. First, miniplates are subjected to minimal mechanical stress, as the lamina is still connected to the vertebral body. Second, the screws are not subjected to repetitive pullout forces because the lamina orientation directs the stresses at the bone–screw interface in a direction perpendicular to the screw (shear). Third, the closing unicortical laminar osteotomy provides progressive stability as it heals in the open position and relieves any stresses across the plate.

There have been clinical reports of persistent axial neck and shoulder pain after laminoplasty.[22] However, this problem was not observed in the patients in our series. This finding confirms the reports of others who have not noted any increase in axial neck and shoulder pain when comparing the various approaches for the treatment of cervical spinal stenosis.[15,17] No patient demonstrated any deterioration in preoperative myelopathy. However, C5 nerve root palsy was observed in 4 (3.8%) of our patients, with 1 having a permanent deficit, which is similar to the 3% to 8% overall rate reported by others.[23] Three patients had residual or recurrent neurologic symptoms caused by central stenosis from an anterior bony spur that delayed resolution of the myelopathy. These patients were treated with anterior cervical decompression and fusion with plating, which led to improvement in the myelopathy.

This retrospective study had several shortcomings, most notably the lack of patient-based outcome measures. Second was the lack of precise measurement of long-term final range of motion. What the study demonstrated is that laminoplasty with miniplates is safe, simple, and effective, that it has a low complication rate, and that it can reliably halt the progressive loss of neurologic function. No patient in this study required revision for fixation failure, which could have led to closure of laminoplasty—demonstrating the important advantage of internal fixation in maintaining the position of the lamina. The incidence of neurologic injury was small and similar to that reported in previous series. Only 4 patients required anterior revision for persistent pathology.

Conclusions

Laminoplasty combined with internal fixation using miniplates in this large retrospective study appears to be a very reliable technique for the treatment of multilevel cervical spinal stenosis.

Authors' Disclosure Statement

Drs. Dimar, Glassman, and Campbell wish to note that they receive consulting fees, royalties, and research grant support from Medtronic Sofamor Danek. No consulting fees, royalties, or research support were

received for this clinical study. Ms. Bratcher, Mr. Brock, and Dr. Carreon report no actual or potential conflict of interest in relation to this article.

References

1. Payne EE, Spillane J. The cervical spine; an anatomico-pathological study of 70 specimens (using a special technique) with particular reference to the problem of cervical spondylosis. *Brain*. 1957;80(4):571-596.
2. Clark E, Robinson PK. Cervical myelopathy: a complication of cervical stenosis. *Brain*. 1956;79(3):483-510.
3. Bernhardt M, Hynes RA, Blume HW, White AA. Cervical spondylotic myelopathy. *J Bone Joint Surg Am.* 1993;75(1):119-128.
4. Dunsker SB. Cervical spondylotic myelopathy: pathogenesis and pathophysiology. In: Dunsker SB, ed. *Cervical Spondylosis*. New York, NY: Raven; 1981:119-134.
5. Eismont FJ, Clifford S, Goldberg M, Green B. Cervical sagittal canal size in spinal injury. *Spine*. 1984;9(7):663-666.
6. Roberts AH. Myelopathy due to cervical spondylosis treated by collar immobilization. *Neurology*. 1966;16(9):951-959.
7. Phillips DG. Surgical treatment of myelopathy with cervical spondylosis. *J Neurol Neurosurg Psychiatry*. 1973;36(5):879-884.
8. Edwards CC 2nd, Riew KD, Anderson PA, Hilibrand AS, Vaccaro AF. Cervical myelopathy. Current diagnostic and treatment strategies. *Spine J*. 2003;3(1):68-81.
9. Yone K, Matsunaga S. Posterior procedures for myelopathy: indications, techniques, and results. *Semin Spine Surg*. 1999;11(4):331-336.
10. Yonenobu K, Hosono N, Iwasaki M, Asano M, Ono K. Laminoplasty versus subtotal corpectomy. A comparative study of results in multisegmental cervical spondylotic myelopathy. *Spine*. 1992;17(11):1281-1284.
11. Zdeblick TA, Bohlman HH. Cervical kyphosis and myelopathy. Treatment by anterior corpectomy and strut-grafting. *J Bone Joint Surg Am*. 1989;71(2):170-182.
12. Kumar VG, Rea GL, Mervis LJ, McGregor JM. Cervical spondylotic myelopathy: functional and radiographic long-term outcome after laminectomy and posterior fusion. *Neurosurgery*. 1999;44(4):771-777.
13. Dante SJ, Heary R, Kramer D. Cervical laminectomy for myelopathy. *Oper Tech Orthop*. 1996;5(1):30-37.
14. Maurer PK, Ellenbogen RG, Ecklund J, Simonds GR, Van Dam B, Ondra SL. Cervical spondylotic myelopathy: treatment with posterior decompression and Luque rectangle bone fusion. *Neurosurgery*. 1991;28(5):680-683.
15. Heller JG, Edwards CC, Murakami H, Rodts GE. Laminoplasty versus laminectomy and fusion for multilevel cervical myelopathy: an independent matched cohort analysis. *Spine*. 2001;26(12):1330-1336.
16. Park AE, Heller JG. Cervical laminoplasty: use of a novel titanium plate to maintain canal expansion—surgical technique. *J Spinal Disord Tech*. 2004;17(4):265-271.
17. Edwards CC 2nd, Heller JG, Murakami H. Corpectomy versus laminoplasty for multilevel cervical myelopathy: an independent matched-cohort analysis. *Spine*. 2002;27(11):1168-1175.
18. Hirabayashi K, Satomi K. Operative procedure and results of expansive open-door laminoplasty. *Spine*. 1988;13(7):870-876.
19. Kohno K, Kumon Y, Oka Y, Matsui S, Ohue S, Sakaki S. Evaluation of prognostic factors following expansive laminoplasty for cervical spinal stenotic myelopathy. *Surg Neurol*. 1997;48(3):237-245.
20. Tomita K, Kawahara N, Toribatake Y, Heller JG. Expansive midline T-saw laminoplasty (modified spinous process-splitting) for the management of cervical myelopathy. *Spine*. 1998;23(1):32-37.
21. Satomi K, Nishu Y, Kohno T, Hirabayashi K. Long-term follow-up studies of open-door expansive laminoplasty for cervical stenotic myelopathy. *Spine*. 1994;19(5):507-510.
22. Hosono N, Yonenobu K, Ono K. Neck and shoulder pain after laminoplasty: a noticeable complication. *Spine*. 1996;21(17):1969-1973.
23. Sakaura H, Hosono N, Mukai Y, Ishii T, Yoshikawa H. C5 palsy after decompression surgery for cervical myelopathy: review of the literature. *Spine*. 2003;28(21):2447-2451.